PARAMEDIC ROUNDS

Prehospital CPAP
Objectives:

By means of facilitator presentation and group discussion, the paramedic will be able to:

- Describe the pathophysiology of CHF and COPD – two common causes of SOB calls
- Explain the risk:benefit ratio of CPAP administration
- Compare current paramedic treatment to the auxiliary protocol for CPAP use
- Propose various patient scenarios where CPAP would be beneficial to patient outcome.
Agenda

• Brief overview of common conditions treated with CPAP

• CHF and COPD pathophysiology review and current treatment

• CPAP
  • what it is/how it works
  • benefits
  • risks/complications
  • protocol
  • different kinds of prehospital CPAP

• Study of Interest
The Function of the Heart:

- Is to move deoxygenated blood from the venous system through the right heart into the pulmonary circulation, and oxygenated blood from the pulmonary circulation through the left heart and into the arterial circulation. (5)
Heart Failure:

- Right side heart failure: Represents failure of the right ventricle to move unoxygenated blood from the venous system into the pulmonary circulation, with an eventual backup in the systemic venous circulation.

- Left side heart failure: Represents failure of the left ventricle to move oxygenated blood from the pulmonary circulation into the arterial circulation with the eventual backup of blood in the lungs. Both types result in decreased forward flow, resulting in poor circulation of oxygenated blood in the body. (5)
Pathophysiology of Congestive Heart Failure Terms:

- **Cardiac output**: Is the amount of blood that the ventricles eject each minute.

- Heart has capacity to adjust C.O, during sleep decreases, exercise increases.

- Determined by the cardiac reserve. Athletes have large cardiac reserve can increase to 5–6 times their resting levels.

- Heart failure pts often use their cardiac reserve at rest; climbing flight of stairs may cause to exceed their cardiac reserve (5)
Cardiac Output:

- Cardiac output = Reflects how often the heart beats each minute (heart rate) and how much blood it pumps with each beat (stroke volume) and expressed as product of heart rate and stroke volume.

- $C.O = \text{heart rate} \times \text{stroke volume}$

- Heart rate: Regulated by the balance between sympathetic nervous system (increase heart rate) and parasympathetic (slows heart rate)

- Stroke volume: Is the function of preload, afterload, and myocardial contractility (5)
Preload/Afterload

- **Preload:** Reflects the volume or loading conditions of the ventricle at the end of diastole, just before the onset of systole.

- If preload increases the stroke volume increases with the Frank–Starling mechanism.

- **Afterload:** Represents the force that the contracting heart muscle must generate to eject blood from the filled heart.

- Main components are the systemic (peripheral) vascular resistance and ventricular wall tension (5).
Afterload:

- When systemic vascular resistance is elevated as with arterial hypertension

- An increased left intraventricular pressure must be generated to first open the aortic valve and move blood out of ventricle and into the systemic circulation. This increase pressures cause increase ventricular wall stress or tension.

- Excessive afterload may impair ventricular ejection and increase wall tension (5)
Myocardial Contractility

- Myocardial Contractility (inotropy): Refers to the contractile performance of the heart.

- Represents the ability of the contractile elements (actin and myosin filaments) Contractility increases cardiac output independent of preload and afterload. Use ATP and Calcium ions (Ca++) in the interaction between actin and myosin filaments during cardiac muscle contraction (5)
Inotropic Agents

- Digitalis and related cardiac glycosides are inotropic agents.

- That exert their effects by inhibiting the NA+/K ion pump.

- Which increases intracellular NA+ this in turn leads to an increase in intracellular CA++ through the NA+/Ca++ exchange pump

- Digitalis may also lower patients heart rates and effect cardiac output, depending on patients condition and physiologic state. (5)
Ejection Fraction

- Ejection Fraction – The portion of blood that is pumped out of a filled ventricle as a result of a heartbeat.

- Systolic Dysfunction; primarily defined as a decrease in myocardial contractility, characterized by and ejection fraction of less than 40%, normal is 65% of the blood that is present in the ventricle at the end of diastole. (5)
Respiratory Terms

- **Tidal Volume (Vt)** – Is the measure of the volume of air inspired and exhaled with each breath at rest. Healthy adult is usually 400–500mls, however can be substantially higher.

- **Residual Volume** – Is the air remaining after expiration. Even with maximal expiratory effort, all the air cannot be emptied from the lungs. This remaining air is the residual volume.

- **Minute Volume (Vm)** – Equals the number of breaths per minute times the volume of each breath (Vt) ie 12 breaths/min X 500cc(Vt) = 6000cc per minute (5)
• Functional Reserve Capacity (FRC) – Is the volume of gas remaining in the lungs at the end of normal tidal exhalation. (positive end expiratory pressure, or P.E.E.P device, limits exhalation and increases the FRC and keeps the alveoli from collapsing.

• Peak inspiratory flow

• Inspired oxygen (Fi02) (5)
Right-Sided Heart Failure

- Right-sided heart failure; impairs the ability to move deoxygenated blood from the systemic circulation into the pulmonary circulation.

- When R ventricular fails there is a reduction in the amount of blood moved forward into the pulmonary circulation and then into the left side of the heart. Causing a reduction of left ventricular cardiac output.

- If right ventricular doesn’t move blood forward, there is accumulation or congestion of blood into the systemic venous system. (5)
Effects of Right–Sided Failure

• Major effect of right–sided heart failure: Is development of peripheral edema.

• Also produces congestion of the viscera, As venous distention progresses Liver engorged, through the blood backs up in the hepatic veins that drain into the inferior vena cava

• May cause Right upper quadrant pain (5)
Causes:

- Causes of Right side heart failure include: Conditions that impede blood flow into the lungs or compromise the pumping effectiveness of the right ventricle.

- Left side heart failure is the most common cause of right side heart failure.

- Right ventricular infarction and cardiomyopathy.

- Stenosis or regurgitation of the tricuspid or pulmonic valves,

- Pulmonary hypertension (5)
Left-Sided Heart Failure

- Left-sided heart failure: Impairs the movement of blood from the low-pressure pulmonary circulations into the high pressure arterial side of the systemic circulation.

- With impaired left heart function there is a decrease in cardiac output to the systemic circulation, and blood accumulates in the left ventricle, left atrium, and pulmonary circulation, which increases pulmonary venous pressure (5).
Left-Sided Heart Failure

- When pressure in the pulmonary capillaries, normal 10 mmHg exceeds the capillary osmotic pressure, normally 25 mm Hg, there is a shift of intravascular fluid into the interstitium of the lung and development of pulmonary edema.

- Episode of pulmonary edema often occurs at night, person has been reclining for some time and the gravitational forces have been removed from the circulatory system (5).
Left Sided Heart Failure Causes:

- Most common causes of left sided heart failure:
  - Acute myocardial infarction
  - Hypertension.
  - Left sided heart failure can occur very rapidly in persons with acute myocardial infarction. Even if area small.
  - Stenosis or regurgitation of the aortic or mitral valves creates left side back flow that results in pulmonary congestion. (5)
Left Sided Heart Failure Cont’d

• Pulmonary pressure rises as result of congestion may progress to produce right sided heart failure.

• Causes of CHF

• Acute MI, Arrhythmia A–Fib (5)
Signs and Symptoms:

- Accessory muscle use
- Bilateral crackles
- Orthopnea – can't lie down
- JVD
- Pedal edema
- Chest pain (possible AMI)
- Abnormal vials Hypertension, tachycardia, rapid and labored respirations
- Decreased Spo2 Speech dyspnea
- Diaphoresis
- Foamy Blood tinged sputum (5)
COPD

Defined as “a progressive, irreversible condition characterized by diminished inspiratory and expiratory capacity of the lungs” (6)0
COPD

- Estimated that approximately 1.9 million Canadians have COPD (5)

- Originally classified as two separate conditions: chronic bronchitis and emphysema

- Currently – considered a collective term – accepted that both conditions frequently co-exist (3)
Risk Factors for COPD

- Genes (alpha antitrypsin deficiency)
- Exposure to particles – smoking, occupational dust, air pollution, etc
- Gender
- Age
- Respiratory Infections
- Socioeconomic Stress
- Nutrition (3)
Pathogenesis of COPD:
- Usually multiple issues, including,
  - Inflammation & fibrosis of the bronchial wall
  - Hypertrophy of the submucosal glands
  - Hypersecretion of mucous
  - Loss of elastic lung fibres & alveolar tissue (5)
Emphysema

- Enlargement of airspaces
- Destruction of lung tissue
- Loss of elastic fibres
- Distal to terminal bronchioles (5)
Pink Puffers

- Lack of cyanosis
- Evidence of accessory muscle use
- ‘Pursed-lip’ (puffer) breathing
- Barrel chest (5)
Bronchitis

- Increased mucous production
- Obstruction of both small & large airways
- Chronic productive cough
- Mismatch of ventilation & perfusion (5)
Blue Bloaters

• Evidence of cyanosis around mouth

• Typically younger in age

• Fluid retention due to right sided heart failure (5)
COPD – Acute Exacerbation

• One of the main causes of hospitalization and death

• Characterized by a change in the patient’s baseline dyspnea, cough and/or sputum – acute in onset (2)
Current Tx for COPD Exacerbation

• $O_2$ via NRB mask
• Bronchodilators
• BVM
• Intubation if required
  ✓ $\text{SpO}_2$ less than 90%
  ✓ Decreased LOC
  ✓ Unable to protect airway (SWORBHP)
Current Tx for Acute CHF

- O2 via NRB mask
- NTG
- Lasix (currently)
- BVM
- Intubation if required
  - SpO2 less than 90%
  - Decreased LOC
  - Unable to protect airway (SWORBHP)
CPAP

Provides:

“Continuous positive pressure throughout the respiratory cycle [which] improves oxygenation and tidal volume”

• Thus decreasing respiratory effort
• Thus easing the required workload of the respiratory muscles
• Assists pulmonary compliance (4)
Benefits of CPAP

- Increased oxygenation and tidal volume
- Improved arterial pH and mental status
- Reduced need for endotracheal intubation
- Reduced risk of infection rates due to less intubation
- Reduced in-hospital stay time
- Decreased mortality
- Improved patient comfort – reduced need for sedation and analgesia (1,4)
Indications CPAP is Working

- Reduced heart rate
- Reduced respiratory rate
- Reduced dyspnea
- Blood pressure returning to normal
- Increasing SpO$_2$
- Decreasing end-tidal CO$_2$
- Improving mental status (1)
CPAP – Risks/Complications:

- Hypotension is a potential complication, but is very rare and should be detected early by serial BP measurements. BP falls below 90 systolic or the patient develops signs and symptoms of shock.

- Do not continue remove CPAP

- Barotrauma and pneumothorax are extremely rare

- Prolonged use in the hospital may cause local skin damage, sinus problems or eye irritation
“The days of oxygen, morphine, lasix and intubate have been replaced by nitrates, ace inhibitors and now more so then ever, the use of early non invasive positive pressure ventilation.”

(Non Invasive Positive Pressure Ventilation – Is it Ready for Prehospital Care? – Dr. Sheldon Cheskes, Medical Director)
How Does It Work?

• Preload is reduced by a decrease in venous return to the left ventricle.

• In patients with acute respiratory distress as a result of COPD we see improvements in lung compliance as well as a decrease in the work of breathing.

(Non Invasive Positive Pressure Ventilation – Is it Ready for Prehospital Care? – Dr. Sheldon Cheskes, Medical Director)
CPAP

• Used successfully in hospitals since the early 1990s, CPAP has proven beneficial for patients with pulmonary edema and chronic obstructive pulmonary disease (COPD), as well as those on dialysis who become fluid overloaded.

• Studies show that services using CPAP reduce the need for intubation from around 25% to 6%
CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) Protocol

• INDICATIONS: Patient is awake and able to follow commands and who is in severe respiratory distress as evidenced by two of three of the following protocol.

• A respiratory rate $\geq 24$ breaths per minute AND/OR

• Sp02 $< 90\%$ at any time AND/OR

• Accessory muscle use
Indications

• And with signs and symptoms consistent with:
  ✓ Exacerbation of chronic obstructive pulmonary disease (COPD), OR
  ✓ Acute pulmonary edema

CONDITIONS:
• Age > 12 and > 40 kg
Absolute Contraindications (DO NOT USE)

- Asthma exacerbation
- Unable to cooperate
- Suspected Pneumothorax
- The patient is intubated
- The patient cannot maintain their airway or there is a threat to the airway (e.g. foreign body airway occlusion, ongoing vomiting or GI Bleeding).
- Decreased mentation (unresponsive to speech, and/or unable to follow commands).
Contraindications Cont’d

• Respiratory rate < 8/min
• Systolic BP < 90 mmHg
• Cardiac arrest
• Major trauma or burns (face, neck, chest/abdomen)
• Facial anomalies
• Inability to sit upright
• Tracheostomy
Procedure

1. While one paramedic is setting up the CPAP equipment, the second paramedic should treat the patient’s underlying condition according to the appropriate treatment protocol.

2. Position the patient sitting upright

3. Carefully explain the procedure to the patient

4. Ensure adequate oxygen supply to the ventilation device (connect the generator to the oxygen source – tank or wall outlet).

5. Assemble CPAP mask, circuit and device.

6. Connect the circuit to the oxygen source according to the manufacturer’s directions.

7. Monitor patients a per BLS standards (to include oximetry if available).
8. Place ETCO2 monitor if available

9. Turn the ON/OFF valve fully on, be sure the gas is flowing, and then apply the delivery device/mask over the mouth and nose with the enclosed straps. Ensure a tight seal of the mask to the patient’s face.

10. Progressively increase the pressure from 5 cmH2O to a max of 15 cmH2O depending on the patient’s response to therapy

11. Confirm amount of CPAP delivery by manometer reading if available. Increase in FiO2 may be required to maintain oxygen saturation > 92%. If using an open CPAP system ensure adequate supply of oxygen is available.

12. Check vital signs and pulse oximetry frequently (every 5 minutes.)
13. Once applied, the mask may be removed for a short time to administer appropriate medication as indicated (for example, Nitroglycerin for CHF and Nebulized bronchodilator therapy for COPD).

14. If respiratory status deteriorates, remove device and consider intermittent positive pressure ventilation via BVM and/or endotracheal intubation.

Removal Procedure:
1. CPA therapy needs to be continuous and should not be removed unless the patient cannot tolerate the mask, requires medications (as per procedure) or experiences respiratory arrest or begins to vomit.

2. Intermittent positive pressure ventilation with a bag–valve–mask, placement of a supraglottic airway or endotracheal intubation should be considered as indicated if the patient is removed from CPAP therapy due to deterioration.
Types of CPAP


- The generator is a simple and rugged device that attaches directly to your portable oxygen regulator’s high-pressure port.

- Combine a Venturi valve and an expansion chamber for generating high, constant flow rates of ventilation gas. The disposable CPAP set combines a large bore oxygen hose and a facemask with PEEP valve.
Demand Flow CPAP

• Demand Flow CPAP device

• Provides oxygen flow only while the patient inspires, resulting in reduced gas consumption and improved respiratory monitoring.

• The PortO2 Vent has an integrated PEEP mechanism with a pressure manometer for conveniently adjustable pressure therapy using a single control knob.
Transport Ventilator with an Integrated CPAP Function:

• Any of these devices offer the superior control of time-cycled, volume-constant, mandatory ventilation WITH the ability to help people breath better BEFORE they stop

• CareVent ALS+

Other transport ventilator’s:
• AutoVent 4000, Draeger Medical Oxylog 3000, Uni-Vent Eagle 754
Disposable CPAP:

- Boussignac CPAP: Respiratory aid device for patients breathing spontaneously. This system is permanently open. Pressure is generated by the injection of gases passing through micro-capillaries (located all around the CPAP device) increasing in speed and generating turbulence therefore creating a "virtual valve."
Smiths Medical Oxy–PEEP:

- Is a high concentration, high flow oxygen diluter with adjustable PEEP. This unique system allows for variable flows, variable O2 concentrations and variable PEEP, all in a pre–packaged kit suitable for the ambulance.
Study of Interest

Basis of the Study

• To determine whether patient’s in severe respiratory distress who are treated with CPAP in out–of–hospital setting have a lower overall rate of tracheal intubation than those who are treated conventionally (8)
Study of Interest

Methods

• Pt’s were randomized through a ‘blinded’ dispatch centre to determine if they were treated with CPAP or usual care

Setting

• Within Nova Scotia, from July 2002 – March 2006
Inclusion Criteria

- Severe resp distress with falling resp efforts
- Accessory muscle use
- Resp rate >25 breaths per minute
- Hypoxia
- Normal LOC
- Pt’s ability to understand/cooperate with CPAP
- Hemodynamically stable
- No chest pain within 3hrs
- Enough portable O2
- > 16 years of age
- No advance directive – DNR

(8)
Table 2. Baseline characteristics (mean [SD] unless otherwise noted).*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Usual Care, N=34</th>
<th>CPAP, N=35</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, median</td>
<td>70.5</td>
<td>69</td>
</tr>
<tr>
<td>Sex, % female</td>
<td>41.2</td>
<td>57.1</td>
</tr>
<tr>
<td>Respiratory rate, breaths/min</td>
<td>37.6 (6.1)</td>
<td>38.2 (8.0)</td>
</tr>
<tr>
<td>SpO₂, %</td>
<td>75.0</td>
<td>81.5</td>
</tr>
<tr>
<td>Pulse rate, beats/min</td>
<td>121.2 (23.8)</td>
<td>112.4 (25.2)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>157.3 (42.6)</td>
<td>162.9 (37.9)</td>
</tr>
<tr>
<td>On-scene time, min</td>
<td>21.1 (9.3)</td>
<td>22.2 (7.4)</td>
</tr>
<tr>
<td>Total out-of-hospital time, min</td>
<td>43.8 (12.4)</td>
<td>41.3 (12.8)</td>
</tr>
</tbody>
</table>

Out-of-hospital clinical impression of CHF, chronic obstructive pulmonary disease or asthma

|                         | 33    | 34    |

*Median values reported for variables that were not normally distributed.
**Figure 1.** Patient flow through the study.
References


References Cont’d


References

• CPAP Pictures

✓ http://www.otwo.com/prod_atv.htm
✓ http://www.eresp.com/
✓ http://www.images.google.ca/images
Questions?
Some Terminology

- **Tidal Volume (TV)** – amount of air that moves into and out of the lungs with each breath

- **Inspiratory Capacity (IC)** – sum of inspiratory reserve volume and TV

- **Inspiratory Reserve Volume (IRV)** – maximum amount of air that can be inhaled from the point of maximal expiration

- **PEEP** – maintains a degree of positive pressure at the end of exhalation (5)