High Frequency Oscillatory Ventilation of the Large Pediatric and Adult Patient

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Objectives

• Brief history of HFOV
• Who will benefit from HFOV
• When should you initiate HFOV
• How do you initiate HFOV
  • Oxygen strategies
  • Ventilation strategies
• When do you discontinue HFOV
A Brief History of HFV

- 1977: Klain & Smith HFJV
- 1982: HFOV in Animals
- 1985: Animal Studies - Role of P-V in Lung Injury
- 1987: P-V Curve using Lung CT in ARDS
- 1990: FDA Approval of 1010 HFV
- 1991: HFOV Approved For Use in Neonates
- 1993: HFOV in Adults
- 1995: HFOV Approved For Use in Peds
- 1997: Ventilator Strategies and Inflammatory Mediators
- 2001:
3100A Ventilator

• Approved in 1991 for Neonatal Application for the treatment of all forms of respiratory failure

• Approved in 1995 for Pediatric Application, with no upper “weight limit”

• For treating selected patients failing conventional ventilation
High Frequency Oscillatory Ventilation
High Frequency Oscillatory Ventilation
3100B Ventilator

• Approved for sale outside the US in 1998 for patients weighing > 35 kg failing CMV

• Approved September 24, 2001 by the FDA for sale in the US
High Frequency Oscillatory Ventilation
IS THERE A DIFFERENCE BETWEEN THE 3100A AND THE 3100B?

Honey please, just calm down. Let me explain....
3100 A

Limit Adjust Button

Piston centering adjustable

3100 B

No Limit Adjust Button

Piston centering connected to I:E Ratio
Who
Will benefit from
HFOV 3100B?
Not-So-Normal Chest Radiograph
HFOV 3100B

- Only Pathology studied to date has been ARDS
- Questions about management of adults with massive airleak?
- Questions about management of Respiratory Failure based on any other disease process
The American – European Consensus Criteria for ARDS

<table>
<thead>
<tr>
<th>Timing</th>
<th>Oxygenation</th>
<th>Chest Radiograph</th>
<th>Pulmonary Artery Occlusion Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute onset</td>
<td>PaO2/FiO2 &lt; 200 torr (regardless of PEEP level)</td>
<td>Bilateral Infiltrates on Frontal chest Radiograph</td>
<td>&lt; 18 mmHg or no Clinical evidence Of left atrial hypertension</td>
</tr>
</tbody>
</table>
ARDS Pulmonary Injury Sequence

• Phase 1 Early Exudative Process

• Phase 2 Proliferative (Day 5-10)

• Phase 3 Fibrotic (Day 10-14)
Acute Respiratory Distress Syndrome

- 150,000 cases / year
- 75,000 deaths / year
- $5,000,000,000 / year
- No curative treatment on the horizon
Etiology of ALI / ARDS

Direct Causes
- Aspiration
- Pneumonia
- Emboli
- Near Drowning
- Reperfusion Injury
- Inhalation Injury

Indirect Causes
- Sepsis
- Shock/Trauma
- Pancreatitis
- Overdose
- Transfusion
- Drug OD
- CP Bypass
Ventilator Induced Lung Injury

- All forms of positive pressure ventilation (PPV) can result in ventilator induced lung injury (VILI)
- VILI is the result of a combination of the following processes:
  - Barotrauma
  - Volutrauma
  - Atelectrauma
  - Biotrauma
Barotrauma

- High airway pressures during PPV can cause lung overdistension with gross tissue injury
- This injury can allow the transfer of air into the interstitial tissues at the proximal airways
- Clinically, barotrauma presents as pneumothorax, pneumomediastinum, pneumopericardium, and subcutaneous emphysema

Slutsky, Chest, 1999
Volutrauma

- Lung overdistension can cause diffuse alveolar damage at the pulmonary capillary membrane.
- This may result in increased epithelial and microvascular permeability, thus, allowing fluid filtration into the alveoli (pulmonary edema).
- Excessive end-inspiratory alveolar volumes are the major determinant of volutrauma.
Atelectrauma

- Mechanical ventilation at low end-expiratory volumes may be inefficient to maintain the alveoli open.
- Repetitive alveolar collapse and reopening of the under-recruited alveoli result in atelectrauma.
- The quantitative and qualitative loss of surfactant may predispose to atelectrauma.
Biotrauma

- In addition to the mechanical forms of injury, PPV activates an inflammatory reaction that perpetuates lung damage.

- Even ARDS from non-primary etiologies will result in activation of the inflammatory cascade that can potentially worsen lung function.

- This biological form of trauma is known as biotrauma.
Lung Inflation Patterns on multi-Scan CT

30 kg Pig
Post Saline Lavage
PCV
mPaw 13 cmH2O
PEEP 5 cmH2O
Lung Inflation Patterns on multi-Scan CT

30 kg Pig
Post Saline Lavage
PCV
mPaw 23 cmH20
PEEP 15 cmH20
Pressure and Volume Swings

During CMV, there are swings between the zones of injury from inspiration to expiration.
Pressure and Volume Swings

During CMV, there are swings between the zones of injury from inspiration to expiration.
Why
Should you treat ARDS with HFOV?
Pressure and Volume Swings

During HFOV, the entire cycle operates in the “safe window” and avoids the injury zones.
During HFOV, the entire cycle operates in the “safe window” and avoids the injury zones.
Breaking the Cycle!!
Lung Recruitment & Protection

• Goal
  • Nearly constant alveolar volume
  • Nearly constant alveolar pressure
  • Lower peak airway pressure

• Method
  • High Frequency Oscillatory Ventilation
  • Optimal Lung Volume
“Open up the lung... and keep it open!”

Burkhard Lachmann 1992
Experimental demonstration of dynamic sustained inflation for lung recruitment during HFOV

Alexandre T. Rotta, MD
March 2003

Pediatric Critical Care Research Laboratory
State University of New York at Buffalo
The Women and Children's Hospital of Buffalo
HFOV and Lung Recruitment

mPaw 15 cmH$_2$O  
Pre-recruitment  
Post-recruitment
Lung Recruitment using HFOV

Recruitment of lavaged animal lung using increasing levels of mPaw
Ventilator Induced Lung Injury (VILI)

- **Barotrauma**
  - extralveolar air caused by structural disruption due to high airway pressure which is often the result of large lung volumes and not high airway pressure per se. "Barotrauma is Volutrauma" Dreyfuss D, et al.

- **Volutrauma**
  - regional lung overdistension caused by overinflation and uneven expansion of the lungs
Ventilator Induced Lung Injury (VILI)

- **Atelectrauma**
  - shear forces generated during cyclic closure and reopening of terminal airways.

- **Biotrauma**
  - activation of effector cells to release inflammatory mediators due to physical stresses associated with mechanical ventilation.
When
Do you initiate HFOV?
Thinking of starting HFOV???
Don’t wait too late!!!
Current Trends in Conventional Ventilation Strategies

- ARDSnet Study (6 ml/kg)
  - High PEEP lower Vt strategies
- PC-Inverse Ratio
- APRV/Bi-Level
- HFOV
- Therapeutic Modalities
  - Lung Recruitment Maneuvers
  - Kinetic Therapy
When Should HFOV be Initiated?

- If FIO$_2$ $>$ 0.60 - 0.70 and PEEP $>$ 10 – 14 cmH$_2$O while not being able to maintain SpO$_2$ $>$ 88%
- Unable to maintain Pplat $<$ 30 cmH$_2$O
- mPaw on CV is $>$ 24 cmH$_2$O
- Patient requiring paralysis for oxygenation
- Earlier intervention better
Open Lung Ventilation

Airway pressure (cm H$_2$O)

Volume above FRC (liters)

Normal

ARDS

Lower inflection point

Upper inflection point
Alveolar Volumetric Changes

Conventional

HFOV

Insp.  \approx\text{Exp.}

Insp.

Exp.
How Do you initiate HFOV?
Let’s try HFOV!!!
\[ \Delta P \]

Hz

mPaw
Theory of Operation

- Controls for Oxygenation and Ventilation are mutually exclusive

- Oxygenation is primarily controlled by the Mean Airway Pressure (mPaw) and the FiO\textsubscript{2}

- Ventilation is primarily determined by the stroke volume (Delta-P) and the frequency of the ventilator.
HFOV = Super CPAP
FiO2 is set with blender

Mean Airway Pressure (mPaw)

Amplitude (ΔP)

Power Knob

Bias Flow

Frequency (Hz)
Oxygen Strategies
Mean Airway Pressure (mPaw)

FiO2 is set with blender
Oxygenation – Clinical Tips

• Initiate HFOV
  • FiO2 of 1.0
  • mPaw of 5 cmH₂O greater than mPaw on CMV
    • Increase mPaw by 1-4 cmH₂O to achieve optimal lung volume
      • Optimal lung volume is determined by increasing SpO₂ while maintain FiO₂ or weaning FiO₂
    • Diaphragm T8-T9 on CXR
  • Maintain mPaw while weaning FiO₂ to < .60
Oxygenation – Clinical Tips

- Follow CXR to assess lung expansion
  - If diaphragm is round and between T8-T9, continue to wean FiO2
  - If diaphragm is flattened and greater than T-9, wean mPaw by 1 cmH2O and reassess CXR
- Should be able to wean FiO2 to < .60 in first 12 hours
  - If unable to reach FiO2 .60, consider recruitment maneuver by increasing mPaw (sustained inflation)
Oxygenation – Clinical Tips

• Ensure adequate intravascular volume and cardiac output
• Consider volume loading or initiate inotropes
  • Improves V/Q matching
  • High intrathoracic pressures can impede venous return and adversely affect cardiac output
• Closely monitor hemodynamic status
• Utilize pulse oximeters and transcutaneous CO₂ monitors to set appropriate HFOV settings in between ABGs
Oxygenation Strategies

• ↑ mPaw until SpO2 stabilizes 88 - 94% and begin to ↓ FiO2 to 60%

• Avoid hyperinflation on CXR

• Optimize preload, myocardial function
Oxygenation

CLINICAL TIPS-
- Must have adequate mPaw and hemodynamic performance
- Perfusion must be matched to ventilation for adequate oxygenation
- Chest x-rays and oximetry are necessary
Oxygenation

PVR is increased with:

- Atelectasis
  - Loss of support for extra-alveolar vessels
- Over expansion
  - Compression of alveolar capillary bed

The lung must be recruited, but guard against over expanding
Ventilation Strategies
Ventilation Strategies

• Alveolar ventilation during CMV is defined as:
  \[ F \times V_t \]

• Alveolar Ventilation during HFV is defined as:
  \[ F \times V_t^2 \text{ (squared)} \]

• Therefore, changes in *stroke volume* delivery (as a function of Delta-P, Freq., or % Insp. Time) have the most significant affect on CO2 elimination
Amplitude ($\Delta P$) Power Knob
SensorMedics 3100B

- Electrically powered, electronically controlled piston-diaphragm oscillator
- mPaw of 5 - 55 cmH₂O
- Pressure Amplitude from 8 - 130 cmH₂O
- Frequency of 3 - 15 Hz
- % Inspiratory Time  30% - 50%
- Flow rates from 0 - 60 LPM
Primary Control for PaCO₂

- **Amplitude (ΔP)**
  - The power knob regulates the force with which the piston moves from baseline
  - The degree of deflection of the piston (amplitude) determines the tidal volume
  - This deflection is clinically demonstrated as the “wiggle” seen in the patient
  - The wiggle factor can be utilized in assessing the patient
Alveolar ventilation during CMV is defined as:

$$ F \times V_t $$

Alveolar Ventilation during HFV is defined as:

$$ F \times V_t^2 $$

Therefore, changes in *stroke volume* delivery (as a function of Delta-P, Freq., or % Insp. Time) have the most significant affect on CO$_2$ elimination
Ventilation Strategies

- CWF – adjust Power Setting to target PaCO2 between 45-55 or normalize pH
- Increase the amplitude in 5 cmH2O increments until the max Delta-P is reached, then decrease the frequency
- Some find that as they increase the amplitude 15-20 cmH2O with no positive effect, consider ET tube deflation and/or decreasing the frequency by 1 Hz provides the desired effect.
- Allow permissive hypercarbia if indicated, keeping pH > 7.20
Ventilation

The **amplitude** is the primary control of ventilation. It is created by the distance that the piston moves, resulting in a volume displacement and a visual CHEST Wiggle. It may also be described as the peak-to-trough swing across the mean airway pressure.

The **power** controls the force with which the piston moves.
Amplitude Attenuation

Airway Pressure Waveforms

HFOV

Conventional
Amplitude Attenuation

Airway Pressure Waveforms

HFOV

Conventional
Frequency (Hertz)
Secondary control of PaCO$_2$ is the set

Frequency

Hertz (Hz): A unit of frequency equal to one cycle per second
Therefore, 1 Hz equals 60 breaths/minute
Initial Frequency Settings

- Guidelines for setting the initial frequency

<table>
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<tr>
<th>Patient Weight</th>
<th>Frequency</th>
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<tbody>
<tr>
<td>Neonates</td>
<td>10 to 15 Hz</td>
</tr>
<tr>
<td>Pediatrics</td>
<td>7 to 9 Hz</td>
</tr>
<tr>
<td>Adults</td>
<td>5 to 6 Hz</td>
</tr>
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</table>

- Adjustments in frequency are made in steps of 1 Hz
Frequency

• To evaluate the effects of changes in frequency with regards to CO$_2$ elimination, let us look at 2 different frequencies
  • 4 Hz
  • 8 Hz
Let's consider a time interval of X.
The lower the frequency setting, the larger the volume displacement.

4 Hz

8 Hz
The higher the frequency setting, the smaller the volume displacement.
Therefore, lower frequencies have a larger volume displacement and improved CO$_2$ elimination.
Secondary control of PaCO₂ is the frequency. The frequency controls the time allowed (distance) for the piston to move. Therefore, the lower the frequency setting, the greater the volume displacement, and the higher the frequency setting, the smaller the volume displacement.
Ventilation Strategy

• If CO₂ retention persists, decreasing cuff pressure to allow gas to escape around the ET tube
• This will move the fresh gas supply from the wye connector to the tip of the ET tube
• Adjust mPaw or flow to increase mPaw by 5 – 7 cmH2O, Deflate cuff to drop mPaw to ordered mPaw
How Should HFOV be Initiated?

- Adequate sedation, analgesia and paralysis
- Set mPaw 5 cmH₂O above mPaw on CMV
  - Consider Alveolar Recruitment Maneuver
- Begin at “POWER” of 4 and adjust P to achieve CWF to Mid Thigh
- Set Hz at 5 - 6 (Adults), 7-9 (Pediatrics)
  - unless compartment syndromes, obesity, etc
- Set % IT at 33% and BF 30 Liters/minute or greater
- Consider ETT cuff leak
When do you discontinue HFOV?
HFOV - Weaning Strategy

• Wean FiO\textsubscript{2} for SpO\textsubscript{2} of 88 - 92%
  • Target FiO\textsubscript{2} .40-.60, may decrease mPaw in increments of 1- 3 cmH\textsubscript{2}O, once FiO\textsubscript{2} < .60
  • Monitor lung volume with CXR
• Amplitude (P) is weaned by increments of 5 cmH\textsubscript{2}O for desired PaCO\textsubscript{2}
• Once optimal frequency is found, leave the frequency
• However you may increase the frequency to raise the PaCO\textsubscript{2}

“Patience, Patience, Patience!!”
HFOV - Weaning Strategy continued...

- Conversion from HFOV to CMV
  - When FiO₂ is < .40, mPaw is weaned to 20-24 cmH₂O, patient can generally cross back over to CMV
  - Improved CXR
  - Target mPaw on CMV equal to HFOV mPaw
    - PC, Vt 6 ml/kg, PEEP >10 cmH₂O, rate 15-20, I:E ratio of 1:2 or 1:1
Low Vt and High PEEP

PRVC, f 24, Vt 5 ml/kg, PEEP 20 cmH₂O, FiO₂ 1.0, I-time 1 sec, mPaw 30 cmH₂O
ABG’s 7.31/44/55/22 (OI 42)

High PEEP

HFOV, mPaw 35 cmH₂O, ΔP 50 cmH₂O, Frequency 4 Hz, FiO₂ 1.0, I-time 33%
ABG 7.31/45/231/21 (OI 14)

HFOV
HFOV works!!!!
HFOV - Weaning Strategy continued...

3. Set up CV to achieve mPaw close to 20 cmH$_2$O (e.g. PCV TV 6 ml/kg, 1:1, PEEP > 10 cmH$_2$O, rate 20)

4. Wean conventionally (normalize I:E in 10% increments, reduce PEEP to <8, breathing trials)

5. Anticipate prolonged neuromuscular weakness
HFOV-Tools for Success

• If considering HFOV, establish institutional criteria for consideration

• Adequate training of medical, nursing and respiratory care staff

• Patience, patience, patience!!
• mPaw can be increased 1-2 cmH2O until optimal lung volume is reached, where an increase in SaO2 that allows a decrease in FiO2 to < 60%; CXR shows the diaphragms are at T-9.
• If starting with a frequency too high, unacceptable CO2 levels may result and suggest that the 3100B will not work.
• Remember that the PaCO2 may initially climb for many patients before it stabilizes and begins to come back down.
Amplitude Selection

• Start at Power of “4” and adjust until the “wiggle” extends to the mid level of patient’s thigh.

• Adjust Amp in increments of 5 cm H\textsubscript{2}O

• Subjectively follow the wiggle

• Objectively follow transcutaneous CO\textsubscript{2} and P\textsubscript{a}CO\textsubscript{2}

• Remember, the goal is not to achieve ‘normal’ P\textsubscript{a}CO\textsubscript{2} and pH, but to minimize VALI.
“Wiggle Factor”

• Reassess after positional changes

• If chest oscillation is diminished or absent consider:
  • decreased pulmonary compliance
  • ETT disconnect
  • ETT obstruction
  • severe bronchospasm

• If the chest oscillation is unilateral, consider:
  • ETT displacement (right mainstem)
Clinical Assessment
Sedation/Neuromuscular Blockade

- Transitioning a patient from CMV to HFOV typically indicates that the patient’s respiratory distress has worsened.

- To facilitate ‘capturing’ the patient, additional sedation may be required.

- Neuromuscular blockade may be required.

- As the patient improves, discontinue the paralysis and wean the sedation as tolerated.
Auscultation

- Listen to the lung fields to primarily assess the presence and symmetry of piston sounds.

  - Asymmetry may indicate improper ETT placement, pneumothorax, heterogeneous gross lung disease, or mucus plugging.

- Pause the piston to perform a cardiac exam and assess heart sounds.

  - With the piston paused you have placed the patient in a CPAP mode and will have maintained P$_{aw}$.
Chest Radiographs

- Typically obtain a chest radiograph 1 hour after initiating HFOV and then Q12-24 hours.

- Assess
  - ETT placement
  - Rib expansion (goal is 9 ribs)
  - Pneumothorax / airleak syndrome
  - Change in lung disease
Suctioning

• Indications:
  • Routine suctioning to ensure the ETT remains patent
    • Frequency of suctioning varies by institution.
    • Our policy is every 12 to 24 hours and prn.
  • Decreased/absent wiggle
    • Possibly from mucus plugs/secrections
  • Decrease in $S_pO_2$ or transcutaneous $O_2$ level
  • Increase in transcutaneous $CO_2$ level
  • Suctioning de-recruits lung volume
    • May be minimized but not fully eliminated with closed suction system.
    • May require a sustained inflation recruitment maneuver following suctioning.
Sustained Inflation (LRM)

- A sustained inflation is a lung recruitment maneuver.

- There are several ways in which to perform a LRM maneuver.

  - The piston is paused (thus leaving the patient in CPAP) and the $P_{aw}$ is increased by 8-10 cm H$_2$O for 30-60 seconds.

  - Once the LRM maneuver is completed, the piston is restarted.

- Potential complications:
When To Utilize A LRM Maneuver

- When initiating HFOV to recruit lung
- After a disconnect or loss of FRC/P$_{aw}$
- After suctioning (even with a closed suction system)
- Inability to wean F$_{iO_{2}}$
- When considering increasing P$_{aw}$

- A recruitment maneuver may recruit lung allowing you to maintain the baseline P$_{aw}$ and, thus, not
Potential Complications of HFOV

- The higher intrathoracic pressures with HFOV may decrease RV preload and require volume administration ± inotropic support.

- Pneumothorax

- Migration/displacement of ETT

- Bronchospasm

- Acute airway obstruction from mucus plugging, secretions, hemorrhage or clot.
Clinical Assessment

- Chest x-rays
  - Obtain the first x-ray at the (1) hour mark to determine the lung volume at that time. Paw may need to be re-adjusted accordingly.
  - Always obtain a CXR, if unsure as to whether the patient is hyper-inflated or has
Clinical Assessment

- Chest x-rays
  - Stopping the piston is not necessary.
  - Do not remove the patient from HFOV.
  - A respiratory therapist, nurse of physician should be at bedside to assure the security of the airway and the patient’s position.
Clinical Assessment

- Chest Wiggle factor (CWF) must be evaluated upon initiation and followed closely after that.
  - CWF absent or becomes diminished is a clinical sign that the airway or ET tube is obstructed.
  - CWF present on one side only is an indication that the ET tube has slipped down a primary bronchus or a pneumothorax has occurred. Check the position of the ET tube or obtain a CXR.
  - Reassess CWF following any position change.
Clinical Assessment

- Cardiac Function:
  - Mean Arterial Pressure
  - Pulse Pressure
  - Heart rate
  - Capillary re-fill
  - CVP
  - Swan-Ganz
  - ECHO for cardiac function
Clinical Assessment

- **Auscultation**
  - Heart sounds may be heard by stopping the piston
  - GI sounds may be heard by stopping the piston for a brief moment
  - Lung sounds may be heard by stopping the piston if patient is breathing spontaneously.

Caution: Eliminate disconnects to listen to the lungs to prevent de-recruiting the lung.
Patient Care

• Suctioning
  - Indicated by decreased or absence CWF, decrease in O2 saturation, or an increase in TcCO2.
  - Remember that each time the patient is disconnected from HFOV, they will potentially de-recruit lung volume.
  - Closed suction catheters may mitigate de-recruitment, and you may have to adjust the delta P to compensate for the attenuation of the delta P due to the right angle adapter.
  - It may be necessary to temporarily ↑ Paw
Suctioning

If using a closed suction catheter remember to:

- Remove the suction catheter all the way from the ET tube.
- Use both oximetry and TcCO$_2$ to monitor the patient for potential disconnects. If the patient disconnects
Patient Care

• **Bronchodilator Therapy**
  - Patients who are actively wheezing or have RAD
  - Administration via bagging—try to coordinate with suctioning
  - IV terbutaline for patients who do not tolerate disconnects
Patient Care

• May turn periodically
  • Protect the airway when turning the head
  • May lay on back, stomach, or side
  • May lay on any surface with the exception of an air mattress, i.e., gel, sheepskin, etc.
• Caution turning frequently if you have to disconnect from ventilator initially
Lung Recruitment with HFOV

PCV (mPaw 30)

HFOV (mPaw 35)

Tip: decreasing $\Delta P$ (at constant power setting) may indicate improving lung volume
Take Home Messages

- Ventilation Strategies *do* affect patient outcomes
- Volume and pressure swings promote lung injury and mediator release
- Identify patients at risk for developing VILI early
  - Before the fibroproliferative stage of ARDS
- Alternative support such as HFOV offers lung protection that may improve outcomes for patients with ARDS
Summary

• Treatment of ARDS continues to evolve
• The study and understanding of contributing factors to ARDS continues
• Past development and clinical study of HFOV has given a good foundation to move forward
• HFOV has been shown to be effective tool in treatment of ARDS

YOUR ACTIONS ALWAYS INFLUENCE SOMEONE!!
Questions??

Feel free to contact me at any time

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HFOV Patient Care