HIGH FREQUENCY OSCILLATORY VENTILATION:

Clinical Management Strategies for Adult Patients

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With increased understanding of the physiology of lung recruitment and the adverse consequences of ventilator induced lung injury (VILI) it has been hoped that improved ventilator strategies will reduce the morbidity and mortality of this common cause of respiratory failure (1). The NIH ARDS network clinical trial recently reported that a conventional ventilator strategy employing a low tidal volume "lung protective" approach [tidal volumes (TV) < 6 ml /kg ideal body weight, inspiratory plateau pressures (Pplat) < 30 cm H_2O] reduced absolute mortality 9% compared with a larger tidal volume (12 ml/kg) strategy (2). Unfortunately, in practice, many critically ill patients with ARDS are unable to achieve oxygenation goals using conventional protective lung approaches (arbitrarily defined as $FiO_2 \leq 60\%$ with Pplat ≤ 30 cmH_2O) and the mortality from ARDS remains unacceptably high. In view of the successful use of high frequency oscillatory ventilation (HFOV) in neonatal and pediatric acute respiratory failure syndromes in the 1980's and early 1990's we became interested in the potential application of this ventilatory mode for adults with severe ARDS. The optimal specific techniques of using HFOV in large patients are continuing to evolve and as with all interventions in critical care, frequent reassessment of the patient and modification of therapeutic strategies as the patient's condition changes is essential.

1. Clinical Trials of HFOV in Adults

In the early 1990's, our group at Wilford Hall Medical Center conducted a non-randomized pilot trial using the SensorMedics 3100B ventilator in adult patients with severe ARDS who were failing conventional ventilation (3). In this trial, 17 adult patients with severe ARDS (mean PaO_2/FiO_2 67 mmHg on 18 cmH_2O PEEP) were treated with HFOV using an "open lung" strategy. Despite the severity of ARDS, 30-day survival was 47%. As had been seen in previous ARDS trials, the PaO_2/FiO_2 ratio was not predictive of survival, however, prior days on conventional ventilation (CV) (e.g. 2.5 days in survivors versus $\geq$ 7 days in nonsurvivors) and the oxygenation index (OI = mPaw X FiO_2/PaO_2) appeared to be predictive (baseline mean OI = 34 in survivors versus OI = 60 in nonsurvivors) of mortality. These findings suggested that earlier intervention in the course of ARDS with HFOV might be more beneficial. In this initial experience with HFOV we attempted to achieve an "open lung" by applying higher mean airway pressure (mPaw) than was used on CV (4). Despite the higher mPaw applied, there was no significant compromise in cardiac output or oxygen delivery. In view of the encouraging results seen with the adult pilot study, a multicenter randomized, controlled trial of oscillatory ventilation in ARDS (MOAT) was initiated in 1997 and completed in December 2000 (5). In this trial, adult patients with ARDS ($PaO_2/FiO_2 < 200$ mmHg on PEEP ≥ 10 cm H_2O) were randomized to HFOV (N= 75) or a conventional pressure control strategy (N= 73). Although this study was not specifically powered to evaluate mortality differences, we observed a nonsignificant trend towards improved overall mortality at 30-days in the HFOV group compared with the CV group (37% versus 52% 30-day mortality,

respectively, p=0.098). This trend toward improved mortality with HFOV was still evident at 6 months (6 month overall mortality rate 47% in the HFOV group and 59% in the CV group, p=0.143). There was no significant difference between groups in new or worsening barotrauma, endotracheal tube obstruction, or adverse hemodynamic effects.

In a retrospective analysis of pretreatment indicators of survival, only peak inspiratory pressure (< 38 cmH2O), but not pre-study days on CV, P/F, or OI showed a significant difference in mortality between HFOV and CV (30-day mortality 26% in the HFOV group versus 52% mortality in the CV group, p=0.018). Although this was a post-hoc finding, it suggests that earlier application of HFOV (when the lung is more compliant) may improve the outcome. Most recently, an additional nonrandomized study of HFOV in 24 adults with ARDS has been published by the Toronto group (6). Patients entered into this study had severe ARDS (mean PaO2/FiO2 98 mmHg, OI 32) and required ≥ 60% FiO2, or Pplat ≥ 35 cmH2O on CV before starting HFOV. Again noted was the finding that³survivors were on CV for significantly fewer days prior to starting HFOV (survivors 1.6 days versus 7.8 days in nonsurvivors). Overall mortality at 30-days was 67%.

2. Technique Of HFOV In Large Patients

When to initiate HFOV

Patients with severe ARDS who are requiring an FiO2 ≥ 60% with a mPaw > 24 may be considered for a trial of HFOV – especially if a "lung protective" target Pplat < 30 - 35 cmH2O cannot be maintained. In practice, patients considered for HFOV have generally already been tried on "high" PEEP (e.g. 20 – 25 cmH2O) and/or pressure control ventilation with extended inspiratory times to raise mean airway pressure. At this point, patients are usually already deeply sedated and on neuromuscular blockade to facilitate oxygenation. We feel that a mPaw ≥ 24 cmH2O while on conventional ventilation is a reasonable threshold at which to consider changing to HFOV because adult patients on HFOV can generally be transitioned back to conventional ventilation when the mPaw on HFOV has been weaned to this level (assuming FiO2 has been weaned to 40 – 50% first). Early institution of HFOV in patients deteriorating on CV may be important to improved survival. In both the Wilford Hall and Toronto non-randomized studies, prolonged time on CV (e.g. > 7 days) predicted a worse outcome.

Preparing to initiate HFOV

Prior to starting a patient on HFOV it is imperative that the patient's airway is suctioned and known to be patent. If bronchoscopy is contemplated (e.g. to evaluate for opportunistic infection) it should be performed prior to initiating HFOV. This will also allow direct visualization of the airway to document the degree of patency of the endotracheal tube. Narrowing or obstruction of the endotracheal tube with mucus or blood clots may greatly impede delivery of the oscillatory waveform and make ventilation difficult.

Adequate titration of sedation, analgesia, and neuromuscular blockade should be performed while the patient is still on conventional ventilation. The patient's intravascular volume status should be reassessed keeping in mind the higher mPaw that will be used with HFOV and the potential for hypotension secondary to elevated intrathoracic pressures and reduced preload.

3. Initial HFOV Settings

Oxygenation

The main determinant of oxygenation during HFOV is the mPaw, which is generally initiated at 5 cmH$_2$O higher than the mPaw noted during conventional ventilation. Hemodynamically unstable patients may be started on a mPaw either the same or 2 – 3 cmH$_2$O above mPaw during conventional ventilation. Brief hypotension shortly after starting HFOV is usually managed with a trial of fluid boluses to improve preload. Whether patients should be started on even higher mPaw (a "sustained inflation" as an alveolar recruiting maneuver) such as 40 cmH$_2$O for 40 – 60 seconds when HFOV is first initiated is not yet clear (7). If the patient requires suctioning for gross secretions visible in the endotracheal tube or has a disconnect from the circuit, we favor doing a brief sustained inflation in the hopes of re-inflating areas of atelectasis that may have occurred even with short periods of suction or circuit disconnection. FiO$_2$% is usually set at 100% after the transition to HFOV, and then tapered using oximetry guidance to maintain SpO$_2 \geq$ 88%. If the SpO$_2$ (or PaO$_2$) has not improved enough to allow weaning of FiO$_2$%, the mPaw is raised in 3-5 cmH$_2$O increments at 30 – 60 minute intervals in the hopes of improving lung recruitment. The time course of oxygenation change after initiation of HFOV (or after a given increase in mPaw) is quite variable. Some patients may slowly improve oxygenation only after a period of several hours. Vigilance and patience are required during the early phase of treatment.

The maximum mPaw we can generally obtain with the SensorMedics 3100B is 45 – 55 cmH$_2$O. Patients with large bronchopleural fistulas or endotracheal cuff leaks may have difficulty achieving a desired mPaw without increasing the bias flow. In some patients with very severe airleaks we have had to use maximum bias flow (60 lpm) on the oscillator.
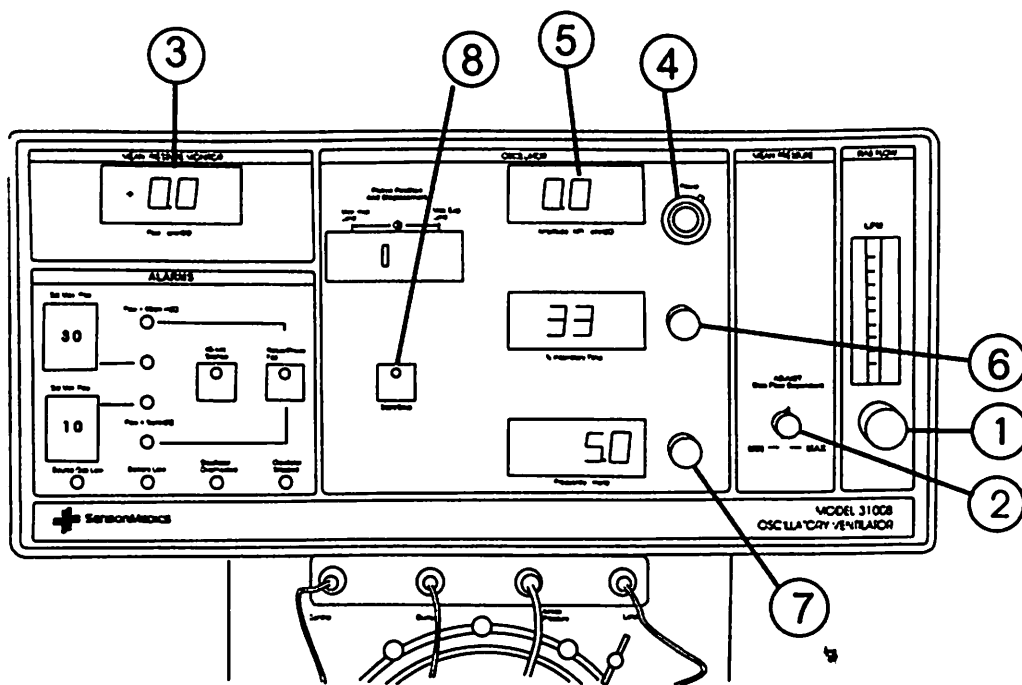
Figure 1: 3100B Front Panel Controls

3100B Front Panel Controls

1. Bias Flow

2. Mean Pressure Adjust

3. Mean Airway Pressure

4. Power

5. Amplitude (Delta P)

6. % Inspiratory Time

7. Frequency

8. Start / Stop

Ventilation

The main determinants of $PaCO_2$ elimination are the pressure amplitude of oscillation (ΔP) and the frequency setting (hertz). Increasing the ΔP and decreasing the frequency (Hz) increase delivered tidal volume and lower $PaCO_2$. Conversely, decreasing ΔP and increasing frequency (Hz) will reduce delivered tidal volume and allow $PaCO_2$ to rise. The ΔP is generally initiated at either a value where the patient's chest vibrates down to their mid-thigh or in our experience, a value of 20 – 30 cmH_2O above what the patient's $PaCO_2$ was on CV (e.g. $PaCO_2$ of 60 mmHg would mean starting the ΔP at 80 cmH_2O). Initial frequency is usually set at 5 Hz. Patients who demonstrate rapidly rising $PaCO_2$ on HFOV should have aggressive increases in ΔP (10 to 20 cmH_2O) and reduced Hz to the lowest value achievable (3 Hz on the SensorMedics 3100B). An adjunct to improving $PaCO_2$ elimination is to briefly disconnect the patient from HFOV and vigorously manually ventilate the patient with a PEEP-valve equipped resuscitation bag. Aggressive action is required for a rapidly rising $PaCO_2$ during initiation of HFOV because improvements in $PaCO_2$ do not occur as quickly as is noted when changes are made to conventional ventilators. We obtain an ABG 15 to 20 minutes after starting HFOV to determine the trend of the $PaCO_2$. Subsequent ABG's are generally obtained at 30 to 60 minute intervals until stabilization occurs.

An interesting observation in some patients with hypercapnea is that a deliberately induced cuff leak may improve the $PaCO_2$. The presence of a cuff leak may allow the elevated end-tidal PCO_2 in the airway to be "washed-out" by the bias flow gas – much like occurs with tracheal insufflation of gas at the carina with small caliber airway catheters (8). In practice, if a patient remains with severe hypercapnea (causing a pH < 7.2) despite maximum ΔP and lowest frequency settings, we will induce a small endotracheal tube cuff leak by withdrawing air from the cuff pilot balloon sufficient to lower the mPaw about 5 cmH_2O. The mPaw is then readjusted to its previous value while maintaining the cuff leak. Using this technique we have seen occasional marked reductions in $PaCO_2$ over a short time (20 – 30 minutes).
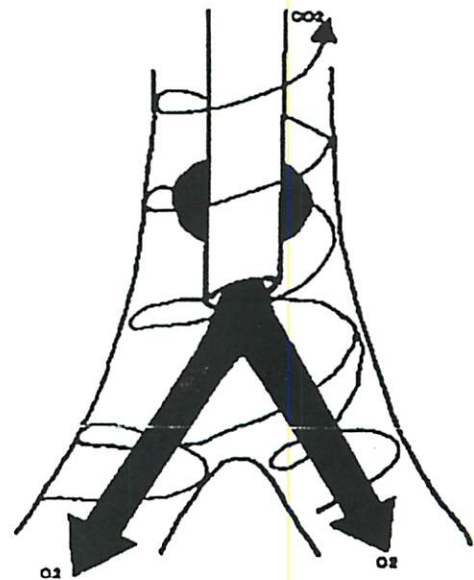
Patient Positioning

Hemodynamically stable patients are generally positioned with the head of the bed elevated approximately 30 degrees. Care must be taken with the patient's head and neck positioning to ensure a smooth interface (free of kinks) between the endotracheal tube and oscillator circuit. This often requires propping the oscillator circuit on folded towels near the endotracheal tube interface. Prone positioning has been utilized successfully in adult patients on HFOV with demonstrated improvement in oxygenation (9). As with all patients who are mechanically ventilated in the prone

position, a team of physicians, nurses, and respiratory therapists should be present to ensure that repositioning occurs safely without dislodging tubes and lines. If prone positioning is anticipated, it should ideally be accomplished prior to initiating HFOV. Because of the relatively inflexible oscillator circuit, the patient may have to be manually ventilated with a resuscitation bag while turning during HFOV. High vigilance for mucus plugs and excessive airway secretions should be maintained shortly after turning from prone to supine positions.

4. Complications During HFOV

Hypotension

Occasionally, patients will develop hypotension shortly following transfer to HFOV or as mPaw is raised. This usually implies relative hypovolemia and responds to intravenous fluid boluses. It is important to keep in mind that relatively "high" CVP or PCWP (e.g. mid to high 20's) may indicate the patient is still hypovolemic because of the effect of the elevated airway pressures being transmitted to the measured intravascular pressures. This is especially true as mPaw exceeds values of 30 – 35 cmH20. If the patient is hypotensive, we will still generally administer serial fluid boluses (e.g. 500 ml normal saline or packed RBC's if anemic) until the CVP or PCWP has clearly increased by 5 – 10 mmHg. At that point, if hypotension persists we add vasopressors (e.g. dopamine, epinephrine, norepinephrine) and reconsider the differential diagnosis of the hypotension.

Pneumothorax

It is important to realize that tension pneumothorax occurring during HFOV may not cause changes to the displayed mPaw or ΔP as the patient develops progressive hypotension and desaturation (10). A high index of suspicion is necessary for pneumothorax and confirmation (if time permits) requires an immediate portable chest radiograph. If in doubt, it is preferable to place a chest tube on the suspected side while waiting for the chest radiograph. It may be difficult by auscultation alone to detect the side of the pneumothorax - especially during HFOV because of the background noise of the ventilator and the diffuse transmission of airway sounds, however, the loss of chest wiggle that usually occurs on the affected side will provide an important physical clue. If a previously attempted central venous line (either internal jugular or subclavian vein) or thoracentesis is apparent, we usually will empirically place the chest tube on that side if time does not permit radiographic confirmation of pneumothorax. We discourage placing 14 gauge needles into the anterior thorax for suspected pneumothorax. If a pneumothorax was not present, the needle insertion has now likely caused one and will need immediate follow-up with a chest tube. It is always preferable to place a chest tube with direct palpation of the underlying lung to confirm placement of the tube in the pleural space. If a pneumothorax was not present then at least a bronchopleural fistula has not been created iatrogenically. To date there has been no convincing clinical evidence that the frequency of pneumothorax in adults is less or greater with HFOV compared with conventional ventilation. Several case reports and small series indicate that HFOV may provide successful oxygenation and ventilation in the presence of large bronchopleural fistulas and this has been our experience as well.

Endotracheal Tube Obstruction

Subtotal occlusion of the endotracheal tube or airway may result in refractory hypercapnea. An abrupt rise in $PaCO_2$ during HFOV in an otherwise stable patient should be considered an obstructing or narrowed endotracheal tube until proven otherwise. If this is suspected, a suction catheter should be passed immediately to ensure patency of the endotracheal tube. Urgent bronchoscopy can then be performed either during HFOV or with a brief interruption of HFOV during manual bagging to visually inspect the airway. We have seen cases of endotracheal tubes obstructed by blood clots or mucus causing a "ball" valve effect that allowed passage of a suction catheter but would not allow exhalation to occur. This diagnosis can only be made by bronchoscopy. A sudden increase in ΔP (with no change in mPaw) may indicate a mainstem intubation or increase in airway resistance. A drop in mPaw (with no significant change in ΔP) may indicate a new or worsening airleak (10).

5. Sudden Stoppage of the HFOV

Another aspect of HFOV is that there is no backup conventional ventilation mode. The oscillator may suddenly stop if decompression of the circuit occurs. This is usually because a pressure-regulating mushroom valve or other external connection has become loose or disconnected. For this reason, it is imperative that a resuscitation bag with PEEP valve and attached oxygen source is always positioned at the head of the patient for immediate use. Should the oscillator suddenly stop, the patient should be immediately disconnected from the circuit and manually bag-ventilated while trouble shooting the ventilator.

6. Weaning From High Frequency Oscillatory Ventilation

The principle goal of using HFOV in treating patients with ARDS is to achieve a nontoxic FiO_2 (<60%) while minimizing ventilator induced lung injury. When patients respond with improved oxygenation, the first weaning maneuver therefore is to reduce the FiO_2 before any reduction is considered in mPaw. We attempt reduction of FiO_2 to 40% with a target SpO_2 > 90% before attempting reductions in mPaw. If the patient can maintain a SpO_2 > 90% on FiO_2 40% we will than start a gradual reduction of mPaw (e.g. decrease 2 – 3 cmH_2O q 4 – 6 hours as tolerated). If the SpO_2 decreases during mPaw reduction, we will try to resume the previous mPaw that was able to maintain a SpO_2 > 90% on FiO_2 40%. It is important not to decrease mPaw too rapidly in an attempt to get the patient off HFOV. If the lung derecruits and desaturation occurs, it can take many hours to regain the lost volume. In our experience, attempting reductions in mPaw while the required FiO_2 is still at 50 – 60 % will more often result in desaturations requiring re-institution of higher mPaw settings. Once a mPaw of 20 – 24 cmH_2O has been achieved while maintaining an FiO_2 of 40%, the patient can be switched back to a trial of conventional ventilation.

We typically set up the conventional ventilator to achieve a mPaw of 20 +/- 2 cmH_2O by using pressure control mode (PCV) with peak pressure set to achieve a delivered tidal volume of 6 – 8 ml/kg predicted body weight and inspiratory plateau pressure < 30 – 35 cmH_2O. In determining the appropriate peak pressure setting it is important to titrate pressure based on delivered inspiratory tidal volume. For this reason we

favor using ventilators that display both delivered and exhaled tidal volumes in the pressure control mode. Titration of pressure control based on exhaled tidal volume alone in our experience often leads to overestimation of the peak pressure required – especially in patients with cuff leaks or bronchopleural fistulas. The conventional ventilator is initially set up using PCV with an I:E ratio of 1:1 and 12 cmH_2O PEEP with a respiratory rate of 20 - 25. The mean airway pressure, inspiratory plateau pressure, and delivered tidal volume are immediately checked when the patient is changed to the conventional ventilator and small adjustments to PEEP or pressure are made to ensure that the mPaw is within 2 -3 cmH_2O of what it was on HFOV. When the mean airway pressure is closely matched to what it was on HFOV we have found that most patients are easily transferred to conventional ventilation and are able to maintain a SpO_2 > 90% on the same FiO_2. An arterial blood gas is obtained 15 - 20 minutes after transfer to conventional ventilation to guide further ventilator adjustments.

7. Other Supportive Issues During HFOV

Sedation and Paralysis

During HFOV, the patient is kept deeply sedated with a combination benzodiazepine (e.g. lorazepam, midazolam), and narcotic (e.g. fentanyl, morphine) infusion. Prior to initiation of HFOV, the patient is also pharmacologically paralyzed with a long acting neuromuscular blocker (e.g. cis-atracurium). Many patients are already paralyzed on conventional ventilation prior to being placed on a trial of HFOV and if so, the paralysis is continued. A nursing protocol for care of the paralyzed patient is essential to ensure comprehensive care of the patient (e.g. skin care, ankle splints, mouth care, etc). It is important to recognize that sinus tachycardia or hypertension developing during HFOV may indicate inadequate pain relief or sedation in a paralyzed patient. As patients are weaned from HFOV and are approaching transfer back to conventional ventilation we have observed that continued neuromuscular paralysis may not be necessary. As the mPaw is approaching 20 – 24 cmH_2O, we have tried discontinuing the paralytic agent while continuing sedation and analgesia with success. Once stabilized on HFOV, some patients may be managed with sedation and analgesia alone. Typically, patients have not required continued paralysis after transfer back to pressure control ventilation with an I:E ratio of 1:1. We have also substituted propofol for the benzodiazepine as an adjunct to sedation as weaning is progressing. Propofol may allow quicker "wake-up" times to permit trials of spontaneous breathing on pressure support ventilation during the final phase of weaning from conventional ventilation. Nevertheless, one may anticipate prolonged sedation and muscle weakness after many days or weeks of benzodiazepine and neuromuscular blocker drug infusions that may limit final extubation even when oxygenation is no longer an issue.

Suctioning and Bronchoscopy

Tracheal suction should be done prior to initiation of HFOV. During the early hours and days of HFOV we try to limit interruption of HFOV to perform suctioning unless there are gross secretions in the airway or evidence of atelectasis on chest radiograph. Tracheal suction lowers carinal pressures and may allow alveolar derecruitment to occur - usually manifested as a lower SpO_2 or requirement for a higher FiO_2. Additionally, placement of an inline right-angle suction adapter in the HFOV circuit may impair the delivered waveform and impair ventilation. For this reason, we recommend attaching the HFOV circuit directly to the endotracheal tube (without an inline suction adapter) until it is clear that adequate $PaCO_2$ elimination can occur. If required, bronchoscopy can be performed during HFOV through a right angle adapter or during a brief interruption of HFOV. In unstable patients requiring high FiO_2, the duration of the bronchoscopy should be as brief as possible.

Deep vein thrombosis prophylaxis

Patients on HFOV are deeply sedated and pharmacologically paralyzed during the initial phases of their illness. The lack of mobility puts these patients at increased risk for thromboembolism. For this reason, we aggressively use pneumatic boots, compression stockings, and subcutaneous low dose heparin if no contraindications exist (e.g. intracranial hemorrhage, recent spinal surgery). We favor the use of prophylactic dose low molecular weight heparin (e.g. enoxaparin 30 mg SQ bid or 40 mg SQ q day) since it has a reportedly lower incidence of heparin-induced thrombocytopenia. In addition to aggressive attempts at prophylaxis for deep vein thrombosis we also use lower extremity ultrasound screening every 72 to 96 hours to look for clot development that would require full anticoagulation or caval filter placement.

8. Summary

HFOV is an important alternative method of mechanical ventilation for severe ARDS in large patients. It should be considered for use in patients requiring high mPaw ($\geq$ 24 cmH_2O) on conventional ventilation – especially if the FiO_2 requirements exceed 60% and Pplat is unable to be maintained $\leq$ 30 – 35 cmH_2O. Failure to improve the oxygenation index within the first 24 – 48 hours is indicative of a poor response to HFOV. In our experience, nonresponders to HFOV have an extremely high mortality from ARDS – approaching 100%. Late phase fibroproliferative ARDS, when the alveolar architecture is severely damaged, is less likely to respond. Adjunctive therapies that are currently being investigated in combination with HFOV include prone positioning, nitric oxide, aerosolized prostacyclin, surfactants, and partial liquid ventilation. All of these adjunctive therapies have been demonstrated to improve oxygenation, however, whether mortality or ventilator-free days in adults will be improved awaits demonstration by randomized, controlled trials.

9. Guidelines for HFOV 3100B in ADULTS

1. Set initial mPaw at 5 cmH_2O above conventional ventilator mPaw (consider initial alveolar recruiting maneuver with 40 cmH_2O for 40 – 60 secs if severe hypoxemia).

2. Set power to achieve initial ΔP at chest wiggle to mid-thigh or "20 + $PaCO_2$".

3. Set Hz at 5.

4. Set IT to 33% (may increase to 50% if difficulty with oxygenation; this may further raise carinal pressure an additional 2 – 4 cmH_2O).

5. If oxygenation worsens, increase mPaw in 3 – 5 cmH_2O increments Q 30 minutes until maximum setting (approximately 45 – 55 cmH_2O).

6. If $PaCO_2$ worsens (but pH > 7.2), increase ΔP in 10 cmH_2O increments Q 30 minutes up to maximum setting. After maximum ΔP achieved, if necessary, may decrease Hz to minimum of 3 Hz.

7. If severe hypercapnea with pH < 7.2, bag patient, set maximum ΔP, Hz at 3, and try small cuff leak $\approx$(5 cmH_2O and then compensate bias flow); rule out obstruction in endotracheal tube with bronchoscopy.

8. If oxygenation improves, gradually wean FiO_2 to 40%, then slowly reduce mPaw 2-3 cmH_2O q 4 – 6 hours until 22 – 24 cmH_2O range.

9. When above goal met, switch to PCV (initial settings: peak pressure titrated to achieve delivered TV 6 ml/kg, Pplat < 30 - 35 cmH_2O), I:E 1:1, PEEP 12 cmH_2O, rate 20 – 25, mPaw should be 20 cmH_2O (+/- 2 cmH_2O).

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