High-Frequency Jet Ventilation
of
Infants and Children

J. Bert Bunnell, Sc.D.
Chairman and Chief Executive Officer
Bunnell Incorporated
436 Lawndale Drive
Salt Lake City, Utah 84108

Phone: 801-467-0811, x33
Email: Bert@bunl.com
Website: www.bunl.com

It is usually more important for clinicians to know *how* to use a ventilator rather than worrying about *which* ventilator to use. However, in some cases which ventilator is used can be extremely important. Such was the case a couple of years ago at the Broward General Medical Center in the United States (Fort Lauderdale, Florida).

A previously healthy 7-year old girl was swinging on a bar supported by a concrete partition at a movie theater when the partition collapsed and fell on her. She suffered a severe crush injury to the upper chest with severe bilateral lung contusions and pneumothoraces. In surgery, extensive tearing of the membranous portion of the trachea involving both left and right mainstem bronchi were discovered. After prolonged and complicated surgery to repair the airway, the child proved to be extremely difficult to ventilate. [1]

Initial ventilation with high frequency oscillation (SensorMedics 3100A) was unsuccessful. The patient's $PaCO_2$ rose to 285 torr, and her arterial pH dropped to 6.65. Conventional ventilation (Seimens Servo 300) using pressure-regulated volume control and pressure control at rates up to 140 breaths per minute (bpm) was not much better. The patient's $PaCO_2$ was never better than 170 torr, and her arterial pH never rose above 6.80.

Broward General clinicians had never used the Bunnell Life Pulse High Frequency Jet Ventilator (the "Jet") outside their newborn intensive care unit (NICU). However, they found they could ventilate this 25-kg child with the Jet using a 5.5 mm ID LifePort endotracheal adapter. Using the Jet's lowest rate of 240 bpm and shortest inspiratory time of 0.020 seconds to avoid gas trapping, Broward clinicians were able to reduce the patient's $PaCO_2$ to 72 torr after 1 hour and 15 minutes of HFJV. At that point, her arterial pH rose to 7.21. After two hours of HFJV using a peak inspiratory pressure (PIP) of 40 cm H_2O, the child was being hyperventilated with $PaCO_2$ = 26 torr and arterial pH = 7.52.

Weaning Jet PIP proceeded without incident over the next several days. Bronchoscopy on day 7 revealed healing of the tracheal bronchial tear without granuloma formation or presence of tracheitis. The patient's chest tubes were removed on day 10. She was extubated on day 11 and discharged home on day 16.

Why was HFJV so successful in this case?

The Life Pulse High Frequency Ventilator operates differently than all other ventilators. Inspired gas is injected into the endotracheal tube via a jet nozzle at rates between 240 and 660 bpm (4 to 11 Hz). The nozzle is imbedded in either a special endotracheal tube (Mallinckrodt "Hi-Lo Jet®" ET tube) or a special 15-mm ET tube adapter (Bunnell "LifePort®"). Both the ET tube and the adapter have two side ports, one for gas injection, and one for distal airway pressure monitoring. (The LifePort adapter is designed to estimate the pressure at the end of the ET tube using the pressure measured at the tip of the adapter and the known internal diameter and approximate length of the ET tube.) (See Figure 1.)

A small plastic "Patient Box", which contains the valve that creates the HFV breaths and a pressure transducer for monitoring airway pressure, is placed in the bed near the patient's head. (See Figure 2.) This design eliminates most of the compressible volume between the inspiratory valve and patient, so that each HFV breath has sufficient power to penetrate deep into the patient's airways.

Distal pressure monitoring enables microprocessor feedback-control of HFJV gas delivery. (See Figure 3.) The Jet's driving or servo pressure (labeled *"SERVO PRESS"* on the Life Pulse front panel) automatically rises and falls to ensure that the set PIP is delivered regardless of changes in the patient's lung mechanics. This design enables Servo Pressure to be used for patient monitoring purposes. (If Servo Pressure rises, the patient's lung compliance or airway resistance is improving. If Servo Pressure falls, the opposite is true, and the operator is advised to look for signs of atelectasis or to consider suctioning the patient, respectively.)

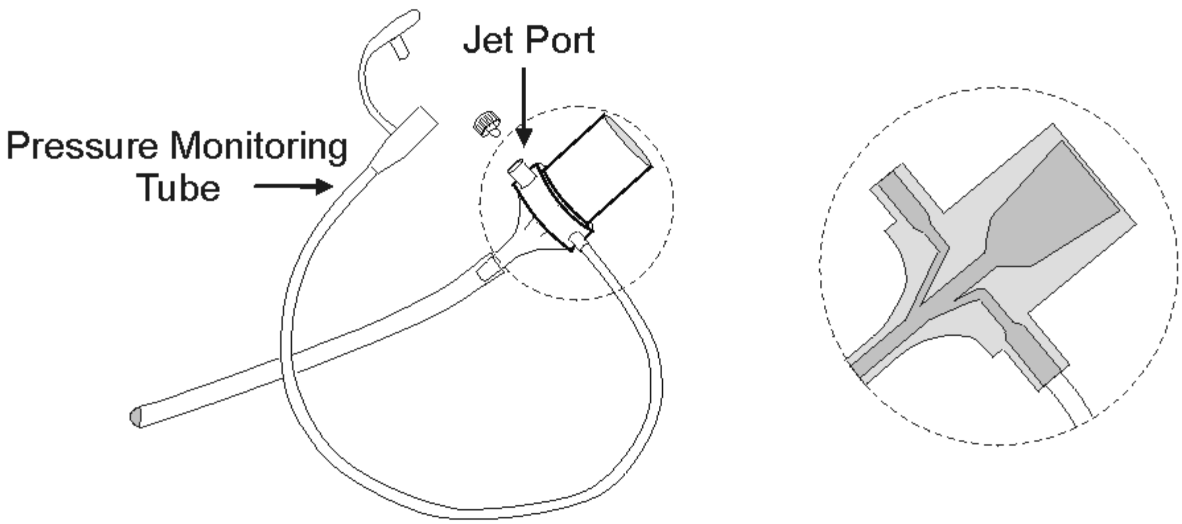

Figure 1: LifePort Endotracheal Tube Adapter

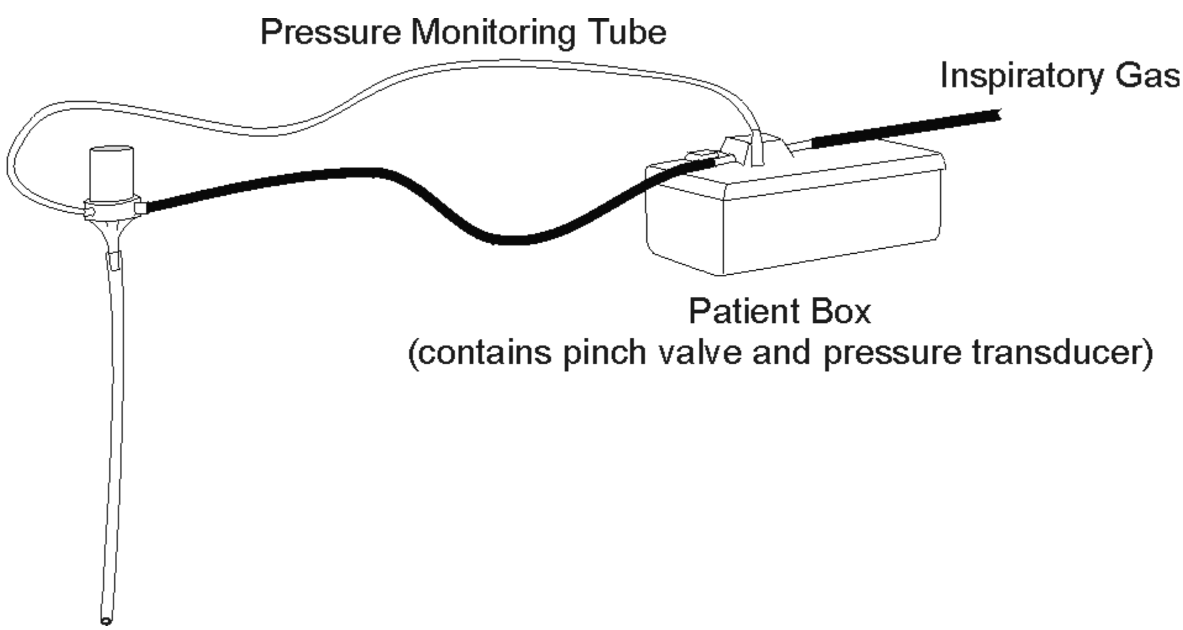

Figure 2: "Patient Box" Delivers HFJV and Monitors Airway Pressure

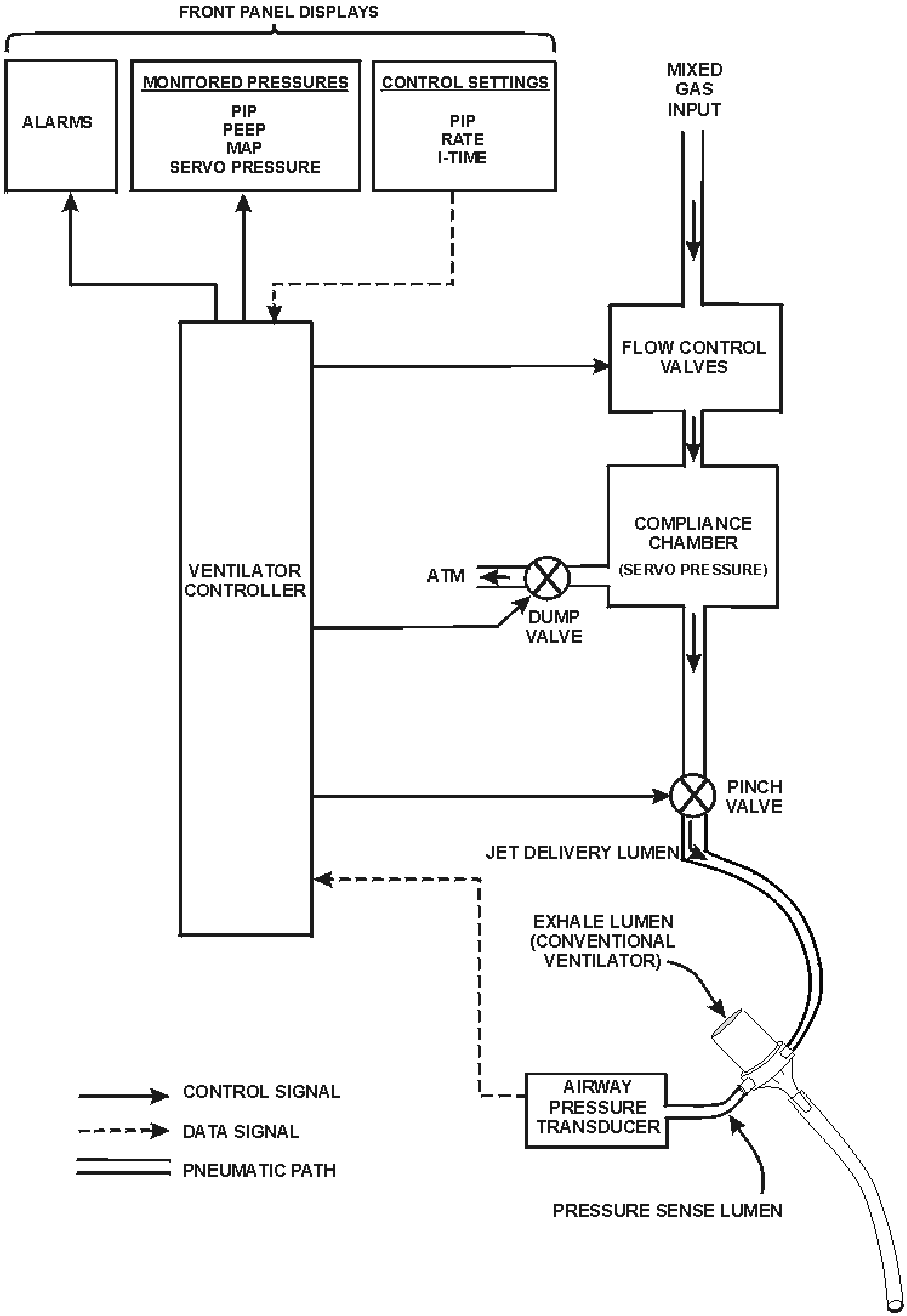

Figure 3: Feedback Control of PIP Using “Servo Pressure”

A conventional ventilator is always used in tandem with the Life Pulse. (See Figure 4.) It provides gas for the patient's spontaneous breathing, delivers occasional "sigh" breaths to recruit collapsed alveoli if atelectasis is present, or otherwise provides backup ventilation as needed.

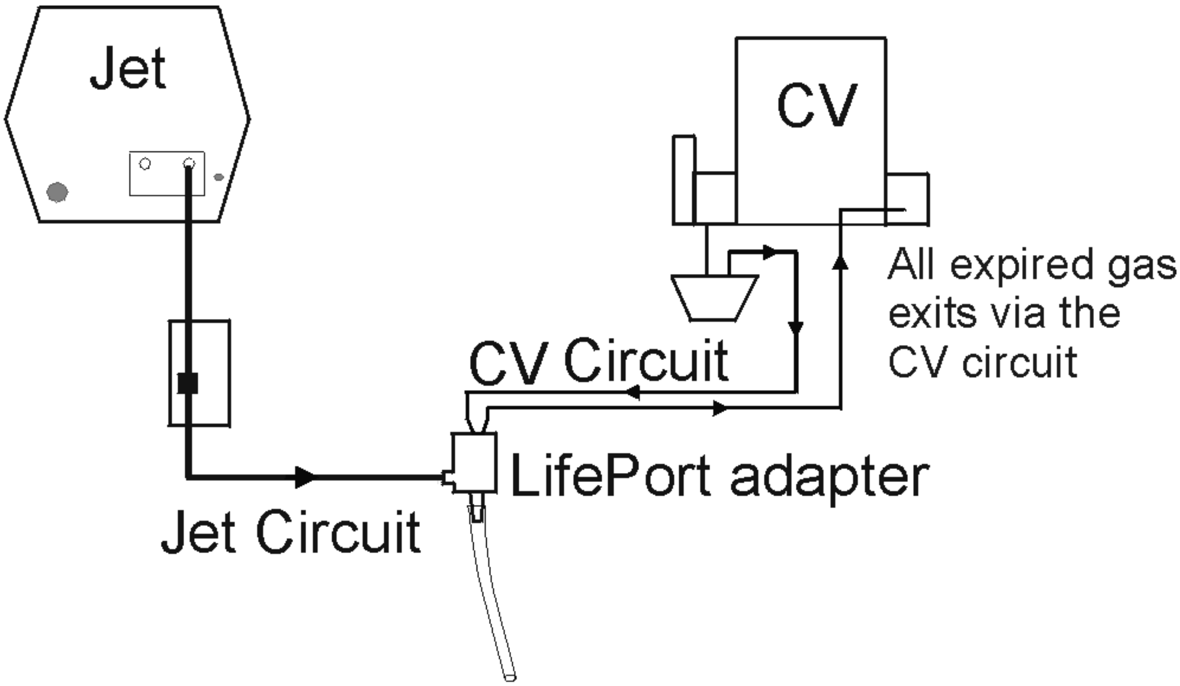

Figure 4: A conventional ventilator is always used in parallel with the Jet. It provides gas for the patient's spontaneous breathing, and it delivers occasional "sigh" breaths to recruit collapsed alveoli if atelectasis is present.

High frequency ventilators do not try to mimic normal breathing. They seek to take advantage of a phenomenon called *resonant frequency* whereby less energy (i.e., applied airway pressure) is necessary to move gas in and out of the lungs. As resonant frequency is approached, gas momentum supplies the energy to overcome lung compliance, and lung recoil supplies the energy to send gas back out of the lungs. Timing and forces interact to conserve energy, and most of the applied airway pressure is used to overcome airway resistance. Therefore, less pressure is required to move gas in and out of the lungs.

The Life Pulse squirts gas into the lungs as fast as possible, then allows passive exhalation. Inspired gas penetrates down the center core of the airways where resistance to flow is lowest, leaving considerable CO_2 in the airways at the end of inspiration. (See Figure 5.) *Effective* dead-space volume is thereby reduced since only a portion of the anatomic dead space is used. Thus, HFJV allows one to use very small tidal volumes, even smaller than anatomic dead space.

HFJV provides more effective ventilation (i.e., lower $PaCO_2$) and comparable oxygenation at mean airway pressures that are considerably less than those required during conventional ventilation. However, since smaller tidal volumes are effective with HFJV, higher PEEPs can be used safely. (See Figure 6.) Small tidal volumes also mean that lung compliance has little influence on gas distribution within the lungs.

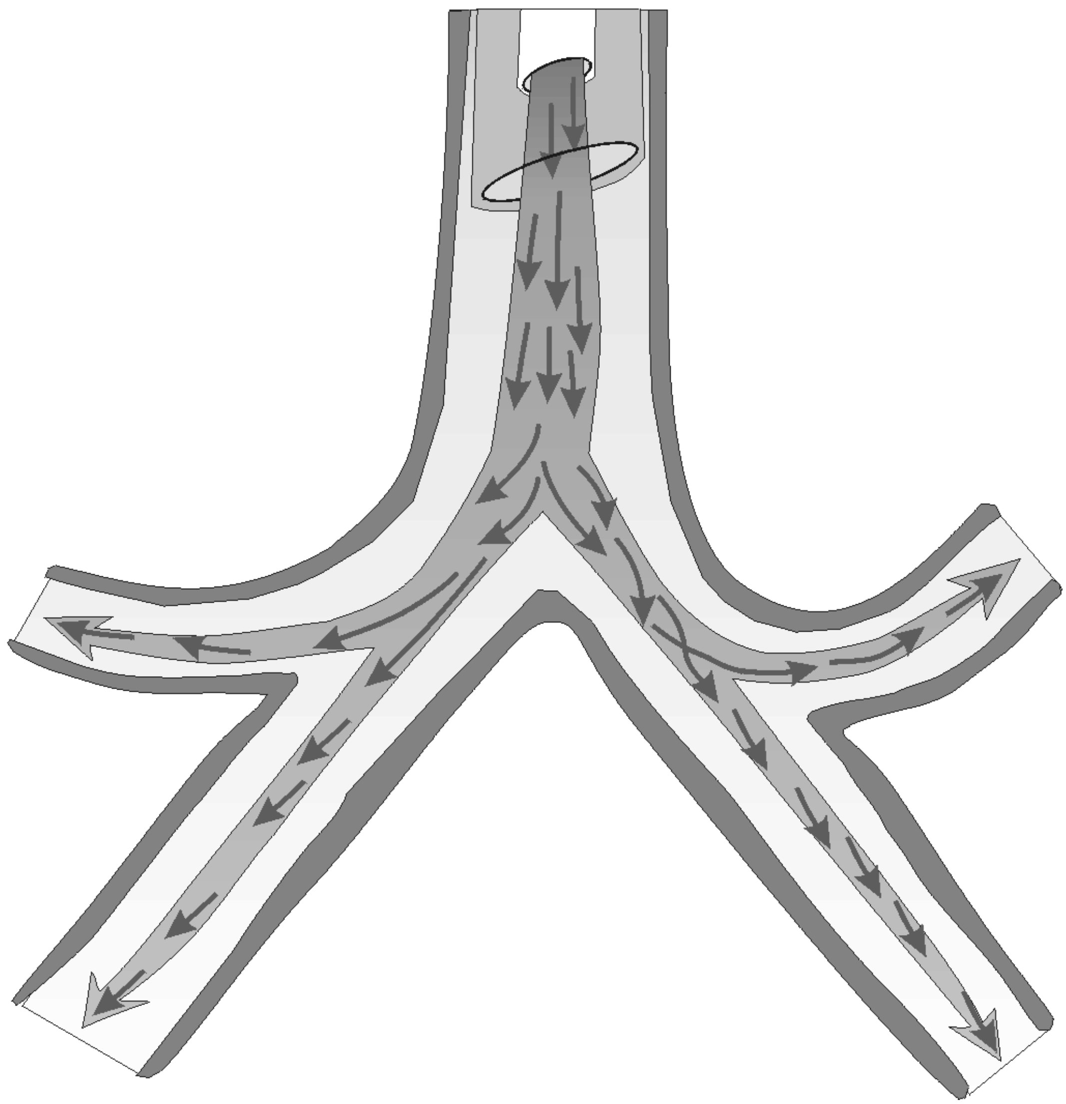

Figure 5: The Jet squirts gas into the lungs as fast as possible. The inspired gas penetrates down the center core of the airways where resistance to flow is lowest, leaving considerable CO_2 in the airways at end-inspiration. *Effective* dead-space volume is thereby reduced since only portions of the anatomic dead space are used.

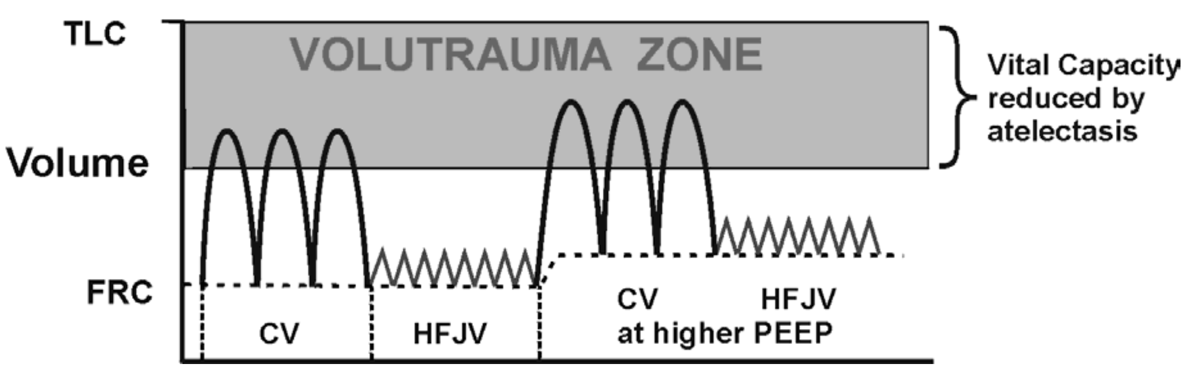

Figure 6: Since smaller tidal volumes are effective with HFJV, higher PEEPs can be used safely.

Expired gas flows out passively during HFJV, seeking its path of least resistance in the annular or "unused" spaces around the highly accelerated inspired gas. The net effect of several HFJV cycles: fresh gas advances down the core of airways while exhaled gas moves out along airway walls, which facilitates mucociliary clearance. (See Figure 7.)

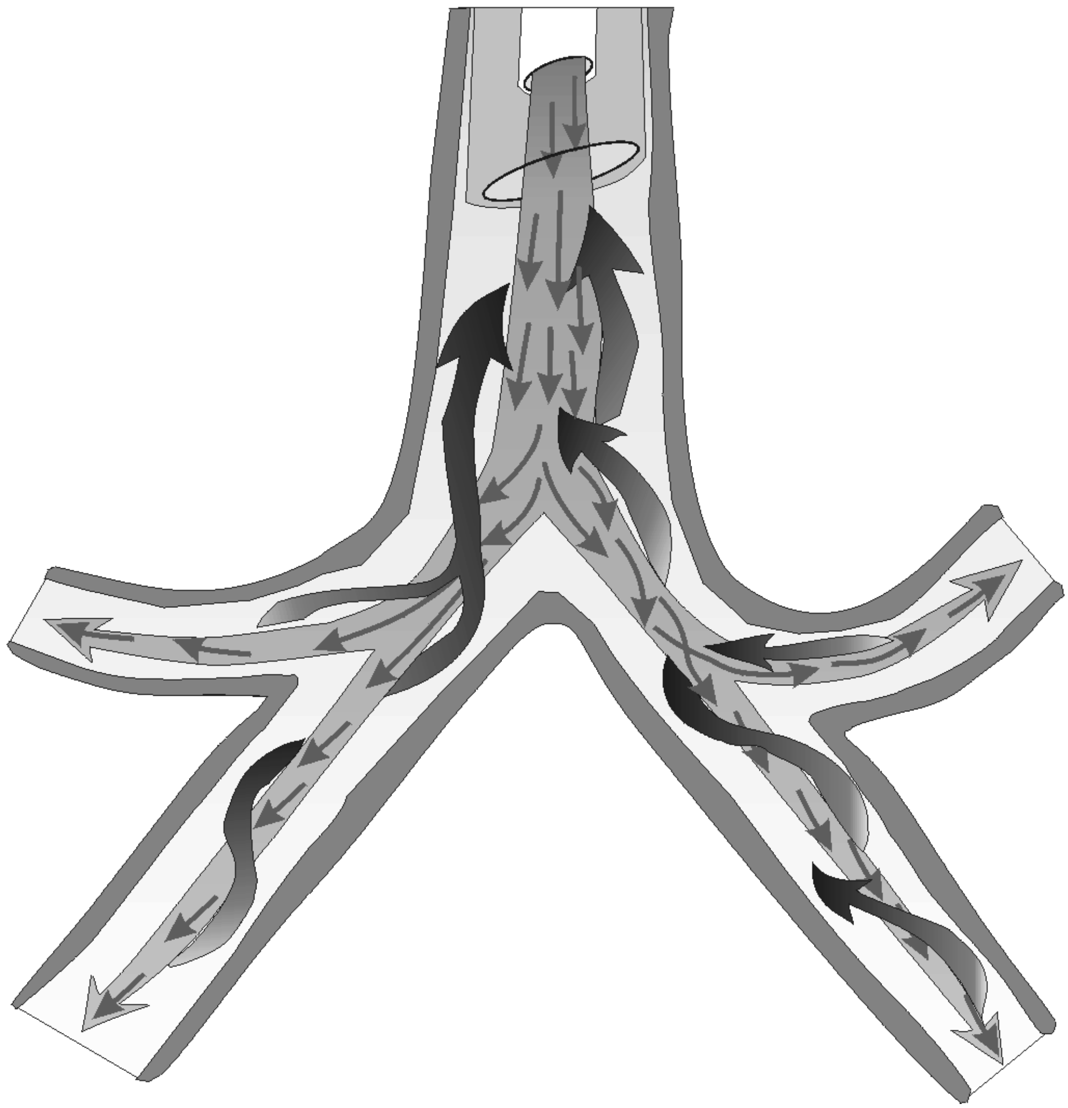

Figure 7: Expired gas flows out passively during HFJV, seeking its path of least resistance in the annular or "unused" spaces around the highly accelerated inspired gas. By moving out along the airway walls, expired gas facilitates mucociliary clearance.

The shortest possible inspiratory time usually works best with HFJV; it maximizes inspiratory velocity and minimizes I:E, which allows more time for exhalation to avoid gas trapping. These characteristics also minimize mean airway pressure, which is very useful when treating airleaks and for ventilation during and after cardiac surgery. The high velocity inspirations also enable ventilation of patients with upper airway leaks and tracheal tears, since the inspired gas shoots right past the leaks as in the above patient from Florida. Likewise, HFJV also works well for treating tracheal bronchial fistula and broncho-pleural fistula in newborn infants.

Why didn't HFOV work for this patient with a tracheal tear?

HFOV is most often used in conjunction with higher mean airway pressures. Higher mean airway pressure can stabilize surfactant-deficient alveoli, but it can be counterproductive when severe air leaks or hemodynamic problems are present.

Gas enters and exits the lungs more symmetrically with HFOV compared to HFJV because the gas is pushed in and sucked back out. Inspired gas enters slower but exhaled gas comes back out faster in HFOV compared to the "jetted" inspirations and passive exhalations of HFJV. The "active" exhalations enable faster rates to be used with HFOV without encountering gas trapping. However, this is only possible when relatively high mean airway pressure ($P\overline{aw}$) is used.

When lower mean airway pressure is used, active exhalation promotes airway collapse ("choke points") because intraluminal pressure drops faster than the pressure in the alveoli that surround the airways. Thus, more gas trapping can occur, and mean *alveolar* pressure can exceed mean *airway* pressure when the mean airway pressure is too low. [2,3,4]

Raising mean airway pressure counteracts the creation of choke points, so HFOV is most often used in conjunction with higher mean airway pressure.[5] Most hybrid high frequency ventilators, such as the so-called "flow interrupters", operate like high frequency oscillators inasmuch as they also use active expiration. So they too are most often used with higher mean airway pressures.

How is gas trapping avoided during HFJV? The shortest possible inspiratory time of 0.020 seconds is used, even with the Jet's lowest rate of 240 bpm. This short I-time maximizes inspiratory gas velocity and minimizes I:E, which allows more time for exhalation to avoid gas trapping. If gas trapping is occurring during HFJV, the monitored PEEP on the front panel of the Life Pulse will be higher than the set PEEP on the conventional ventilator. A *High Mean Airway Pressure* alarm may also be activated if gas trapping builds up slowly over time.

It is also important to remember that conventional ventilator breaths, when delivered in tandem with HFJV, can also lead to gas trapping and inadvertent PEEP. Although intermittent mandatory ventilation (IMV) breaths can be very helpful for oxygenation at times (see below), they can also create lung injury, as we all know.

How does one know where to set Rate with HFJV?

Optimal frequency during all types of high frequency ventilation produces the best ventilation with the lowest airway pressure (i.e., smallest V_T) and no gas trapping. The primary determinant of optimal frequency is lung compliance, which is primarily determined by the patient's size. (An adult's lung compliance is greater than that of a child, which is greater than that of a term newborn, which is greater than that of a preemie.)

Venegas and Fredberg found that the resonant frequency of the smallest preemie with the stiffest lungs is approximately 40 Hz (2400 bpm!).[6] However, they also found that it makes very little difference in airway pressure to ventilate the smallest infants at frequencies as low as 10 Hz (600 bpm). In other words, 600 bpm, where the danger of gas trapping is greatly reduced, works just as well as 2400 bpm.

As lung compliance goes up, optimal frequency goes down. Thus, any patient larger than the smallest preemie will require a frequency less than 600 bpm. As airway resistance goes down, optimal frequency also goes down because more time is needed for exhalation. Thus, when inadvertent PEEP is detected, HFJV rate should be reduced.

Most clinicians start premature infants on the Life Pulse at 420 bpm. They then have the option of increasing the rate to as high as 660 bpm if lung compliance is particularly low, or the option of decreasing the rate to as low as 240 bpm if airway resistance is particularly high.

How does one control blood gases during HFJV?

Several investigators have found CO_2 elimination to be proportional to frequency times tidal volume squared (V_T^2) during HFV.[7,8] Thus, clinicians should choose a frequency consistent with patient's size and condition (i.e., lung compliance and resistance), and adjust PIP to produce the desired $PaCO_2$. It is important to note, however, that the real determinant of V_T is $\triangle$P, the difference between PIP and PEEP. There are many cases where raising PEEP is the appropriate way to reduce tidal volume to raise a low $PaCO_2$ because a low PaO_2 also exists.

Mean airway pressure and appropriate management of lung volume are the primary determinants of oxygenation during HFJV just as they are in every other form of mechanical ventilation. Since HFV enables safe use of PEEPs that are higher than those used during conventional ventilation, it is very useful to separate lung volume recruitment from alveolar stabilization when one is attempting to improve oxygenation.

Conventional ventilator rates ranging from zero to ten breaths per minute are typically used during HFJV. More IMV breaths are used when atelectasis is compromising oxygenation. The larger conventional tidal volumes are much more effective in recruiting collapsed alveoli than HFV breaths even when the latter are delivered with higher PEEP.

Once evidence of lung volume improvement is indicated by rising oxygen saturation (SaO_2) using pulse oximetry, the conventional ventilator rate should be reduced in favor of higher PEEP. Although similar PaO_2s may be produced with either HFJV with 10 conventional IMV breaths per minute and a PEEP of 5 cm H_2O or HFJV with the conventional ventilator providing only PEEP at 10 cm H_2O, the latter is considered much safer. In other words, trading higher PEEP for fewer IMV breaths is a good exchange for most patients.

Once alveolar recruitment is evident by improved O_2 saturation, lung recruitment using IMV breaths should be discontinued. However, when IMV rate is reduced, mean airway pressure will fall. Thus, the monitored mean airway pressure indicated on the front panel of the Life Pulse must be maintained by raising PEEP in order to stabilize the lungs and maintain adequate oxygenation.

If SaO_2 falls when IMV rate is reduced, PEEP is evidently too low, and it should be raised immediately. **I**f you wait too long to raise PEEP, you will need to re-recruit collapsed alveoli by temporarily increasing IMV breath rate. As O_2 saturation improves, IMV rate can be brought back down while PEEP is kept at the higher level.

Figure 8 illustrates a flowchart for finding optimal PEEP using the principles just discussed.

To recruit lung volume in the absence of atelectasis, just raise PEEP. While many neonatologists are reluctant to use PEEP > 6 cm H_2O, higher levels are frequently indicated for premature infants. If PEEP is too high, it interferes with cardiac output, but risk of lung injury is associated more with large (i.e., conventional) tidal volumes, especially when provided on top of PEEP, rather than PEEP itself. Most preemies need PEEP of 7 to 8 cm H_2O during HFJV.[9]

With non-homogeneous lung disease where spotty atelectasis may be present, concomitant IMV at low rates (2-5 bpm) may be helpful to improve ventilation/perfusion matching.[10]

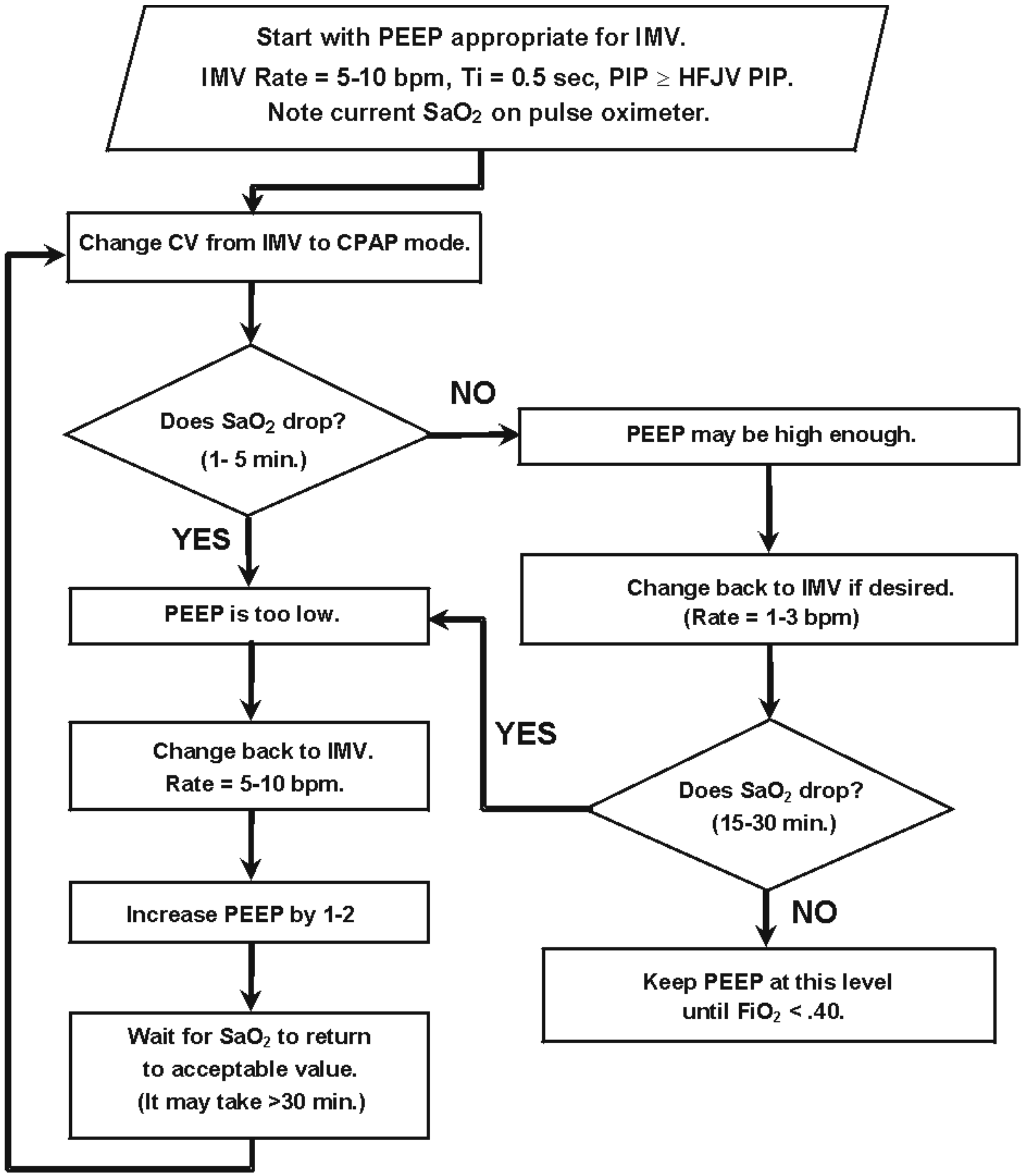

Figure 8: Flowchart for Finding Optimal PEEP during HFJV.

<u>Using HFJV in Newborn and Pediatric ICUs</u>

The Life Pulse HFJV has been used in the United States since 1983. (It was the first high frequency ventilator approved for clinical use by the U.S. Food and Drug Administration in 1988.) In the past two decades of animal and human trials, several theories have been proposed to explain the success and failures of various clinical applications. The following discussion is a current summary of what we know and don't know about how to properly use this technology.

Clinical disorders that are at least theoretically amenable to HFJV treatment include:

1. Restrictive Lung Diseases (conditions accompanied by low lung compliance such as pneumonia, tension pulmonary interstitial emphysema [PIE], diaphragmatic hernia, and pulmonary hypoplasia)

   The first multi-center, randomized, controlled trial of HFJV in America compared Life Pulse treatment with rapid rate, short inspiratory time, conventional ventilation in 144 newborn infants with severe PIE.[11] Successful treatment occurred in 61% of the HFJV-treated infants compared to 37% success in the infants treated conventionally. Survival rate, excluding those infants rescued from failure on conventional ventilation via pre-established crossover criteria, was 65% in the HFJV treated infants compared to 47% in controls.

2. Atelectatic Lung Diseases (such as respiratory distress syndrome [RDS] where alveoli can be safely opened through the use of high mean airway pressure HFV, or HFV in conjunction with low-rate IMV and optimal PEEP)

   A multi-center, randomized, controlled clinical trial of HFJV versus conventional ventilation for the treatment of 130 premature infants with uncomplicated RDS found a lower incidence of chronic lung disease at 36 weeks post-conceptional age (20% vs. 40%, respectively).[9] Only 5.5% of the HFJV-treated infants were discharged home on oxygen vs. 23% of the conventionally treated infants. There was no difference in survival by study design (crossover criteria were established to maximize survival in both treatment groups). All infants studied received surfactant within the first eight hours of life.

3. Some Obstructive Lung Disorders (such as meconium and other aspiration pneumonias, where HFJV is effective in facilitating removal of aspirated material and mucus, and some airway stenoses where HFV improves ventilation/perfusion matching)

   A single-center randomized controlled trial of HFJV and conventional ventilation in 24 neonates with respiratory failure and persistent pulmonary hypertension of the newborn (PPHN).[12] Most of the infants in the HFJV-treated group (8 of 11) and nearly half (5 of 13) of the conventionally treated infants had either meconium aspiration or sepsis pneumonia. Treatment failure within 12 hours of study entry occurred in only 2 of the HFJV-treated infants vs. 7 of the conventionally-treated infants ($p = 0.17$). ECMO was used to treat 4 of 11 HFJV infants vs. 10 of 13 control infants ($p = 0.11$). Zero of nine surviving HFJV-treated infants developed chronic lung disease compared to four of ten surviving controls ($p = 0.08$). Survival without ECMO in the HFJV group was 5 of 11 (45%) vs. 3 of 13 (23%) in the control group ($p = 0.26$). The authors estimated that 66 patients in each group would be required to establish statistical significance in these survival figures with 95% confidence and 80% power.

4. Pulmonary Airleak Syndromes (where low mean airway pressure HFJV with its smaller tidal volumes and lower intrapulmonary pressure amplitudes facilitate healing)

   Abrupt, high-velocity HFJV insufflations “shoot” inspired gas right past large, upper airway leaks and fistulae, allowing them to heal as in the case presented at the beginning of this article. With other air leaks such as pneumothoraces and PIE, there are other explanations for HFJV success.

   Most pneumothoraces and PIE start near the terminal bronchioles of premature infant lungs because that is where they are most compliant. (The alveoli are surfactant deficient and have yet to fully form.) Thus, when positive pressure conventional ventilation is applied, the terminal bronchioles take more than their fair share of mechanical stress.

The very small tidal volumes and short inspiratory times of HFJV make it difficult for bulk gas flow to reach the terminal bronchioles. In addition, the very high velocity HFJV inspirations will avoid areas of the lungs where airway resistance is increased.

With PIE, extrapulmonary gas dissects into the interstitial spaces surrounding the alveoli as well as into the interstitial spaces within airway walls. The only direction this gas can move within the airway walls is *upstream* from where the gas is leaking out of the terminal bronchioles. Thus, airway resistance is increased upstream of the leak. HFJV will therefore avoid going into areas affected by PIE because the disease process increases airway resistance in those areas. (See Figure 9.)

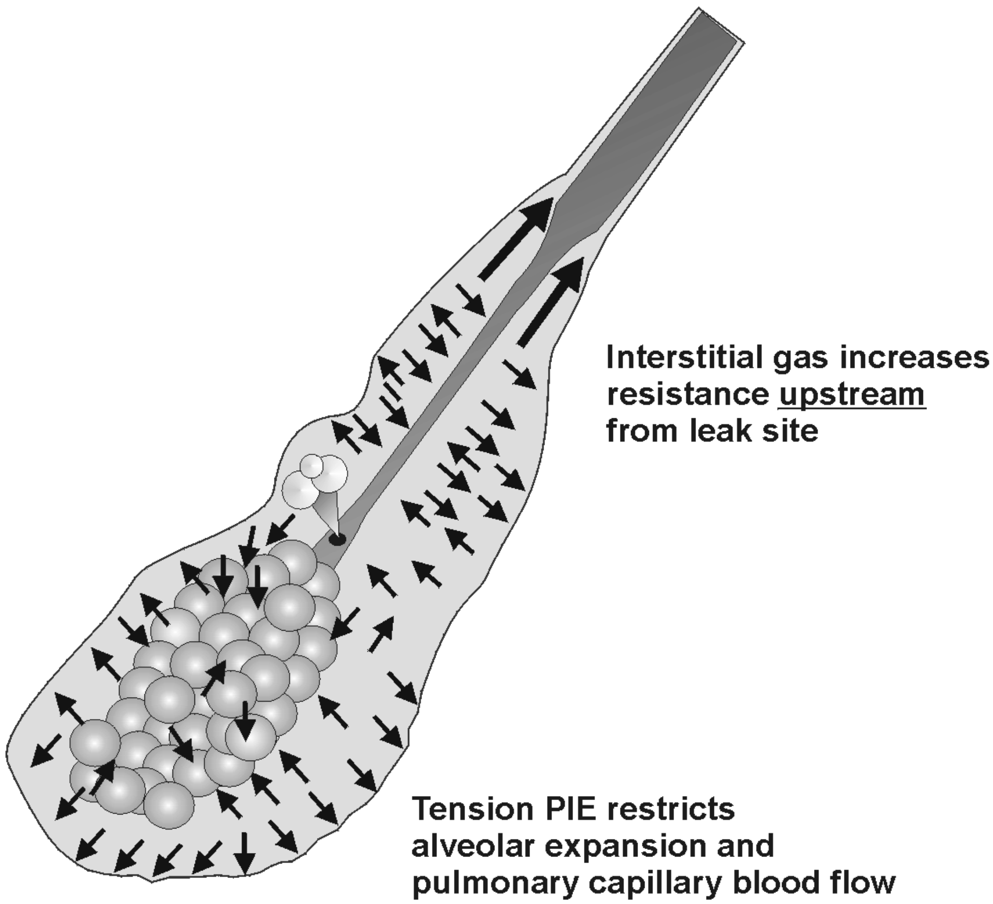

Figure 9: PIE dissects into airway walls upstream from its point of origination, increasing airway resistance and decreasing the opportunity for high velocity gas to reach the leak.

Gonzalez and associates demonstrated the value of HFJV in treating pneumothoraces by measuring the gas flowing through chest tubes during treatment with the Life Pulse vs. conventional ventilation in six infants with severe pulmonary disease.[13] Controls on the infants' ventilators were set to produce nearly identical arterial blood gases at PEEP = 4 cm H_2O and the patients were switched from conventional ventilation to HFJV. Mean airway pressures dropped from a mean of 15.0 to 9.7 cm H_2O from CV to HFJV ($p < 0.05$). The mean gas flow rates through the infants' chest tubes likewise dropped from 227 to 104 mL/min. ($p < 0.05$)

5. Cardiac Surgery Patients and PPHN (where low mean airway pressure HFJV with its lower intrapulmonary pressure amplitudes avoids interfering with cardiac output)

   The ability of HFJV to hyperventilate while using lower mean airway pressure is a great asset when treating patients with cardiac problems. During surgery, the small tidal volumes and low mean airway pressure allow the surgeon to move the lungs out of the way in order to visualize and work on the heart.

   After surgery, the Life Pulse can gently hyperventilate the patient to encourage increased pulmonary blood flow while mean airway pressure is kept down. Several studies have highlighted the capabilities of HFJV in these regards.[14,15,16,17]

   HFJV is also very effective in improving oxygenation in premature infants with PPHN using hyperventilation. However, it is extremely important to note that hypocapnia has been associated with severe periventricular leukomalacia in premature infants as discussed in the next section. It is probably much safer to use nitric oxide in conjunction with HFJV in this patient population.[18,19]

Adverse Side Effects Associated with HFJV

Early applications raised the specter of increased tracheal-bronchial mucosal damage with HFJV.[20] Infants in end-stage respiratory failure were treated after many days of high-pressure conventional ventilation in the early days of HFJV (beginning in 1983), and a high incidence of necrotizing tracheal bronchitis (NTB) was frequently noted at autopsy in those infants who died. As HFOV came into clinical use, it too became associated with NTB.[21]

Clinically significant NTB was rarely diagnosed in survivors. An increased incidence of NTB in any of the several randomized clinical trials comparing HFJV to CV has never been shown. While tracheal inflammation was commonly found in infants treated with HFJV or HFOV, it was rarely clinically significant. After a number of animal studies and further clinical applications, it was concluded that NTB is a predictable adverse side effect in extreme rescue cases, regardless of the mode of ventilation, and humidification is of the utmost importance in lessening this injury.[22,23]

Although the etiology of NTB appears to be multifactorial, improved humidification of inspired gases is credited with reducing the appearance of NTB in recent clinical studies. With the Life Pulse Ventilator, humidification is partly automatic and partly manually controlled. The temperature set point in the humidifier cartridge determines the quantity of water put into the flowing gas. Thus, the “Cartridge Temperature” can be manually adjusted up or down to maintain adequate humidification with minimal condensation in the patient circuit near the endotracheal tube. The temperature of the gas delivered to the patient is automatically maintained near 37°C by keeping the “Circuit Temperature” at its 40°C default setting.

When condensation is observed at the patient’s endotracheal tube, one knows 100% relative humidity has been reached. If there is no condensation, relative humidity is something less than 100%, and there is no easy way to determine how much less. Thus, it is wise to maintain a small amount of condensation there during HFJV by raising or lowering the set Cartridge Temperature.

More recent studies of HFJV have raised concern over an increased incidence of severe neuro-imaging abnormalities such as intraventricular hemorrhage (IVH) and periventricular leukomalacia (PVL).[24] Clark and associates conducted a meta-analysis to determine if premature neonates treated with HFV are at greater risk of developing IVH or PVL the infants treated with conventional ventilation.[25] They included every randomized controlled study comparing HFV with conventional ventilation in the treatment of respiratory distress syndrome.

The meta-analysis showed that there was an association between the use of HFV and PVL, but not IVH. However, one study, the "HIFI Study" sponsored by the U.S. National Institutes of Health in the mid-1980s, was much larger than all the other studies. In addition, it was conducted before there was widespread appreciation of the importance of maintaining optimal lung volume during HFV. When the HIFI study was removed from the analysis, the association between HFV and PVL disappeared.

Studies have linked hyperventilation and decreased cerebral blood flow to PVL.[26,27] Two studies have described severe IVH and PVL associated with hyperventilation and HFV.[24,28] Another study with surfactant found associations between rapid gas exchange and PVL.[29] These authors hypothesized that the increased incidence of PVL was caused by overventilation due to rapid changes in lung mechanics.

In two studies where HFJV was compared to conventional ventilation for the treatment of RDS in premature infants, severe neuroimaging abnormalities were increased when PEEP was kept too low (i.e., 5 cm H_2O).[9,24] The low PEEP also led to lower PaO_2 and lower $PaCO_2$ in the affected patients. The hypothesis here is that low PEEP during HFJV leads to hyperventilation because PIP is kept unnecessarily high in order to maintain adequate mean airway pressure. The difference between PIP and PEEP, ΔP, creates the tidal volume, which in the case of HFV has the greatest affect on CO_2 elimination. (See discussion above.) Thus, these infants could have suffered hypoxemic cerebral injuries because they had less blood supplied to the brain, and that blood carried less oxygen.

<u>HFJV Contraindications and Limitations</u>

While there are no known contraindications to HFJV, it should theoretically not be of much benefit in treating lung disorders such as asthma wherein airway resistance is uniformly increased. Low rates and long expiration times should be more effective here. However, some recent work with HFJV and helium-oxygen mixtures has demonstrated interesting potential for treating such disorders.

Tobias and Grueber improved ventilation in a one-year old infant with respiratory syncytial virus and progressive respiratory failure related to bronchospasm with the Jet using a mixture of air and 80% helium/20% oxygen from a tank.[30] Thus, if a patient requires no more than 80% oxygen with nitrogen, there may be room to try a "heliox" in order to improve ventilation.

HFJV has also been used successfully with nitric oxide (NO).[18,19] However, NO does not work with HFJV when the gas is administered through the conventional ventilator circuit only.[31] Unpublished data indicate that the gas in the conventional ventilator circuit mixes with the gas from the HFJV circuit. However, NO must be administered via the HFJV circuit in order for the patient to realize any beneficial effect from the gas.

The limitations of HFJV with the Life Pulse Ventilator are related to both its design and how it is used. Passive exhalation, for example, limits the upper frequency to which an operator can adjust the Rate setting in order to achieve optimization. Gas trapping will occur when the frequency is too high for the patient's size. (Larger patients require lower rates. <u>Note</u>: the largest known patient successfully treated with the Life Pulse Ventilator to date weighed 28 kg at the time of treatment.) HFJV rate must also be reduced in some patients when they improve. (Lungs that are more compliant require lower rates.)

Operators must be prepared to suction right after initiation of HFJV. HFJV very effectively facilitates mucociliary clearance, and excess secretions can quickly interfere with proper gas exchange if they are allowed to pool at the distal tip of the endotracheal tube.

Airway suctioning may cause alveolar collapse, and HFJV breaths may be too small to provide re-recruitment after suctioning. In such cases, the conventional ventilator rate must be temporarily increased to re-expand the lungs. However, if IMV rate is routinely left at relatively high levels (e.g., 10 bpm), injured lungs may not heal. Thus, concomitant IMV during HFJV must be managed appropriately. (Increase IMV rate to 5-10 bpm to actively recruit collapsed alveoli. Decrease rate to 0-3 bpm when atelectasis resolves. Cease IMV [i.e., use CPAP] when air leaks are present.)

How to Optimize HFJV

The following list is an attempt to summarize all the considerations one needs to weigh in order to use HFJV most effectively:

1. Learn when to start HFV without hesitation (i.e., when indicated by pathophysiology, weighing risks versus benefits, associating ventilator patterns with current or potential injuries, etc.).
2. Match ventilator strategy to patient's pathophysiology.
3. Choose HFJV rate based upon patient size and lung time-constant.
4. Use minimum T_I or I:E ratio to maximize inspired gas penetration and minimize gas trapping.
5. Find and use optimal PEEP or $P\overline{aw}$, paying particular attention to whether one needs to recruit more lung volume or just stabilize the volume already recruited.
6. Temporarily increase concomitant IMV rate only as needed to recruit collapsed alveoli.
7. Once recruitment is successful, IMV rate should be reduced and PEEP must be optimized to support the open alveoli without compromising cardiac output.
8. Keep $PaCO_2$ in proper range using PIP or PEEP. (Use PIP if PaO_2/F_IO_2 is satisfactory. Raise PEEP if PaO_2 and $PaCO_2$ are too low.)
9. Don't drop PEEP or allow mean airway pressure to fall until F_IO_2 is below 0.40. (When other settings are reduced, raise PEEP to keep $P\overline{aw}$ constant.)
10. Adjust settings rationally as patient's condition changes.
11. Do not stop prematurely. (If you get a bad blood gas, reassess and adjust strategy.)
12. Do not change back to conventional ventilation too soon. (Injured lungs take time to heal!)

Conclusions: Weighing the Risks and Benefits of HFJV

While there is still considerable uncertainty about what causes IVH and PVL in premature infants, we are certain about how to mitigate hypoxemic cerebral injuries. In the multicenter, randomized study using HFJV for RDS, those infants who were treated with increased PEEP had better oxygenation, more appropriate ventilation, and less cerebral injury compared to those infants treated with lower PEEP.* [The incidence of severe IVH or PVL in the "optimal" PEEP HFJV subgroup was 3 of 34 (8.8%) vs. 9 of 27 (33.3%) in the "low" PEEP subgroup.[9]]

Proper alveolar-volume recruitment is critical for good oxygenation. Therefore, finding and maintaining optimal PEEP or $P\overline{aw}$ is also crucial. In addition, optimal PEEP helps the HFJV operator maintain appropriate $PaCO_2$ in the patient, because he will not have to raise PIP in order to provide adequate mean airway pressure.

HFJV is not for every patient, but it may provide incredible benefits if it is used on the appropriate patient in the appropriate way at the appropriate time. There are very few things one needs to know to be successful with HFJV beyond a solid knowledge of pulmonary pathophysiology, respiratory therapy, and common sense.

When all is said and done, HFJV is just like every other form of assisted ventilation. In the majority of cases, the most important consideration is *how* it is used. If in doubt, the HFJV novice is wise to pick up the telephone or dash off an email and ask those clinicians with more HFJV experience for help.

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* Mean PEEP in the higher vs. lower groups $\cong$ 7 vs. 5 cm H_2O. Arterial to alveolar PaO_2 ratio [a/AO_2] was measured at 2, 6, 12, and 24 hours after study enrollment. Mean values at 2, 6, and 24 hours for the higher vs. lower PEEP groups were statistically different ($p < 0.05$). (Mean a/AO_2 in first 24 hours on higher vs. lower PEEP ~ 0.29 vs. 0.23, respectively.) Mean $PaCO_2$ values for the higher vs. lower PEEP groups were statistically different at 2, 6, and 12 hours. (Mean $PaCO_2$ in first 24 hours on higher vs. lower PEEP $\cong$ 37 vs. 33 torr.)

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