Factors Influencing Pulmonary Volumes and CO₂ Elimination during High-frequency Jet Ventilation

J. J. Rouby, M.D.,* G. Simonneau, M.D.,† D. Benhamou, M.D.,‡ R. Sartene, Ph.D., M.D.,§ F. Sardnal, M.D.,† H. Deriaz, M.D.,§ P. Duroux, M.D.,∥ P. Viars, M.D.**

An external spirometric method using a differential linear transformer was used to measure tidal volume (V₉) and to determine factors influencing CO₂ elimination and HFJV-induced “PEEP effect” in 15 critically ill patients under HFJV. V₉ increased with increasing driving pressure (DP) and decreasing frequency (f) and was influenced little by changes in I/E ratio. CO₂ elimination, as reflected by the measurement of PaCO₂, was mainly influenced by the absolute level of V₉ rather than by the product V₉ × frequency (PaCO₂ = 5715/V₉, r = 0.75, P < 0.05). The primary phenomenon explaining HFJV-induced “PEEP effect” was intrapulmonary gas trapping due to incomplete exhalation of the first V₉ administered: the spontaneous relaxation times of these first V₉ were longer than expiratory time allotted to the ventilatory settings. HFJV-induced “PEEP effect” increased with I/E ratio, DP, and f and was markedly influenced by the mechanical properties of the total respiratory system. At given ventilatory settings, HFJV-induced “PEEP effect” was greater in patients with a normal or elevated time constant of the total respiratory system (τRS) than in patients with a low τRS. These results suggest that HFJV should not be used in patients with chronic obstructive pulmonary disease and asthma, and should be preferentially administered to patients having stiff lungs or decreased chest wall compliance. (Key words: Carbon dioxide; elimination. Lung: volume. Ventilation: high-frequency jet.)

Since the early 1980s, high-frequency jet ventilation (HFJV) has been widely used in animals and humans for short or prolonged periods, with the underlying idea that adequate gas exchange could be achieved with high respiratory rate and small tidal volumes, either less than or approximate to the volume of the patient's dead space volume. Unfortunately, this assumption has not been substantiated until now, mainly because of a lack of any satisfactory technique for measuring tidal volume. Four basic problems arise when measuring tidal volume in patients under HFJV. First, HFJV is generally delivered with the use of an open ventilatory system, allowing entrainment of atmospheric gas into the patient, and, consequently, tidal volume results from the addition of an additional volume entrained by the Venturi effect to the jet gas volume delivered by the ventilator. This implies the presence of a continuous bias flow of gas complicating tidal volume measurement. Second, in most HFJV devices, inspiratory and expiratory phases are not clearly differentiated so that conventional volume meters cannot be used. Third, since entrainment is greatly dependent on resistance to gas flow, any interposition of measurement apparatus on the expiratory line may by itself modify entrainment and consequently tidal volume. Last but not least, most variable pressure drop flow meters do not have a sufficiently high frequency response necessary to follow the rapid changes in the upper airways. Suprisingly, these methodologic problems have not been discussed or mentioned in most reviews8-7 or recent papers8,9 concerning HFJV.

It has also been demonstrated that HFJV can improve arterial oxygenation in patients with acute respiratory failure by creating a “PEEP effect” at the alveolar level.10,11 However, the underlying mechanism of this “PEEP effect” has not been clearly assessed in these studies mainly because of a lack of any technique for analyzing HFJV breath by breath.

Since most conventional direct spirometric methods appear inappropriate to measure tidal volume during HFJV, we used an external spirometric method with a double aim: first, to determine factors influencing tidal volume and CO₂ elimination in patients under HFJV; second, to describe the mechanisms of HFJV-induced “PEEP effect.” In a preliminary study we assessed the accuracy of the method for measuring small volumes at high frequencies.

Methods

Patients

Fifteen critically ill patients hospitalized in the Surgical Intensive Care Unit of the Pitie Hospital were selected according to the following clinically determined criteria: 1) presence of respiratory failure necessitating mechanical ventilatory support; 2) presence of a stable hemodynamic condition without hypovolemia; 3) presence of an arterial catheter for cardiovascular monitoring; 4) absence of

* CHA, ier grade, Clinical Chief, Critical Care Unit.
† Assistant Professor in Pneumology.
‡ Staff Anesthesiologist.
§ Research Specialist.
∥ Professor of Pneumology.
** Professor of Anesthesiology, Chairman.

Received from the Département d’Anesthésie, Centre Hospitalo-Universitaire Pitie-Salpêtrière, 83 Boulevard de l'Hôpital, 75651 Paris Cedex 13, France, and Service de Pneumologie, Centre Hospitalo-Universitaire Antoine Béclère, 157 rue de la Porte de Trivaux, 92140 Clamart, France. Accepted for publication May 30, 1985. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, New Orleans, Louisiana, October 1984.

Address reprint requests to Dr. Rouby: Département d’Anesthesie, C.H.U. Pitie-Salpêtrière, 83 Boulevard de l'Hôpital, 75651 Paris Cedex 13, France.
known cardiac disease; 5) absence of chronic obstructive pulmonary disease; 6) absence of cerebral edema; 7) absence of unilateral acute lung disease.

Severe acute respiratory failure was not required in order to obtain a wide range of static respiratory compliance. Characteristics of patients are summarized in table 1. Static respiratory compliance (CRS) was measured using a 2–1 syringe as previously described.10 CRS was calculated as the slope of the curve between 500 and 1,500 ml during deflation. Before the study, all patients already were receiving large doses of morphine (0.20 mg·kg⁻¹·h⁻¹) according to their acute status.12,13 During the study, they were paralyzed with pancuronium 0.1 mg·kg⁻¹·h⁻¹. HFJV was administered only for the time of the study, and conventional positive-pressure ventilation was used as prolonged ventilatory support throughout the course of the respiratory insufficiency.

In addition, eight critically ill patients were studied in order to assess the accuracy of the differential linear transformers for measuring changes in lung volume (see below). Three had postoperative respiratory failure, and five were suffering from chronic coma related to cerebrovascular stroke. Since the procedure consisted of short periods of pulmonary inflation using HFJV, the only criteria for selection was the presence of respiratory failure, necessitating mechanical ventilatory support. Before the procedure, each patient received an intravenous dose of morphine (0.5 mg·kg⁻¹) and was paralyzed with pancuronium 0.1 mg·kg⁻¹.

All conscious patients were informed of the procedure and gave approval. Since they were receiving large doses of narcotics and were mechanically ventilated, informed consent also was required from the patient’s closest relative. In case of comatose patients, informed consent was obtained only from the patient’s closest relative. Authorization was given by the Clinical Investigation Committee of this institution.

**EQUIPMENT**

HFJV was delivered by a ventilator model VS 600® (Acutronic Medical Systems AG Switzerland). As shown in figure 1, air and oxygen were supplied under a pressure of 4 atm, mixed with a blender and pulsed by an electronically controlled solenoid valve with 2.2 mm internal diameter through a noncompliant Teflon® tube with 0.7-cm diameter and 120-cm length. This tube was connected to an injector cannula with a 1.8-mm internal diameter.
and 4-cm length, inserted into a three-way swivel adapter fixed to the tracheostomy or endotracheal tube. On the front panel of the ventilator there was a control knob of a second-stage pressure regulator, which allowed smooth control of driving pressure (DP), i.e., the pressure in the uncompliant Teflon® tube during the inspiratory cycle, from zero to maximum input pressure. This driving pressure continuously was monitored with the use of a high-pressure calibrated transducer, CZ 9029 Schlumberger®. Gas for entrainment was provided by an open anesthesia circuit connected to the three-way swivel adapter, which delivered warmed and humidified gases (30 l·min⁻¹) at the same F₁O₂ as the jet. The third part of the three-way swivel adapter enabled exhalation at atmospheric pressure (no PEEP valve). Inspiratory—expiratory (I/E) ratio and frequency (f) could be changed independently. Minute ventilation delivered by the ventilator increased with I/E ratio, DP, and f. Airway pressure was monitored continuously with a 1.65-mm internal diameter polyethylene catheter advanced into the trachea 10 cm distal to the tip of the injector cannula and connected to a calibrated Quartz pressure transducer 1290 A Hewlett-Packard®. The catheter tubing and transducer were filled with air, and the entire system was calibrated to a frequency response of 7 Hz. Mean airway pressure (Paw) was obtained by electronic damping of the signal.

**Evaluation of Pulmonary Volumes**

_The Differential Linear Transformer_. Changes in rib cage and abdominal circumferences were measured simultaneously with two differential linear transformers (DLT) connected to two bands around the patient and positioned at the nipple and umbilicus levels (fig. 2). The DLT system was composed of a sensor mounted on a flexible but nonextensible belt. The sensor (Schaewitz LN Industries, Gaillard, France) was composed of two parts, a differential linear transformer, inside of which a ferromagnetic core could freely move. With the rib cage and abdominal movements, the penetration of the core inside the transformer created a magnetic flux variation, directly proportional to rib cage and abdominal perimeter variations. The physical characteristics of the sensor were as follows: thermic drive 0.01% °C⁻¹, linearity 0.25%, sensitivity 20 mV·V⁻¹·mm⁻¹, frequency response 250 Hz. The belts were made of a 2-cm-wide nonextensible tissue. A 4-cm-long piece of rubber was fixed to the middle of each belt, thus enabling elastic recoil, in accordance with respiratory movements. The frequency response of the entire system—sensor plus belt—was 60 Hz.

_Calibration Procedure_. The respiratory system of a paralyzed ventilated patient can be considered as an open system with a single part having a single independent variable. Therefore, changes in pulmonary volume induced by mechanical ventilation can be deduced from changes in rib cage or abdominal circumferences using two motion—volume calibration curves. These calibration curves were established in each patient before administration of HFJV. Patients were disconnected from the ventilator and, using a specially calibrated 2—1 syringe, successive manual injections of 50 ml of air were administered up to a pulmonary inflation level of 2,000 ml. DLT signals—i.e., changes in rib cage and abdominal perimeters corresponding to each 50-ml pulmonary inflation—were recorded using a Gould ES 1000® recorder. The total procedure—40 successive inflations of 50 ml—was repeated in triplicate. Two motion—volume calibration curves were constructed on a X-Y diagram as follows: each 50-ml inflation was reported on the x-axis, and the corresponding changes in rib cage or abdominal perimeter (in mm) were reported on the y-axis. The motion—volume curves then were constructed and subsequently used to deduce changes in pulmonary volume from changes in rib cage or abdominal perimeters.

**Validation of the Method in High-frequency Ventilatory Conditions**. DLT accuracy for measuring small volumes at high frequencies was assessed in eight patients. Following the calibration procedure, the jet gas volume delivered by the ventilator (Vjet), which is independent of the output mechanical load, was measured using either DLT or a Tissot® spirometer at four frequencies: 100, 200, 300, and 400 · min⁻¹.

Using DLT, Vjet was measured as follows: the two lateral ports of the swivel adapter were closed in order to suppress any gas entrainment; the ventilator then was connected to the patient with the expiratory circuit closed, and the lungs were inflated at varying frequencies from apneic functional residual capacity up to 1,500 ml · DLT
TABLE 2. Comparative Value of Vjett Measured in Eight Patients by Using Either DLT or a Tissot® Spirometer (mean ± SD). Vjett Is Varied by Increasing f at Constant DP (3 atm) and I/E Ratio (0.43)

<table>
<thead>
<tr>
<th>f breaths·min⁻¹</th>
<th>100</th>
<th>200</th>
<th>400</th>
<th>600</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vjett ml (DLT)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic belt</td>
<td>217 ± 10</td>
<td>123 ± 9</td>
<td>69 ± 1</td>
<td>49 ± 1</td>
</tr>
<tr>
<td>Abdominal belt</td>
<td>216 ± 10</td>
<td>123 ± 9</td>
<td>68 ± 1</td>
<td>48 ± 1</td>
</tr>
<tr>
<td>Vjett ml (Tissot®)</td>
<td>209 ± 10</td>
<td>114 ± 10</td>
<td>66 ± 2</td>
<td>45 ± 1</td>
</tr>
</tbody>
</table>

Moreover, the same values were found for Vjett using either the thoracic or abdominal belt. In these paralyzed patients, the rib cage and abdomen did have the same proportionate contributions to Vjett at all frequencies. In other words, their respiratory system could be considered as a system having only one degree of freedom. Consequently, changes in pulmonary volume could be independently deduced from changes in rib cage perimeter or from changes in abdominal perimeter.

Definition of the Pulmonary Volume Measured. As shown in figure 3, several pulmonary volumes can be defined during HFJV from resulting variations in rib cage perimeter.

When the patient is disconnected from the ventilator, the baseline corresponds to the apneic functional residual capacity (FRC). It must be pointed out that DLT does not enable the measurement of the absolute value of FRC. Once HFJV is started, there is a transitory phase during which gases are trapped in the lungs, leading to an increase in rib cage perimeter and corresponding to an increase in FRC. This phase is followed by a stabilization of FRC.

At this moment three different pulmonary volumes can be described: 1) the increase in FRC (ΔFRC) corresponding to the end-expiratory airway pressure; 2) the mean pulmonary volume above apneic FRC (ΔV) corresponding to mean airway pressure (Paw) previously described using the clamps method10,11; 3) the tidal volume (V₂), which is the sum of Vjett and entrained volume (E). Vjett was measured by using the inflating procedure described above and E was calculated as V₂ - Vjett.

Once HFJV is stopped, there is a passive exhalation that depends on the mechanical properties of the lungs, chest wall, and breathing circuit. Previous studies have demonstrated the essentially exponential character of this passive exhalation curve in anesthetized subjects16:

\[ V = V₀ \cdot e^{-t/τ_{RS}} \]

where V is the volume exhaled, Vo the total exhaled volume, t time, and τRS the time constant of the total respiratory system. In fact time required for returning to apneic FRC depends on τRS, which can be calculated by measuring the interval required for thoracic volume (ΔFRC + V₂) to decrease to 36.8% of its end-inspiratory...
TABLE 4. Respiratory Effects of Increasing DP during HFJV (I/E ratio 0.48, frequency 200·min⁻¹) in 15 Patients (mean ± SD)

<table>
<thead>
<tr>
<th>Driving pressure (atm)</th>
<th>1.8</th>
<th>2.2</th>
<th>2.6</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_T$ (ml)</td>
<td>156 ± 30</td>
<td>187 ± 37*</td>
<td>199 ± 46†</td>
</tr>
<tr>
<td>$V_{jet}$ (ml)</td>
<td>70 ± 11</td>
<td>89 ± 9*</td>
<td>101 ± 10†</td>
</tr>
<tr>
<td>$E$ (ml)</td>
<td>85 ± 33</td>
<td>98 ± 40*</td>
<td>98 ± 48</td>
</tr>
<tr>
<td>$P_{ACO_2}$ (mmHg)</td>
<td>32 ± 10</td>
<td>29 ± 9*</td>
<td>26 ± 7†</td>
</tr>
<tr>
<td>pH</td>
<td>7.51 ± 0.09</td>
<td>7.55 ± 0.09*</td>
<td>7.58 ± 0.07†</td>
</tr>
<tr>
<td>ΔFRC (ml)</td>
<td>528 ± 320</td>
<td>852 ± 470*</td>
<td>976 ± 452†</td>
</tr>
<tr>
<td>ΔV (ml)</td>
<td>615 ± 323</td>
<td>948 ± 464*</td>
<td>1079 ± 493†</td>
</tr>
<tr>
<td>$P_{aw}$ (cmH₂O)</td>
<td>8 ± 2.7</td>
<td>12 ± 2.9*</td>
<td>14 ± 3.2†</td>
</tr>
<tr>
<td>$P_{ao_2}$ (mmHg) (FIO₂ 0.4)</td>
<td>63 ± 34</td>
<td>67 ± 29</td>
<td>72 ± 34</td>
</tr>
</tbody>
</table>

* $P < 0.05$ compared with DP 1.8 atm.  
† $P < 0.05$ compared with DP 2.2 atm.

value. Each $V_T$ administered has a spontaneous relaxation time (TEVT), which can be measured from the passive exhalation curve as shown in figure 3. The more the $V_T$ is administered at a high degree of lung inflation, the more the TEVT is shortened: the first $V_T$ administered ($V_{T1}$), has a longer TEVT (TEVT1) than the second $V_T$ administered ($V_{T2}$), which has a longer TEVT (TEVT2) than the third $V_T$ administered etc. (fig. 3).

PROCEDURES

Eight different ventilatory settings were used at random in 15 patients in order to determine respiratory changes induced by increasing frequency (f), DP, and I/E ratio (table 3). After a steady state of 20 min at FIO₂ 0.4, $V_T$, $V_{jet}$, $P_{aw}$, ΔFRC, ΔV, heart rate (HR), systolic pressure, diastolic pressure, and mean arterial pressure (SAP, DAP, MAP) were recorded in triplicate on the Gould ES 1000® recorder. Two successive speeds were used, 20 mm·s⁻¹ and 45 mm·s⁻¹, the latter enabling analysis of time intervals up to 0.02 s. Arterial blood gas was also measured simultaneously. All data were expressed as mean ± SD and were compared using analysis of variance and Student's paired t test. $P < 0.05$ was considered significant.

In further defining the relationship between $P_{ACO_2}$, $V_T$, and f, it was determined whether $P_{ACO_2}$ fitted best when plotted versus $V_T$ × f or versus $V_T$ alone. Linear regression models were used as follows: 1) $Y = a + bx$; 2) Log $Y = a + bx$; 3) Log $Y = a + b$ Log $X$, by the Hewlett-Packard® statistical package program. The multiple correlation coefficient, R², was used as the criterion in deciding which model best fit these data.

Results

RESPIRATORY EFFECTS RELATED TO VENTILATORY SETTINGS

Effects of Increasing DP (table 4). As shown in figure 4A, increasing DP from 1.8 to 2.6 atm significantly increased $V_T$, $V_{jet}$, and E. A significant decrease in $P_{ACO_2}$ and signific

ificant increase in pH resulted. Simultaneously, ΔFRC, ΔV, and $P_{aw}$ significantly increased. $P_{ao_2}$, arterial pressure, and HR remained unchanged.

Effects of Increasing I/E Ratio (table 5). As shown in figure 4B, increasing I/E ratio from 0.25 to 0.67 significantly increased $V_{jet}$, insignificantly increased $V_T$, and significantly decreased E. A slight decrease in $P_{ACO_2}$ and an increase in pH resulted. Simultaneously, ΔFRC, ΔV, and

![FIG. 4. Effects of increasing driving pressure (DP), inspiration/expiration ratio (I/E ratio), and frequency (f) on tidal volume ($V_T$), jet gas volume delivered by the ventilator (0), and entrainment (0) in 15 patients under HFJV (mean ± SD). * $P < 0.05$ compared with control value.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931403/ on 06/01/2017)
TABLE 5. Respiratory Effects of Increasing I/E Ratio during HJV (DP 2.6 atm, frequency 200·min⁻¹) in 15 Patients (mean ± SD)

<table>
<thead>
<tr>
<th>I/E Ratio</th>
<th>0.25</th>
<th>0.45</th>
<th>0.67</th>
</tr>
</thead>
<tbody>
<tr>
<td>V̇ (ml)</td>
<td>184 ± 35</td>
<td>199 ± 46</td>
<td>201 ± 56</td>
</tr>
<tr>
<td>V̇jet (ml)</td>
<td>78 ± 5</td>
<td>101 ± 10*</td>
<td>120 ± 11†</td>
</tr>
<tr>
<td>E (ml)</td>
<td>109 ± 33</td>
<td>98 ± 48*</td>
<td>77 ± 54†</td>
</tr>
<tr>
<td>Paco₂ (mmHg)</td>
<td>50 ± 8</td>
<td>26 ± 7*</td>
<td>26 ± 8</td>
</tr>
<tr>
<td>pO₂ (mmHg)</td>
<td>7.52 ± 0.11</td>
<td>7.58 ± 0.07*</td>
<td>7.60 ± 0.12</td>
</tr>
<tr>
<td>ΔFRC (ml)</td>
<td>360 ± 349</td>
<td>976 ± 452*</td>
<td>1527 ± 567†</td>
</tr>
<tr>
<td>ΔV (ml)</td>
<td>449 ± 355</td>
<td>1073 ± 453*</td>
<td>1627 ± 556†</td>
</tr>
<tr>
<td>Ṗaw (cmH₂O)</td>
<td>6 ± 2.7</td>
<td>14 ± 3.2*</td>
<td>22 ± 4.3†</td>
</tr>
<tr>
<td>Pao₂ (mmHg) (FiO₂ 0.4)</td>
<td>72 ± 40</td>
<td>72 ± 34</td>
<td>85 ± 47</td>
</tr>
</tbody>
</table>

* P < 0.05 compared with I/E 0.25.
† P < 0.05 compared with I/E 0.43.

Paw significantly increased. Pao₂, arterial pressure, and HR remained unchanged.

Effects of Increasing Frequency (table 6). As shown in figure 4C, increasing f from 100·min⁻¹ to 600·min⁻¹ significantly decreased V̇T, V̇jet, and E. A marked increase in Pco₂ and a decrease in pH resulted. At 100·min⁻¹ with a V̇T of 369 ± 62 ml, all patients were in respiratory alkalosis (pH = 7.65 ± 0.08 and Pco₂ = 21 ± 6 mmHg). At 400·min⁻¹, with a V̇T of 129 ± 47 ml, pH and Pco₂ were in the normal range, 7.40 ± 0.08 and 42 ± 11 mmHg, respectively. At 600·min⁻¹, with a V̇T of 79 ± 54 ml all patients were in respiratory acidosis (pH = 7.34 ± 0.09 and Pco₂ = 53 ± 15 mmHg). As shown in figure 5, a significant relationship was found between Pco₂ and V̇T; Pco₂ = 57.15/V̇T, r = 0.75, P < 0.05. No significant relationship was found between Pco₂ and the product V̇T·f. Simultaneously, ΔFRC, ΔV, and Paw significantly increased, resulting in an insignificant increase in Pao₂.

RESPIRATORY EFFECTS RELATED TO PULMONARY MECHANICS

Factors Involved in the Increase in Mean Lung Volume. As illustrated in figure 3, intrapulmonary gas trapping occurred because expiratory time (TE) was of shorter duration than the spontaneous relaxation times (TÊV̇T) required by the thorax to exhale the first tidal volumes. TÊ, which depends only on f and I/E, according to the following equation:

\[ TE = \frac{60}{f(I/E + 1)} \]

was of 0.21 s. The spontaneous relaxation time of the first V̇T administered, TÊV̇T1, was of 2.5 s; consequently, the major part of the first insufflation was trapped in the lungs. The spontaneous relaxation time of the second VT administered decreased to 1 s. Here again, a good part of the second V̇T was trapped in the lungs. Intrapulmonary gas trapping ceased at the eighteenth insufflation when TÊV̇T18 equaled TE.

Influence of the Mechanical Properties of the Total Respiratory System on the Increase in Mean Lung Volume. As shown in figure 6, identical ventilatory settings—f 200·min⁻¹, I/E ratio 0.43, and DP 2.6 atm—were applied to two patients with different respiratory mechanics. Patient 11, who was suffering from multiple trauma, had mild respiratory failure related to pulmonary contusion of the right inferior lobe. His CRS was 73 ml·cmH₂O⁻¹, and his tRS was 0.41 s and his calculated total respiratory resistance

TABLE 6. Respiratory Effects of Increasing f during HJV (DP 2.6 atm, I/E ratio 0.43) in 15 Patients (mean ± SD)

<table>
<thead>
<tr>
<th>f·min⁻¹</th>
<th>100</th>
<th>200</th>
<th>400</th>
<th>600</th>
</tr>
</thead>
<tbody>
<tr>
<td>V̇ (ml)</td>
<td>369 ± 62</td>
<td>199 ± 46*</td>
<td>129 ± 47*</td>
<td>79 ± 34*</td>
</tr>
<tr>
<td>V̇jet (ml)</td>
<td>186 ± 25</td>
<td>101 ± 10*</td>
<td>57 ± 5*</td>
<td>40 ± 4*</td>
</tr>
<tr>
<td>E (ml)</td>
<td>184 ± 51</td>
<td>98 ± 48*</td>
<td>73 ± 44*</td>
<td>38 ± 31*</td>
</tr>
<tr>
<td>Pco₂ (mmHg)</td>
<td>21 ± 6</td>
<td>26 ± 7*</td>
<td>42 ± 11*</td>
<td>53 ± 15*</td>
</tr>
<tr>
<td>pH</td>
<td>7.55 ± 0.08</td>
<td>7.58 ± 0.07*</td>
<td>7.40 ± 0.08*</td>
<td>7.34 ± 0.09*</td>
</tr>
<tr>
<td>ΔFRC (ml)</td>
<td>860 ± 480</td>
<td>976 ± 452</td>
<td>1,275 ± 574*</td>
<td>1,356 ± 609*</td>
</tr>
<tr>
<td>ΔV (ml)</td>
<td>1,033 ± 480</td>
<td>1,079 ± 453</td>
<td>1,347 ± 546*</td>
<td>1,400 ± 586*</td>
</tr>
<tr>
<td>Ṗaw (cmH₂O)</td>
<td>14 ± 3.6</td>
<td>14 ± 3.2</td>
<td>17 ± 3.6*</td>
<td>19 ± 3.8*</td>
</tr>
<tr>
<td>Pao₂ (mmHg) (FiO₂ 0.4)</td>
<td>70 ± 40</td>
<td>72 ± 34</td>
<td>82 ± 32</td>
<td>74 ± 24</td>
</tr>
</tbody>
</table>

* P < 0.05 compared with the precedent frequency.
(τRS/CRS) was 5.6 cmH₂O-L⁻¹·s⁻¹. Patient 15 was suffering from severe bilateral bacterial pneumonia. At the time of the study his CRS was 33 ml·cmH₂O⁻¹, his τRS was 0.20 s and his calculated total respiratory resistance was 6.1 cmH₂O-L⁻¹·s⁻¹. Patient 15 had a smaller increase in mean lung volume than patient 11, although identical ventilatory settings were applied. This phenomenon was related to the fact that TE(VT) equaled Tₑ earlier on in patient 15 in the presence of a steeper passive exhalation curve. TE(VT) equaled Tₑ at the eighteenth inscription in patient 11, who had compliant lungs, whereas TE(VT) equaled Tₑ at the twelfth inscription in patient 15, who had noncompliant lungs.

Respiratory effects of increasing respiratory frequency were quite different in patients 11 and 15. As illustrated in figure 7, the increase in f from 100 min⁻¹ to 600 min⁻¹ at a constant I/E ratio of 0.43 and a constant DP of 2.2 atm, induced a marked increase in Paw, ΔV, and ΔFRC in patient 11. Because of his "normal" passive exhalation curve, TE(VT) always remained longer than Tₑ, thus provoking an additional increase in mean lung volume whenever f was increased.

As illustrated in figure 8, different results were found in patient 15. Once HFJV was started at 100·min⁻¹, mean lung volume increased, because TE(VT) was 1.8 s, whereas Tₑ was 0.42 s. At 150·min⁻¹, TE(VT) was 0.23 s, whereas Tₑ was 0.28 s and no additional increase in mean lung volume occurred. At 200·min⁻¹, TE(VT) was 0.18 s, whereas Tₑ was 0.21 s and mean lung volume remained unchanged. At 250·min⁻¹, TE(VT) was 0.15 s, whereas Tₑ was 0.18 s and mean lung volume remained unchanged. At 400·min⁻¹, TE(VT) was 0.08 s, whereas Tₑ was 0.10 s and mean lung volume remained unchanged. At 600·min⁻¹, TE(VT) was 0.05 s, whereas Tₑ was 0.07 s and mean lung volume remained unchanged. Finally, in this patient presenting "stiff" lungs with a low τRS of 0.20 s, the steep portion of the passive exhalation curve was rapidly reached; because the tidal volumes administered at frequencies above 100·min⁻¹ had a shorter TE(VT) than Tₑ, no additional increase in mean lung volume occurred with increasing f.

**Discussion**

**CRITIQUE OF METHODS**

In this study, an external spirometric method using a differential linear transformer enabled continuous lung volume monitoring in patients under HFJV. Variations in pulmonary volumes were deduced from changes either in rib cage perimeter or in abdominal perimeter. In a previous study, this device had been used to discriminate abdominal and thoracic contribution to VT in five post-

**FIG. 7.** Respiratory effects of increasing f in patient 11 from 100 to 600·min⁻¹ at a constant I/E ratio of 0.43 and a constant DP of 2.2 atm. Tₑ = time constant of the total respiratory system.
operative inductance plethysmography, another method of indirect spirometry measuring variations in lung volumes from
variations in the cross-sectional area of the thorax, also
has been used to monitor lung volume during high-fre-
cuency oscillation \(^{18}\) or during spontaneous high-frequency breathing. \(^{19}\) In fact, any indirect spirometric
method, based on the principles defined by Konno and
Mead, \(^{15}\) offers several advantages over conventional
methods of direct spirometry for measuring pulmonary
volumes during high-frequency ventilation. First, it avoids
interposition of any apparatus on the respiratory pathways
that could modify resistance to gas flow and consequently
entrainment and tidal volume. Second, the frequency re-
sponse of these devices is very high, enabling an accurate
measurement of small volumes at high frequencies. The
entire DLT system—sensor plus belt—had a frequency
response of 60 Hz, whereas the higher frequency used in
this study was 10 Hz. Third, with the continuous moni-
toring of lung volume changes, an analysis breath by
breath enables an understanding of high-frequency ven-
tilation-induced increase in FRC.

DLT undoubtedly can accurately measure small vol-
umes at low frequency in spontaneously breathing pa-
nents \(^{14}\) as well as small volumes at high frequencies in
paralyzed patients whose lungs are inflated for a short
period (table 2). The validity of DLT in measuring small
volumes at high frequencies in paralyzed patients under
HFJV can reasonably be assumed on the following argu-
ments: first, thoracic and abdominal values of \(V_T\) were
found equal up to frequencies of 400 \(\text{min}^{-1}\), indicating
that neither the abdomen nor the rib cage had reached
their resonant frequency. At 600 \(\text{min}^{-1}\) the abdominal
signal could not be clearly individualized in most patients,
suggesting that the resonant frequency of the abdomen
had been exceeded. Consequently, tidal volume was de-
duced only from the thoracic signal. However, since the
resonant frequency of the chest wall in most humans is
much higher than 10 Hz, \(^{20-22}\) it is quite unlikely that an
important error was made in the measurement of tidal
volume at 600 \(\text{min}^{-1}\). Second, the validity of the mea-
surements of mean pulmonary volume above apneic FRC
is indirectly confirmed by the pressure/volume data. If
one divides \(\Delta V\) by \(P_{aw}\), the concurrence is obvious with
CRS at any frequencies. This provides an intrinsic vali-
dation of the two different methods used: the DLT system
for indirectly measuring \(\Delta V\) and the gross-syringe for di-
rectly measuring pulmonary volume.

**Factors Influencing Tidal Volumes and \(\text{CO}_2\) Elimination**

Considering our results, it is evident that tidal volume
was influenced mainly by frequency and driving pressure
and little by I/E ratio (fig. 4): \(V_T\), Vjet, and entrainment
were markedly decreased by increasing frequency and
markedly increased by increasing driving pressure. Ele-
vation of I/E ratio above 0.43 only induced a slight and
insignificant increase in tidal volume, since the marked
rise in Vjet was counterbalanced by a parallel decrease in
entrainment. These results are in keeping with theoretic
statements suggesting that driving pressure rather than
I/E ratio is a primary determinant of gas entrainment. \(^{23}\)

The present study also demonstrated that HFJV cannot
always be considered as a “low tidal volume ventilation.”
Using a frequency of 100 \(\text{min}^{-1}\), a driving pressure of
2.6 atm, and an I/E ratio of 0.43, we found a mean tidal
volume of 369 ml. Deep respiratory alkalosis with a severe
hypocapnia resulted in all patients, suggesting a dramatic
increase in alveolar ventilation. Normalization of the acid-
base balance was obtained in all cases by decreasing tidal
volume via an increase in frequency. We should note that
most of the clinical trials concerning HFJV have been
performed using similar frequencies—100-150 \(\text{min}^{-1}\)—
similar driving pressures—2-2.5 atm—and similar I/E ratio—0.25-0.43—with the underlying idea that small
tidal volumes close to the deadspace volume were admin-
istered. Our results clearly show that this assumption is
not always verified mainly because gas entrainment is at
least equal to or greater than Vjet.

Finally, we found that \(\text{CO}_2\) elimination, as reflected by the
measurement of \(P_{a\text{CO}_2}\), was mainly influenced by the
absolute level of tidal volume rather than by the product
tidal volume by frequency (fig. 5). Because our patients
were in a stable hemodynamic and clinical condition dur-
ing the study and heavily sedated by large doses of mor-
phine, it can be assumed that variations in \(P_{a\text{CO}_2}\) reflected
changes in \(\text{CO}_2\) clearance rather than variations in \(\text{CO}_2\)
production. Therefore, the fact that \(P_{a\text{CO}_2}\) was closely
related to tidal volume suggests that bulk convection by
direct alveolar ventilation rather than augmented disper-
sion \(^{24}\) plays a major role in gas transport during HFJV.

**Factors Influencing HFJV-Induced “PEEP Effect”**

The present study confirms our previous data showing
that HFJV-induced “PEEP effect” depends both on venti-
latory settings and pulmonary mechanics. \(^{10,11}\) It also en-
ables one to understand the mechanisms involved. The
primary phenomenon leading to an increase in intrathoracic pressures is intrapulmonary gas trapping due to
incomplete exhalation of the first tidal volumes admin-
istered. This intrapulmonary gas trapping occurs mainly
because spontaneous relaxation times of the first tidal vol-
umes administered are longer than expiratory time allott-
ed to the ventilatory settings. Gas trapping ceases when
spontaneous relaxation time, which shortens with lung
inflation, equals expiratory time (fig. 3). It is clear that any modification in the ventilatory settings leading to a shortening of expiratory time will induce an increased “PEEP effect.” This is observed mainly when increasing I/E ratio, or, to a lesser extent, when increasing frequency in patients with compliant lungs (fig. 7). Similarly, any increase in tidal volume without modifying expiratory time will lengthen spontaneous relaxation time and consequently increase HFJV-induced “PEEP effect.” This is observed when increasing driving pressure.  

On the other hand, for a fixed expiratory time and a given tidal volume, namely at fixed ventilatory settings, the spontaneous relaxation time will depend on the mechanical properties of the total respiratory system, lungs plus chest wall. In the presence of “stiff” lungs, which tend to decrease the time constant of the total respiratory system, HFJV-induced “PEEP effect” will be moderate because of a short spontaneous relaxation time, which will rapidly equal expiratory time (fig. 6). In case of normal lungs, of the total respiratory system will have a higher time constant and HFJV-induced “PEEP effect” will be greater because of a longer spontaneous relaxation time, which will equal expiratory time at a later stage. In the presence of abnormally compliant emphysematous lungs, which tend to increase the time constant of the total respiratory system, HFJV will markedly increase lung volume. In such patients, time required for spontaneous relaxation time to equal expiratory time will be dramatically long because of the shape of the passive exhalation curve. In a first approximation, it can be assumed that the time constant of the total respiratory system, which characterizes the time course of the passive exhalation of a paralyzed patient, is the product of resistance (R) by compliance (C). Although the assumption is not entirely valid—mainly because inertia, which is negligible at low frequencies, becomes an important factor at high frequencies—this model RC provides a useful approximation of the relationship between HFJV-induced “PEEP effect” and the mechanical properties of the lungs and chest wall. It is now well documented that HFJV can induce marked increases in lung volume in patients with normal or elevated CRS. This study demonstrates that this HFJV-induced “PEEP effect” is greatly dependent not only on I/E ratio and driving pressure but also on frequency (fig. 7). In contrast, HFJV moderately increases lung volume in patients with low CRS. In these patients, HFJV-induced “PEEP effect” is dependent mainly on I/E ratio, to a lesser extent on driving pressure, and little on frequency (fig. 8). In fact, there are two distinct responses to the increase in frequency, depending on the underlying pulmonary mechanics. In patients whose respiratory disease is characterized by predominant decrease in lung volume, lung stiffness, or chest wall rigidity, increase in frequency decreases tidal volume without changing mean pulmonary volume above apneic FRC and intrathoracic pressure. In distended patients in whom CRS and airway resistance are already increased, increase in frequency decreases tidal volume with a simultaneous increase in mean pulmonary volume above apneic FRC and intrathoracic pressures.

The specific contribution of elevated airway resistance to HFJV-induced increase in lung volume was not measured in this study. However, several previous studies have demonstrated that, during HFJV, airway diameters increase during inspiration and decrease during expiration. It is likely that expiratory flow limitation plays an important role in the genesis of HFJV-induced “PEEP effect.” It is also clear that any increase in bronchial reactivity may amplify this phenomenon and lead to pulmonary overdistention.

### Clinical Implications

In the past decade, HFJV has been proposed as a new mode of mechanical ventilatory support for patients with various forms of respiratory failure. Certain studies or clinical reports have shown that HFJV can be more efficient than conventional positive-pressure ventilation to treat patients with broncho-pleural fistula severe ARDS or mild postoperative respiratory failure. Other studies have demonstrated that HFJV is at least as efficient as conventional ventilation to treat various forms of acute respiratory failure, such as severe postoperative respiratory failure, respiratory failure occurring in immunocompromised patients, or in infants with hylane membrane disease.

In a previous study, we suspected that HFJV, as any type of high-frequency ventilation, could be deleterious in distended patients. In the past, Carlon et al. reported that they had to interrupt HFJV in a patient with chronic obstructive pulmonary disease. The present study largely confirms that the occurrence of inadvertent PEEP is quasiconstant in patients with chronic obstructive pulmonary disease because of the mechanical properties of their respiratory system. Therefore, HFJV should be considered to be contraindicated in all types of respiratory failure, characterized by increased compliance and elevated resistance, namely in acute respiratory failures complicating chronic obstructive pulmonary disease or asthma. In contrast, HFJV appears as a good indication in all types of respiratory failure characterized by decreased lung volumes, low pulmonary compliance, or chest wall rigidity. Good results have been reported in patients with ARDS. In these patients with stiff lungs, Pao2—upon which arterial oxygenation is depending—can be increased more efficiently by increasing I/E ratio than by increasing driving pressure, and remains unaffected by changes in respiratory frequency. HFJV can achieve similar oxygenation and CO2 elim-
nation as conventional ventilation does at equivalent P\textsubscript{aw} and lower peak airway pressure, thus decreasing the incidence of pulmonary barotrauma. For similar reasons, obese patients with decreased functional residual capacity, low chest wall compliance, and increased inflation pressures during conventional mechanical ventilation might benefit from HFJV.

In conclusion, this study provides evidence for the principal indications and contraindications of HFJV in respiratory failure: this method should not be used in patients with asthma or chronic obstructive pulmonary disease and should be preferentially administered to patients having stiff lungs or decreased chest wall compliance. Its potential advantage over conventional modes of ventilation has yet to be demonstrated conclusively. Further clinical comparative studies are required.

The authors thank the medical and nursing staff of the Surgical Intensive Care Unit of La Pitié Hospital for their help, G. Debrabender for technical assistance, and Mrs. F. Berkane and C. Mataguez for typing the manuscript.

**References**


2. Smith RB: Ventilation at high respiratory frequencies, high frequency positive pressure ventilation, high frequency jet ventilation and high frequency oscillation. Anesthesia 37:1011–1018, 1982


