REVIEW

High-frequency Jet Ventilation*  
Theoretical Considerations and Clinical Observations

Graziano C. Carlon, M.D., F.C.C.P.;† Cole Ray, Jr, R.R.T.;‡  
Mary Kathryn Pierri, M.D.;§ Jeffrey Groeger, M.D.;|| and  
William S. Howland, M.D., F.C.C.P.¶

High-frequency jet ventilation (HFJV) describes a technique of mechanical respiratory support based on the delivery of gases under conditions of constant flow and low pressure. Among the benefits ascribed to HFJV are lessened interference with hemodynamic function and reduced danger of barotrauma. The theoretical and technical aspects of HFJV are discussed and the clinical experience with 39 patients in respiratory failure reported. Synchronization of HFJV with heart rate was attempted in three patients. Cardiac output and ejection fraction increased in all of them. At present, results suggest that HFJV may be the ideal form of support for patients with major airway disruption. The available data also indicate that extensive clinical trials are warranted to define advantages and limits of this form of ventilation.

Positive pressure mechanical ventilation and the development of intensive care units have radically changed the prognosis of acute respiratory failure in the last 20 years.¹

There are, however, specific instances of respiratory failure that present difficult problems, when positive pressure ventilation must be instituted.

Bronchopleural or Tracheoesophageal Fistulas

Positive pressure ventilation provides tidal volume by increasing alveolar pressure during each inspiratory cycle. A discontinuity of the tracheobronchial tree, with communication to the intrapleural space or the digestive tract, is essentially equivalent to an additional, very compliant lung. Accordingly, when airway pressure increases, part of the tidal volume will be lost through the fistula. The fraction of tidal volume lost will be directly proportional to the size of the pathologic opening and the airway pressure and inversely proportional to the compliance of the lungs.

In patients with respiratory failure and reduced compliance, this fraction will be considerable. Delivering larger tidal volumes is usually not effective, since this requires either higher inspiratory pressures, with resulting greater air leak or lower flow rates and prolonged inspiratory times. Although some patients can be successfully treated with intermittent mandatory ventilation (IMV) or application of counter pressure to the pleural cavity in coincidence with positive breaths,² the prognosis is often poor.³

Loss of Compliance and Barotrauma

Five to 20 percent of all patients who receive positive pressure ventilation will have some form of barotrauma (pneumomediastinum, pneumopericardium, subcutaneous emphysema, or pneumothorax).⁴ High peak inspiratory pressure, associated with severe decrease of compliance, and the existence of regions within the lungs with considerably different compliance are among the most common factors leading to barotrauma.⁵ When positive pressure ventilation must be continued, the danger of progressive parenchymal disruption is increased. A specific disease entity associated with marked loss of compliance is lung fibrosis. Although presently not common, there are reasons to believe that its prevalence may increase in the future. Many forms of therapy used in the treatment of malignant dis-

*From the Memorial Sloan-Kettering Cancer Center, New York.
†Clinical Chief, Department of Critical Care.
‡Technical Director, Department of Respiratory Therapy.
§Assistant Attending, Department of Critical Care.
¶Clinical Assistant Attending, Department of Critical Care.
||Chairman, Department of Critical Care.

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Reprint requests: Dr. Carlon, Memorial Sloan-Kettering Cancer Center, 1275 York Avenue, New York 10021

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eases (alkylating agents, bleomycin, nitrogen mustard, methotrexate, or radiation therapy) may cause as a serious side effect the development of lung fibrosis. A fibrotic lung can be uncompliant and have poor elastic recoil, so that both inspiration and expiration require active efforts. When acute respiratory failure develops in patients with lung fibrosis, conventional forms of respiratory support, such as IMV and PEEP, may not be successful. Work of breathing is greatly increased, and PEEP may not reexpand lungs with major structural changes, such as diffuse fibroelastic proliferation. Delivering a large tidal volume with a conventional mechanical ventilator often produces a high peak inspiratory pressure, greatly increasing the risk of airway disruption and hemodynamic embarrassment. In addition to chemotherapy, lung fibrosis can rapidly develop in other forms of respiratory failure, such as Pneumocystis carinii pneumonia and intrapulmonary hemorrhage in the presence of thrombocytopenia. Finally, accelerated fibroelastic proliferation may occur in almost all forms of acute respiratory failure; indeed, according to some authors, accumulation of an abnormal interstitial collagen, where the elastic component is very poorly represented, is ultimately responsible for the irreversible changes of lung structure.

A form of ventilatory support that might prove effective in some of the difficult clinical conditions described should have the following characteristics:

1. Provide adequate alveolar ventilation with small tidal volumes and low inspiratory pressure, or
2. Deliver higher tidal volume, when necessary, without increasing airway pressure or decreasing flow rate.
3. Minimally interfere with hemodynamic function.

Since 1967, it has been known that it is possible to maintain normal arterial blood gases by delivering frequent (50 to 60/min) bursts of air and oxygen through a small cannula inserted in the upper airways. This technique was defined as high frequency positive-pressure ventilation (HFPPV). Sjostrand et al subsequently developed a mechanical ventilator, characterized by negligible internal compliance, which was powered by the pressure of the gas source and could deliver 20 to 100 breaths/min of predetermined tidal volume. This ventilator has been utilized in many clinical applications. Since tidal volume is delivered through a narrow catheter (3 to 4 mm), HFPPV was useful where insertion of a large endotracheal tube could obstruct surgical procedures (laryngeal surgery, bronchoscopy, etc).

Clinical and experimental research demonstrated some important characteristics of this form of ventilation. (1) Normal PaCO₂ could be maintained with tidal volumes only slightly larger than dead space. (2) Peak airway pressure was considerably lower than with conventional ventilation, although mean airway pressure was minimally different. (3) Hemodynamic function was not impaired by mechanical ventilation with HFPPV, partly because elevated airway and intrapleural pressure did not develop. (4) Spontaneous ventilation ceased at normocapnia, when respiratory rates ≥60 breaths/min were delivered. Experiments in cats demonstrated that the train of afferent impulses carried by the vagus nerve, in response to stimulation of pulmonary stretch receptors, rapidly ceased on institution of HFPPV, thus inhibiting inspiration.

Rare case reports of the use of HFPPV in the treatment of respiratory failure were published, with encouraging but inconclusive results. In 1977, Klain et al described a technical modification of HFPPV. Gases were delivered under high pressure (30 to 50 psig; 1,600 to 2,600 mm Hg) through a very small cannula (1 to 2 mm in diameter) inserted into the upper airway. This technique was defined as high-frequency jet ventilation (HFJV). Gases are accelerated during their passage through the cannula (injector), and their pressure decreases. The jet stream that exits from the distal opening of the injector (nozzle) enters a space where a much larger volume of static gases is present (mixing chamber). In clinical practice, this space is represented by the upper airways or an endotracheal tube. An energy transfer occurs, with the jet stream losing speed and pressure while it accelerates the gases present in the mixing chamber. If additional gases are delivered to a side port communicating with the mixing chamber, they will be "entrained" to replace those which have moved forward. Increasing the gas source pressure will provide larger entrained volumes, without substantial change in peak inspiratory pressure.

Klain and Smith developed an emergency ventilator that could be used without the need for orotracheal intubation. The characteristic of this ventilator, which can deliver variable tidal volumes without increasing airway pressure, closely matches the specifications of the hypothetical device that might be useful in some forms of respiratory failure as previously described. In the past three years, a number of clinical studies have been performed to define the conditions in which HFJV could be more advantageous than conventional support.

TECHNICAL CONSIDERATION

The ventilator we used is essentially a timer that
drives a solenoid valve. Respiratory rate (solenoid valve frequency) and inspiratory/expiratory time (solenoid valve duty cycle) can be adjusted independently. Gases are delivered under a pressure of 0 to 50 psig from an air-oxygen blender, which is connected to pressurized gas sources (tanks, centralized medical gas supply, etc). A short inspiratory line of low volume and compliance connects the air-oxygen mixture to an injector cannula having a diameter of 1 to 1.6 mm. This is placed into the removable port of a bronchoscopic swivel connector and inserted into a 5 to 10-mm-diameter endotracheal tube. A separate air-oxygen source is attached to the side port of the swivel connector, to provide gases for entrainment. A threshold resistor placed into the expiratory line allows application of PEEP. The response time of the solenoid valve (10 msec) is adequate to deliver accurate tidal volumes for respiratory rates greater than 300 breaths/min, with inspiratory time of 25 to 35 percent for each respiratory cycle.

**Clinical Experience**

In the last two years, 39 patients at our institution have had respiratory failure that did not respond to conventional means of mechanical ventilation. Criteria used to reach this conclusion included inability to obtain a PaO₂ > 70 mm Hg on FIO₂ = 0.40 or a PaH of < 7.35 units, with any manipulation of IMV, tidal volume, and PEEP.

A review of all patients treated (Tables 1 and 2) leads to some general considerations. HFJV was always successful in restoring, at least temporarily, adequate alveolar ventilation and arterial oxygen (PaO₂ > 70 mm Hg on FIO₂ 0.40; PaCO₂ < 50 mm Hg; and PaH 7.35 to 7.45 units) when the major source of respiratory failure was large airway disruption (bronchopleural and tracheoesophageal fistulas). Fifteen of the 20 patients with airleaks so large that conventional mechanical ventilation could not provide adequate support survived. Indeed, in only two patients was progression of respiratory failure the ultimate cause of death.

When HFJV was instituted in patients who had respiratory failure associated with barotrauma (pneumothorax or pneumomediastinum) with or without lung fibrosis, the success rate was considerably lower. Only 3/19 patients survived, although improvement of blood gases could be obtained in 15/19 patients. In these patients HFJV was instituted when sequential system failure or the patient's underlying disease had progressed to the point that improvement of oxygenation alone was not sufficient to reverse the lethal trend. Furthermore, in all patients on whom autopsy was performed, profound and irreversible modifications of lung structure, consistent with the terminal picture common to many forms of respiratory failure, were consistently found. Respiratory failure associated with multisystem failures always carries an extremely poor prognosis especially in immunosuppressed patients. It is not logical to expect that changing the modality of respiratory support may radically improve the picture.

**Table 1—High-Frequency Jet Ventilation (HFJV): Clinical Experience in 39 Patients**

<table>
<thead>
<tr>
<th>Cause of Respiratory Failure</th>
<th>No. of Patients</th>
<th>Survivors</th>
<th>Temporary Normalization of PaO₂/PaCO₂</th>
<th>Duration of HFJV, days*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major airway disruption</td>
<td>20</td>
<td>15</td>
<td>20</td>
<td>1-46</td>
</tr>
<tr>
<td>Acute respiratory failure</td>
<td>19</td>
<td>3</td>
<td>15</td>
<td>2-30</td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
<td>18</td>
<td>35</td>
<td>2-30</td>
</tr>
</tbody>
</table>

*Median duration of ventilation = 8 days.

**Table 2—Primary Acute Respiratory Failure: Analysis of 19 Patients Treated with HFJV**

<table>
<thead>
<tr>
<th>Reason for Discontinuing Conventional Ventilation</th>
<th>Diagnosis</th>
<th>Associated Problems</th>
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<tbody>
<tr>
<td>Barotrauma in the presence of peak airway pressure &gt;70 cm H₂O</td>
<td>Gram negative (5), viral (1), or <em>Pneumocystis carinii</em> (2) pneumonia</td>
<td>Disseminated cancer (8)</td>
</tr>
<tr>
<td>Severe hypoxemia (8)</td>
<td></td>
<td>Immunosuppression (8)</td>
</tr>
<tr>
<td>Diffuse lung fibrosis causing peak airway pressure &gt;70 cm H₂O</td>
<td>Prolonged chemotherapy and radiation therapy (5)</td>
<td>Disseminated cancer (8)</td>
</tr>
<tr>
<td>Severe hypoxemia (9)</td>
<td>Bone marrow transplant within prior 3-6 months (4)</td>
<td>Immunosuppression (9)</td>
</tr>
<tr>
<td>Diffuse pulmonary hemorrhage (2)</td>
<td>Chemotherapy-induced thrombocytopenia</td>
<td>Renal failure (5)</td>
</tr>
<tr>
<td> </td>
<td> </td>
<td>Coagulopathy (7)</td>
</tr>
</tbody>
</table>

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A detrimental effect generally associated with positive pressure ventilation, especially when PEEP is added, is depression of cardiac function. The explanations offered range from increased intrapleural pressure, which decreases venous return to the right heart, to higher pulmonary vascular resistances, overdistension of the right ventricle, and deformation of the left ventricular cavity.

Several therapeutic interventions are possible to reverse the cardiac embarrassment, such as intravascular volume expansion and inotropic drugs. Each approach has well-recognized advantages and disadvantages. Several authors have suggested that allowing the patient to support part of their minute ventilation spontaneously using (IMV) may also improve cardiac output by improving venous return. This contention has been questioned by others. The explanation of the contrasting observations may depend on the increased aortic transmural pressure associated with lower intrapleural pressure. This represents the increased afterload for the left ventricle; thus, in patients with very uncomplacent lungs, the same inspiratory effort that increases venous return to the right heart may decrease output from the left ventricle. Ideally, intrapleural pressure should decrease during ventricular emptying, to enhance venous return, and increase during ventricular contraction, to reduce transmural aortic pressure and therefore afterload.

With conventional ventilation, each breath spans several heart beats, so that synchronization of respiration and heart rates is impossible. HFJV, on the other hand, is normally delivered at rates of 100 to 150 breaths/min, very similar to the natural rate of cardiac contraction. Synchronization of HFJV with the cardiac cycle is, therefore, theoretically possible, so that inspiration coincides with ventricular contraction and expiration with ventricular relaxation. Transmural aortic and right atrial pressures may then be separately influenced with each breath.

The results of attempts at synchronization of peak inspiratory pressure with the openings of the aortic valve are summarized in Table 3. Ejection fraction constantly increased when transmural aortic pressure was decreased during ventricular contraction. Although no conclusions are warranted by a threepatient sample, further investigation is certainly justified.

The empirical clinical findings indicate that HFJV can be successful in some forms of respiratory failure. It is, however, apparent that the introduction and utilization of a new technology demands extensive experimental verification of each theoretical assumption. HFJV introduces new concepts in respiratory management. Gas source pressure, injector cannula diameter, and gas entrainment are not considered during conventional ventilation. Thus, principles of ventilation with HFJV in clinical practice require identification of rules and creation of nomograms to relate manipulation of variables unique to HFJV to the results desired. As with all other forms of mechanical ventilation, complete understanding of advantages and limits of HFJV will require extensive clinical use.

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Table 3—Cardiac Performance with Synchronized HFJV

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Ejection Fraction, %</th>
<th>Cardiac Output, L/min</th>
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<tbody>
<tr>
<td></td>
<td>E</td>
<td>A</td>
</tr>
<tr>
<td>1</td>
<td>57</td>
<td>83</td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>75</td>
</tr>
<tr>
<td>3</td>
<td>61</td>
<td>70</td>
</tr>
</tbody>
</table>

*A, Asynchronous jet ventilation; S, peak inspiratory pressure on HFJV synchronous with the opening of the aortic valve.

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