High Frequency Jet Ventilation and Intermittent Positive Pressure Ventilation*

Effect of Cerebral Blood Flow in Patients After Open Heart Surgery

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Attenuation of ventilator-synchronous pressure fluctuations of intracranial pressure has been demonstrated during high frequency ventilation in animal and human studies, but the consequences of this effect on cerebral blood flow have not been investigated in man. We compared the effects of high frequency jet ventilation and intermittent positive pressure ventilation on CBF in 24 patients investigated three hours after completion of open-heart surgery. The patients were investigated during three consecutive periods with standard sedation (morphine, pancuronium): a. IPPV; b. HFJV; c. IPPV. Partial pressure of arterial CO₂ (PaCO₂: 4.5-5.5 kPa) and rectal temperature (35.5 to 37.5°C) were maintained constant during the study. The CBF was measured by intravenous ¹³³Xe washout technique. The following variables were derived from the cerebral clearance of ¹³³Xe: the rapid compartment flow, the initial slope index, i.e., a combination of the rapid and the slow compartment flows, and the ratio of fast compartment flow over total CBF (FF). Compared to IPPV, HFJV applied to result in the same mean airway pressure did not produce any change in pulmonary gas exchange, mean systemic arterial pressure, and cardiac index. Similarly, CBF was not significantly altered by HFJV. However, important variations of CBF values were observed in three patients, although the classic main determinants of CBF (PaCO₂, cerebral perfusion pressure, Paw, temperature) remained unchanged. Our results suggest that in patients with normal systemic hemodynamics, the effects of HFJV and IPPV on CBF are comparable at identical levels of mean airway pressure.

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Intermittent positive pressure ventilation, particularly when applied with positive endexpiratory pressure, improves blood oxygenation by increasing volume and surface area of the lung for gas exchange. However, changes of lung volume and increased airway pressure have important side effects on cardiac output and regional blood flow. The magnitude of these hemodynamic changes is largely dependent on Paw and pulmonary compliance as well as on baroreceptor reflexes and blood volume. To avoid these untoward effects, Oberg and Sjöstrand proposed a mechanical ventilation technique using low tidal volumes at a high respiratory rate. However, to obtain an adequate pulmonary gas exchange, Paw during HFJV had to be similar to Paw during IPPV. If lung inflation was moderate and the regulation of systemic arterial pressure by baroreceptors was not modified, systemic hemodynamic effects of HFJV and IPPV were similar for identical values of Paw. In contrast, HFJV was less detrimental on the cardiovascular system than IPPV in case of cardiocirculatory shock.

Transmission of intrathoracic pressure to the intracranial space occurs through the venous system or directly on the cerebrospinal fluid space via thoracic vertebral foramina. Impairment of cerebral blood outflow across valveless veins and an increased cerebrospinal fluid pressure can increase cerebral venous and intracranial pressures. Results of studies in animals and in man have, however, demonstrated that, compared to conventional mechanical ventilation, HFJV had a similar effect on intracranial pressure.

The specific relationship between cerebral venous pressure and ICP, and the mechanisms of interaction of these two pressures on CBF are poorly understood. The effects of intracranial hypertension and an increased cerebral venous pressure on CBF are attenuated by the CBF autoregulation, the reflex venoarteriolar contraction, and the waterfall mechanism. Rapid fluctuations in ICP induced during IPPV may result from momentary variations in brain volume and may be associated with decreased CBF. However, animal studies have shown that CBF is not influenced by the respiratory variations of ICP and cerebral venous pressure for both ventilatory supports (IPPV, HFJV) at different values of ICP and Paw.

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Cerebral Blood Flow after Open Heart Surgery (Pittet, Forster, Suter)

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Since the effect of ventilatory modes on CBF has not yet been studied in humans, the purpose of this study was to compare the effect of HFJV and of IPPV on cerebral and systemic hemodynamics in patients presenting no CNS pathologic condition.

METHODS

Patients

Twenty-four patients (four women and 20 men), mean age 58.6 ± 4.8 years (mean ± SD), mean weight 72.3 ± 10.2 kg, were investigated at least two hours after a classic intravenous anaesthesia (midazolam 0.1 mg/kg/h, fentanyl 3.0 ± 0.8 mg) for elective open-heart surgery. Patients presenting unstable angina, unilateral pulmonary pathology, chronic obstructive pulmonary disease, or asthma were excluded from the study. All patients gave their informed consent to the study which was accepted by the committee for ethics in human research of our institution.

Equipment

The HFJV was delivered with a respirator. Air and oxygen were supplied with a pressure of 4 atm, mixed with a blender and pulsed by an electronically controlled solenoid valve through a noncompliant 120 cm long and 0.7 cm inner diameter connecting tube. This tube was connected to an endotracheal jet-type tube with three separate lumens: the main lumen was used as conventional ventilation, the first auxiliary lumen (1 mm diameter) as the tracheal airway pressure monitoring according to Brichant et al., and the second auxiliary lumen as the "jet insufflation lumen" during HFJV (2 mm ID). Gas for entrainment by Venturi effect was provided by an anesthetic circuit connected to a respirator delivering a constant flow (30 L/min) of heated and humidified gas at the same FIO2 as jet ventilation. The expiration was allowed by an open T-shaped piece without a PEEP valve. The driving pressure of the ventilator, inspiratory/expiratory time ratio (I/E), and respiratory frequency were independently adjustable.

The IPPV was administered using a volumetric respirator connected to the main lumen of the jet endotracheal tube. Gases were humidified and heated to 37°C using a cascade humidifier.

Respiratory and Hemodynamic Measurements

Airway pressure was measured using a 1 mm ID polyethylene catheter connected to a quartz pressure transducer and recorded on a chart recorder. The Paw was calculated electronically. Dynamic total respiratory compliance during IPPV was calculated by dividing peak airway pressure by inspiratory tidal volume.

Mean systemic arterial pressure, mean right atrial pressure, mean pulmonary arterial pressure, and pulmonary capillary wedge pressure were measured using a radial arterial cannula and a 7-F triple-lumen, Swan-Ganz catheter, respectively, connected to calibrated quartz pressure transducers positioned at the midaxillary line. Cardiac output was measured in duplicate using thermocoupling technique and a bedside Edwards computer. Cardiac index, total systemic vascular resistance index, and total pulmonary vascular resistance index were calculated using standard equations.

Systemic arterial blood samples were drawn for the measurement of PaO2, PaCO2, and pH with standard electrodes within one minute following the measurement of cardiac output. Oxygen saturation of hemoglobin (SaO2) was measured with a CO-oximeter.

The CBF was measured using a cerebralograph, according to a modified technique, after injection of 10 mCi of 133Xe diluted in a saline solution into the vena cava superior during end-expiration. The CBF was measured using three hemispheric detection probes placed on either side of the head at precisely identical anatomic localization. End-tidal 133Xe was monitored by constant aspiration of expired gas through a catheter placed in the main lumen of the endotracheal jet tube. An interval of 20 minutes was allowed between two measurements.

Based on the cerebral clearance of 133Xe, the following variables of cerebral hemodynamics were calculated (mean of six detectors): (1) P; fast compartment flow which is accepted to be gray matter flow; (2) ISI: modified initial slope index according to Risberg et al., representing the monoexponential slope of the early part of the 133Xe concentration curve between 0.5 and 1.5 minutes; this index reflects clearance from fast (2/3) and slow (1/3) compartments and represents the mean CBF; and (3) FF: fractional flow, ie, the ratio of gray matter flow over total CBF. The quality of CBF measurements was assessed by determination of the standard deviation between measured and theoretically expected curves. It was considered satisfactory when this standard deviation was below 1.5. In addition, individual variability of CBF determinations was documented by calculating the variation coefficient (SD/mean of the 24 patients) of CBF values during each of the experimental sessions.

Procedure

The effects of IPPV and HFJV were measured in the surgical intensive care unit two to four hours after the end of cardiac surgery. The study was started when the patient had reached a rectal temperature between 35.5 to 37.5°C, stable systemic hemodynamics with a mean systemic arterial pressure within ± 10 percent of preoperative value, and a stable sinus heart rate. Patients with postoperative cardiopulmonary instability were not included in the study. Prior to the beginning of the study, the patient received 5 mg morphine and 0.1 mg/kg pancuronium, intravenously.

Each patient was examined during three periods of 11 minutes separated by two intervals of 20 minutes: (1) first control period: IPPV1; (2) experimental period: HFJV; (3) second control period: IPPV2.

The FIO2 was maintained at 0.4. The inspiratory minute volume was adjusted to obtain a PaCO2 between 4.5 to 5.5 kPa (IPPV: by changing tidal volume; HFJV: by changing driving pressure of the ventilator).

Statistical Analysis

The data (mean ± SE) of the three experimental sessions were compared with an analysis of variance for repeated measurements followed by a modified Student's t-test (Bonferroni method) when the F-ratio resulted in a p value < 0.05.

RESULTS

The investigation took place 205 ± 60 minutes after the end of the surgery. For technical reasons in Table 1—Respiratory Variables Determined During the Three Experimental Periods*

<table>
<thead>
<tr>
<th></th>
<th>IPPV1</th>
<th>HFJV</th>
<th>IPPV2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency, min⁻¹</td>
<td>10</td>
<td>100</td>
<td>10</td>
</tr>
<tr>
<td>I/E, %</td>
<td>0.33</td>
<td>0.5</td>
<td>0.33</td>
</tr>
<tr>
<td>Driving pressure, bar</td>
<td>—</td>
<td>1.0 ± 0.1</td>
<td>—</td>
</tr>
<tr>
<td>Vr, ml·kg⁻¹</td>
<td>9.2 ± 0.3</td>
<td>—</td>
<td>9.2 ± 0.3</td>
</tr>
<tr>
<td>Paw, mm Hg</td>
<td>4.5 ± 0.4</td>
<td>4.1 ± 0.4</td>
<td>4.8 ± 0.5</td>
</tr>
<tr>
<td>PIP, mm Hg</td>
<td>17.4 ± 1.3</td>
<td>12.4 ± 1.2</td>
<td>18.8 ± 1.4</td>
</tr>
<tr>
<td>PaCO2, kPa</td>
<td>4.9 ± 0.1</td>
<td>5.0 ± 0.1</td>
<td>5.0 ± 0.1</td>
</tr>
<tr>
<td>PaO2, kPa</td>
<td>16.4 ± 1.1</td>
<td>17.5 ± 1.2</td>
<td>17.1 ± 1.6</td>
</tr>
</tbody>
</table>

*Mean ± SE of 24 patients during IPPV, and HFJV, and of 20 patients during IPPV2, I/E, inspiratory/expiratory time ratio; Paw, mean airway pressure; PIP, peak inspiratory pressure. 

p<0.05 between IPPV, and HFJV.
measuring CBF, the latter was not measured in four patients during the second control period. Respiratory data are summarized in Table 1. To obtain PaCO₂ between 4.5 and 5.5 kPa, a tidal volume of 9.2 ± 0.3 ml/kg was used during IPPV and the driving pressure of the HFJV ventilator was set at 1.0 ± 0.1 bar. The PaO₂ values were similar during the two types of ventilatory support. A dynamic total respiratory compliance of 50 ± 5 ml/cm H₂O was measured during IPPV. Peak inspiratory pressure was significantly (p<0.05) lower during HFJV than during the two periods of IPPV, whereas Paw remained in the same range during both modes of ventilation. Hemodynamic data are summarized in Table 2. No significant differences in any of the hemodynamic variables were detected between IPPV and HFJV.

Cerebral hemodynamic data are presented in Table 3. The standard deviation between measured and calculated curves of CBF values was below 1.5, indicating a good quality of CBF measurement and comparable between the three measurements. Average F₁ and ISI values were not significantly modified by HFJV. Fractional flow remained stable between both modes of ventilation, including patients with a large change of F₁ or ISI. Figure 1 presents individual ISI data. The ISI ranged from 26.1 to 61.9 during IPPV, and from 26.0 to 68.3 during IPPV₂. The range of ISI values was increased by 30 percent during HFJV (19.9 to 72.8). Although classic main determinants of CBF (PaCO₂, cerebral perfusion pressure, Paw, temperature) were stable during both modes of ventilation (Table 4), application of HFJV induced in one patient (patient 3) an increase and in two patients (patients 7 and 13), a decrease in CBF (F₁ and ISI) of more than 40 percent from IPPV₁ values.

**Table 2—Hemodynamic Variables Determined During the Three Experimental Periods**

<table>
<thead>
<tr>
<th></th>
<th>IPPV₁</th>
<th>IPPV₂</th>
<th>HFJV</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP, mm Hg</td>
<td>94 ± 4</td>
<td>95 ± 4</td>
<td>96 ± 4</td>
</tr>
<tr>
<td>HR, beats/min⁻¹</td>
<td>96 ± 3</td>
<td>96 ± 2</td>
<td>96 ± 2</td>
</tr>
<tr>
<td>CI, L/min·m⁻²</td>
<td>2.5 ± 0.1</td>
<td>2.5 ± 0.1</td>
<td>2.6 ± 0.1</td>
</tr>
<tr>
<td>RAP, mm Hg</td>
<td>7 ± 1</td>
<td>9 ± 1</td>
<td>8 ± 1</td>
</tr>
<tr>
<td>MPAP, mm Hg</td>
<td>18 ± 2</td>
<td>22 ± 2</td>
<td>20 ± 1</td>
</tr>
<tr>
<td>PCWP, mm Hg</td>
<td>9 ± 1</td>
<td>10 ± 1</td>
<td>9 ± 1</td>
</tr>
<tr>
<td>SVRI, dynes·cm⁻⁵·m⁻²</td>
<td>955 ± 61</td>
<td>940 ± 63</td>
<td>932 ± 42</td>
</tr>
<tr>
<td>PVRI, dynes·cm⁻⁵·m⁻²</td>
<td>95 ± 12</td>
<td>118 ± 13</td>
<td>101 ± 10</td>
</tr>
</tbody>
</table>

*Mean ± SE of 24 patients during IPPV, and HFJV, and of 20 patients during IPPV₂.*

**Table 3—Cerebral Hemodynamic Variables Determined During the Three Experimental Periods**

<table>
<thead>
<tr>
<th></th>
<th>IPPV₁</th>
<th>IPPV₂</th>
<th>HFJV</th>
</tr>
</thead>
<tbody>
<tr>
<td>F₁ (ml/100 g·s⁻¹)</td>
<td>56 ± 4</td>
<td>53 ± 6</td>
<td>56 ± 4</td>
</tr>
<tr>
<td>ISI (units)</td>
<td>44 ± 2</td>
<td>42 ± 3</td>
<td>46 ± 3</td>
</tr>
<tr>
<td>FF (%)</td>
<td>74 ± 2</td>
<td>78 ± 3</td>
<td>76 ± 3</td>
</tr>
</tbody>
</table>

*Mean ± SE of 24 patients during IPPV, and HFJV, and of 20 patients during IPPV₂.*

**Table 4—Mean Arterial Pressure, PaCO₂, Rectal Temperature, and Mean Airway Pressure of the Three Patients With Major Changes of CBF During Application of HFJV**

<table>
<thead>
<tr>
<th>Patient</th>
<th>IPPV₁</th>
<th>IPPV₂</th>
<th>HFJV</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISI, units</td>
<td>3</td>
<td>40</td>
<td>73</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>3</td>
<td>81</td>
<td>81</td>
</tr>
<tr>
<td>PaCO₂, kPa</td>
<td>3</td>
<td>5.54</td>
<td>5.52</td>
</tr>
<tr>
<td>Rectal temperature, °C</td>
<td>3</td>
<td>35.6</td>
<td>35.6</td>
</tr>
<tr>
<td>Paw, mm Hg</td>
<td>3</td>
<td>7.5</td>
<td>5.0</td>
</tr>
</tbody>
</table>

*Discussion*

In the present study, HFJV applied with an identical Paw as during IPPV did not modify pulmonary gas exchange, MAP, and CI. Similarly, no significant differences in mean CBF values (F₁, ISI) were noted. Classic main determinants of CBF remained stable and were comparable during both modes of ventilation. The PaCO₂ and rectal temperature were maintained in a narrow range fixed by our experimental protocol, and MAP did not change. Furthermore, Paw did not vary throughout the experiment, resulting in stable arterial oxygenation and probably in a similar lung volume, and with an identical effect on systemic hemodynamics. This is in agreement with previous studies showing a close relationship between Paw and systemic hemodynamics. In particular, our results confirm that HFJV has no different effect on cardiac output than during IPPV at similar Paw. Our patients had a relatively low dynamic total respiratory compliance (50 ml/cm H₂O), most probably due to a moderate interstitial pulmonary edema due to cardiopulmonary bypass. This relatively low compliance may have attenuated the influence of variations of intrathoracic pressure on cardiac filling pressures, ventricular volumes, and cardiac output.

The CBF measuring method used in the present study (¹³³Xe washout) provides an average flow over a period of 11 minutes. It does not detect changes of CBF that might occur during the course of a single respiratory cycle. Thus, although instant CBF could...
change in relation to ventilator-synchronous fluctuations of ICP, our results demonstrate that these variations of ICP are too small in our patients to have an influence on average flow measured over several minutes. Moreover, as shown in animal studies, respiratory fluctuations of the ICP waveform which most likely reflect variations of cerebral blood volume, are not very important at normal ICP. In presence of an abnormal cerebral compliance, the results of animal studies are conflicting and depend on the type of cerebral injury. Further studies investigating the effect of increase of intrathoracic pressure in patients with poor cerebral compliance are necessary.

Although we did not find significant differences of mean CBF values between HFJV and IPPV for the group studied, HFJV induced in three patients important variations of CBF (Fig 1) in spite of stable classic main determinants of CBF, ie, PaCO₂, cerebral perfusion pressure, Paw, and temperature (Table 4). Several of the following mechanisms could explain these findings: first, this could be related to technical problems of CBF measurement or the quality of our CBF determination was satisfactory, even for the three patients who demonstrated large changes in CBF during HFJV. In addition, the variability of CBF measurement with this method is below 10 percent, and the mean change in these three patients was more than 40 percent. Second, the state of stimulation may have changed during HFJV because the intensive care environment could have been different between the IPPV and HFJV period, even with 5 mg morphine given before the beginning of the study. In addition, similar doses of midazolam and fentanyl were administered during surgery, and the time between the end of surgery and the beginning of the study was similar for all patients. Third, CBF values could also be modified by other factors that we could not assess; eg, the state of neurogenic control of cerebral blood volume can be different for each patient. The marked response of CBF to the application of HFJV in three patients suggests that CBF also depends on uncontrolled and possibly determinant factors.

In conclusion, the results of this study indicate that HFJV has no specific effects on CBF when compared to a conventional mode of ventilation at similar Paw in patients with normal systemic hemodynamics. These observations are in agreement with animal studies. Whether HFJV can preserve CBF in patients with poor cerebral compliance or with circulatory shock, a situation during which HFJV has been shown to induce a lesser degree of hemodynamic impairment than during IPPV when identical levels of Paw are applied, remains to be investigated.

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