Case Report

High-frequency percussive ventilation during surgical bronchial repair in a patient with one lung

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We report the case of a patient that had undergone a left pneumonectomy during which a double-lumen tube was used and an undetected right bronchial laceration occurred. After diagnosis the patient underwent a second operation to repair the tear. The role of high-frequency percussive ventilation in enabling adequate gas exchange during the bronchial repair is described and discussed.

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Tracheobronchial rupture usually requires prompt surgical attention. Ventilatory strategies include the use of double-lumen tracheal tubes or single-lumen tubes with a blocker to occlude the damaged bronchus,1–3 jet ventilation by way of a catheter inserted down the tracheal tube and across the bronchial rupture,4–6 jet ventilation by way of a catheter inserted via the operative field beyond the bronchial rupture,7 and cardiopulmonary bypass when nothing else is possible.8 We describe a ventilatory strategy used during repair of a 3 cm long linear rupture in the right main and intermediate bronchi along their medial margin at the union of the membranous and cartilaginous zones. The lesion was generated, and not detected, 8 h previously during repetitive attempts to site a double-lumen tube for elective left pneumonectomy. During reconstructive surgery high-frequency percussive ventilation (HFPV) and a single-lumen tracheal tube were used. HFPV was developed in the early 1980s by F. M. Bird, and initially used in the treatment of patients with acute respiratory failure owing to smoke inhalation9,10 and trauma,11 and to control intracranial pressure.12 Further studies showed an improvement in gas exchange in cases refractory to conventional mechanical ventilation in patients with acute respiratory distress syndrome.13,14 This ventilation technique has also been applied to paediatric patients and neonates.15,16 However, despite its aforementioned uses in intensive care practice, little is known about HFPV during anaesthesia.

Case report

A 60-yr-old, otherwise healthy man, underwent left pneumonectomy owing to a bronchoepithelial carcinoma. A double-lumen Carlen tracheal tube (39 F) was easily placed in the trachea under laryngoscopy. However, correct placement of the tube was achieved only on the third attempt (with the help of a stylet). Thereafter exclusion of each lung was checked before proceeding with surgery. After an uneventful operation the patient’s trachea was extubated and the spontaneously breathing patient was transferred to the intensive care unit (ICU) for postoperative monitoring. His vital signs were within the normal range. Twelve hours after surgery a routine chest X-ray disclosed a pneumomediastinum. A subsequent CT scan of the thorax showed air in the mediastinum both at its upper portions and at the axial level of the intermediate bronchus (Fig. 1). A fibreoptic bronchoscopy was performed and revealed a 3 cm-long linear rupture in the right main and intermediate bronchi along their medial margin at the union of the membranous and cartilaginous zones. Emergency surgical intervention was considered mandatory. The same surgical team but a different group of anaesthetists were enrolled.
in the second operation. The patient was brought to the operating theatre and routine monitoring (invasive arterial pressure, ECG and pulse oximetry) was started. Anaesthesia was induced with propofol and succinylcholine (1 mg kg$^{-1}$) was given to facilitate orotracheal intubation with a 8.5 single-lumen reinforced tracheal tube. The patient was placed in the left lateral position. Anaesthesia was maintained with propofol and fentanyl and neuromuscular block was continued with cisatracurium besilate. The residual right lung was mechanically ventilated in volume control mode (tidal volume 7 ml kg$^{-1}$; I:E ratio=1:3; ventilatory frequency 12 bpm). After tracheal intubation the end-tidal carbon dioxide was monitored and arterial blood gas analysis before thoracotomy confirmed normoxia and normocapnia (Table 1). The thoracotomy was performed through the fourth right intercostal space. After opening the mediastinal pleura the bronchial rupture was confirmed and repaired with continuous sutures and an intercostal muscle flap. After opening of the chest wall HFPV (500 cycles min$^{-1}$; infinite inspiratory time, i.e. jet ventilator-like) was instigated to minimize air leakage (Table 1, Fig. 2A). Twenty minutes later arterial blood gas analysis revealed hypercapnia and a decrease in pH. To reduce the $P_{aCO_2}$, the HFPV settings were modified (Table 1) to 300 cycles min$^{-1}$. However, 20 min later only the oxygenation had improved, so the HFPV settings were again modified: the percussion rate was returned to 500 cycles min$^{-1}$ and an expiratory oscillatory phase was introduced (i.e. bilevel HFPV), creating a 7 cm H$_2$O pressure gap between inspiration and expiration (4 bpm). After 20 min the $P_{aCO_2}$ had decreased by 0.67 kPa. Up to this point the bronchi had not been repaired and air leakage was still present. Ventilation was modified for the last time by increasing the pressure gap between inspiration and expiration to 11 cm H$_2$O (Fig. 2B) and this reduced the leak even before the repair was completed. After 20 min there was a further reduction in $P_{aCO_2}$. After this measurement, the repair was completed, two chest drainage tubes were positioned, and the chest wall closed, ending the procedure (that lasted ~1 h and 20 min). The patient recovered in the ICU under conventional ventilation; the tracheal tube was removed 24 h later and a CT scan of the thorax performed 4 days after surgery confirmed the resolution of the bronchial leak. The patient’s heart rate and blood pressure remained stable throughout surgery and recovery in the ICU.

Table 1 Surgical steps, arterial blood gas analysis and ventilatory settings during volume-controlled mode (VCM) and during HFPV under different settings.

<table>
<thead>
<tr>
<th>Surgical steps</th>
<th>VCM</th>
<th>HFPV1</th>
<th>HFPV2</th>
<th>HFPV3</th>
<th>HFPV4</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{aO_2}$ (kPa)</td>
<td>21.9</td>
<td>52.0</td>
<td>33.4</td>
<td>39.0</td>
<td>29.6</td>
</tr>
<tr>
<td>$P_{aO_2}/F_{I_2O}$ (kPa)</td>
<td>43.73</td>
<td>52.0</td>
<td>47.86</td>
<td>77.86</td>
<td>59.19</td>
</tr>
<tr>
<td>$P_{aCO_2}$ (kPa)</td>
<td>6.3</td>
<td>8.5</td>
<td>8.4</td>
<td>7.7</td>
<td>7.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.30</td>
<td>7.22</td>
<td>7.22</td>
<td>7.25</td>
<td>7.30</td>
</tr>
<tr>
<td>RR (bpm)</td>
<td>12</td>
<td>–</td>
<td>–</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>$I$ (s)</td>
<td>1.25</td>
<td>–</td>
<td>–</td>
<td>13.8</td>
<td>13.8</td>
</tr>
<tr>
<td>$E$ (s)</td>
<td>3.75</td>
<td>0</td>
<td>0</td>
<td>1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>Cycles min$^{-1}$</td>
<td>–</td>
<td>500</td>
<td>300</td>
<td>500</td>
<td>500</td>
</tr>
<tr>
<td>MAP (cm H$_2$O)</td>
<td>–</td>
<td>22</td>
<td>19</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>MIAP (cm H$_2$O)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>23</td>
<td>23</td>
</tr>
<tr>
<td>MEAP (cm H$_2$O)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>PAP (cm H$_2$O)</td>
<td>–</td>
<td>33</td>
<td>28</td>
<td>33</td>
<td>33</td>
</tr>
<tr>
<td>PEEP (cm H$_2$O)</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
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</table>
Bronchial repair in a patient with one lung

**Discussion**

Bronchial repair is a challenging procedure both for the surgeon and the anaesthetist, even more so in a patient with only one lung. In our patient bronchial rupture was iatrogenic, resulting from double-lumen tracheal tube placement as has been reported previously. Distal airway lesions require the exclusion of the damaged lung, whereas tracheal ruptures are usually treated with high-frequency ventilation (HFV). All modes of HFV share at least three basic elements: a high pressure flow generator, a safety valve, and a breathing circuit connected to the patient. HFV implies a ventilatory frequency above 60 bpm (1 Hz), tidal volumes smaller than dead space, lower peak airway pressures and a more efficient gas exchange than that provided by conventional ventilation.

Although there is no universally accepted classification or defined nomenclature for these various methods, depending on the frequency used, the manoeuvres may be divided into high-frequency jet ventilation (HFJV) and its variant, high-frequency flow interruption, high-frequency oscillation (HFO), and high-frequency positive pressure ventilation (HFPPV). Basically, HFPPV uses lower frequencies (60–300 cycles min⁻¹), while HFO uses higher frequencies (60–2400 cycles min⁻¹). However, this classification does not take into account that HFO can also be used with low working frequencies. HFPPV was introduced more than 20 yr ago to overcome the drawbacks of other HFV modes (e.g. high-frequency oscillation, high-frequency jet ventilation).

HFPPV may be defined as a flow-regulated time-cycled ventilation that creates controlled pressure and delivers a series of high-frequency subtidal volumes in combination with low-frequency breathing cycles. The only system that delivers HFPPV is the VDR-4 (Volumetric Diffusive Respirator, Percussionaire Corporation, Sandpoint, ID, USA): a time-cycled pressure-controlled ventilator equipped with a high-frequency flow generator connected to a device (the phasitron) that provides the interface between the patient and the machine. The phasitron produces mini-bursts of subtidal volumes that generate intrapulmonary percussive waves, hence the denomination ‘percussive ventilation’.

HFPPV combines the positive aspects of conventional mechanical ventilation with those of HFV. It may operate as high-frequency jet ventilation, providing a continuous jet pulsation by the prolongation of inspiratory duration to infinity (Fig. 2A). It differs from high-frequency oscillatory ventilation (HFOV) because its expiratory phase is completely passive and from high-frequency jet ventilation (and HFOV) because of its open circuit. HFPPV can also provide ventilation under two diverse mean airway pressures (bi-level). Finally, it enables safe peak and mean airway pressures as these are determined by the ventilator’s physical properties. The percussion frequency constitutes a critical factor in the management of hypoxia and hypercapnia. In fact, at low percussion frequencies (180–240 cycles min⁻¹) convection of gases is prevalent (which translates into increased washout of carbon dioxide) whereas at high frequencies (300–600 cycles min⁻¹) the diffusion of gases predominates, a phenomenon that may be linked to the increased kinetics of the oxygen molecules.

In our patient ventilation was started with a HFJV-like pattern (500 cycles min⁻¹, infinite inspiratory time, high-frequency jet pulsation mode) with a steady-state mean airway pressure (Fig. 2A). Carbon dioxide retention ensued, and we proceeded to reduce the oscillation frequency from 500 to 300 cycles min⁻¹ (and airway mean pressure), but to no avail. The reduction of the oscillation frequency maintained the Pao₂/Fio₂ ratio and the Paco₂, was unchanged, despite the fact that mean airway pressure was reduced by 3 cm H₂O (from 22 to 19 cm H₂O). Although at this stage the patient’s gas exchange could be considered acceptable, we enhanced carbon dioxide removal by exploiting the bi-level ventilation mode. The VDR-4 allows the use of a low-frequency (about 4 bpm) bi-level mean airway pressure (23 and 16 cm H₂O during inspiratory and expiratory phases, respectively), which led to a 0.67 kPa-decrease in the Paco₂ 20 min after changing from the previous ventilatory strategy. However, it was considered that a larger gap in bi-level mean airway pressure could further improve carbon dioxide removal, and, indeed, when the mean expiratory airway pressure was 12 cm H₂O (Fig. 2B), the Paco₂ dropped to 7.2 kPa. During bi-level ventilation the calculated mean airway pressure was 22 cm H₂O, and the percussion frequency was switched back to 500 cycles min⁻¹. This was done to highlight the fact that carbon dioxide washout depended mainly on the pressure gap created by the bi-level ventilation mode.

![Fig 2](https://example.com/fig2.png)

*Fig 2* Airway pressure plotted against time during surgery to repair the right main bronchus. Each cycle represents one mini-burst generated by the high-frequency percussive ventilator (500 cycles min⁻¹).

(A) High-frequency percussive ventilation used as high-frequency jet pulsation. (B) Bi-level high-frequency percussive ventilation. In both panels the thick line represents instantaneous mean airway pressure.

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Throughout surgery oxygenation remained at a safe level and we were able to reduce the $F_{\text{O}_2}$, from 1 to 0.5. Additionally, the arterial blood pH rose from 7.22 to 7.3. The ability of HFPV to keep acceptable blood gas levels in the face of leaky ruptured bronchi stems from its capability of generating very high pulsatile flows, as the ventilator gas output is servo-adjusted to the output impedance. Interestingly, the VDR-4 itself operates with an open-air circuit that is intended to prevent lung hyperinflation. The system improves gas exchange as a result of both convective and diffusive mechanisms. Thus, the deleterious effects of air leakage on gas exchange were minimized and the surgeon could work on an almost immobile field. Finally, the success of the present approach was achieved in an unusual condition, that is, a patient with ruptured bronchi in his only lung. Under these circumstances the remaining lung had to allow for gas exchange even while partly deflated.

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