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High frequency percussive ventilation (HFPV)

Case reports

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Treatment of acute respiratory failure is still a hot issue in intensive care everyday practice: in the last few years high frequency ventilation techniques have been employed as a therapy for adult respiratory distress syndrome (ARDS) and acute respiratory failure (ARF). We applied high frequency percussive ventilation (HFPV) to 3 patients affected by ARDS or ARF, who did not improve after 24 hours of conventional mechanical ventilation (CMV). All our patient underwent 12 hours of HFPV, and showed an improvement of both respiratory exchange and radiological imaging. Even if the pathogenesis of ARF was quite different, in all patient we registered a good response and no complications.

Key words: **High frequency percussive ventilation - Respiratory failure - Phasitron - Humidification.**

In a previous article,[1] we described the principles of high frequency percussive ventilation (HFPV) and directed particular attention at the technical aspects and function of the volumetric diffusive respiration (VDR®4) system. In this article, we report on the clinical response to HFPV in selected cases from our experience with this method in intensive therapy.

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Several authors have reported their experience with HFPV in neonates with hyaline membrane disease,[2, 3] in patients with neuromuscular disease [4] and in those with acute respiratory failure following chest trauma.[5] In a pilot study on patients with cystic fibrosis, Natale *et al.*[6] compared the effects of HFPV with those of conventional chest physiotherapy. HFPV has also been studied in patients with post-traumatic intracranial pressure.[7, 8] Gallagher *et al.*[9] compared techniques of conventional mechanical ventilation (CMV) and HFPV in patients with respiratory failure following sepsis and trauma.

The use of HFPV has produced a significant improvement in lung gas exchanges at equal airway peak pressure and FiO_2. Moreover, the maneuver has been shown to maintain normocapnia, without causing hemodynamic alterations. Despite these positive results, HFPV is used only occasionally and no clinical protocols for its clinical use have yet been formulated.

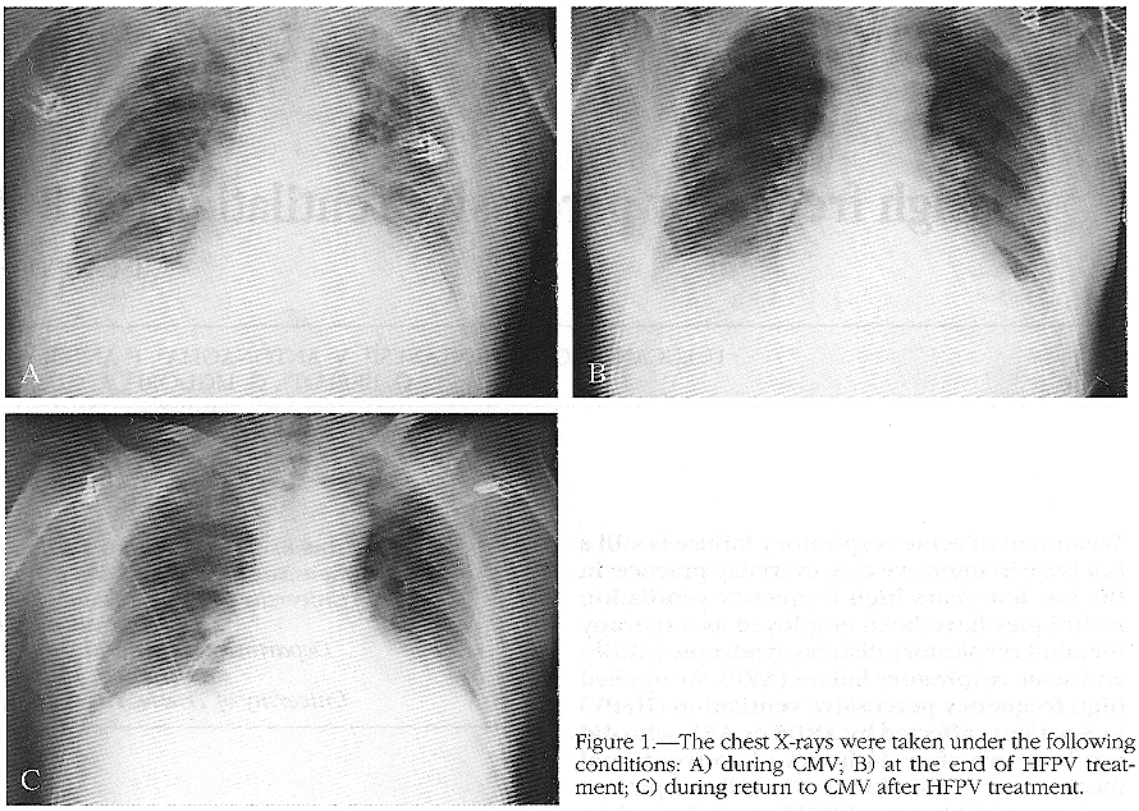

Figure 1.—The chest X-rays were taken under the following conditions: A) during CMV; B) at the end of HFPV treatment; C) during return to CMV after HFPV treatment.

Case series

Case 1.—A 71-year-old man, body weight 69 kg, (SAPS II 40), was transferred from General Surgery to the Intensive Care Unit (ICU) with a diagnosis of acute pancreatitis. On admission, the patient was alert and cooperative, breathing spontaneously with the aid of supplemental oxygen delivered by a mask (FiO_2=0.5). A prior CT scan demonstrated at the level of the abdomen tumefaction of the pancreas, accompanied by retro- and peritoneal effusion; at the level of the chest there was bilateral pleural effusion. According to the patient's medical history, he suffered from generalized obliterating arteriopathy and had been hospitalized 7 years earlier for myocardial infarction. Physical examination disclosed tachypnea, moist noises in the left lung base, a chest X-ray (Figure 1A-C) demonstrated thickening of the left lung base (Figure 1A). In light of the deteriorating blood gas profile (PaO_2/FiO_2=138.8; pH=7.47; PCO_2=31.6 mmHg), tracheal intubation was performed and constant flow CMV instituted (Puritan Bennet 7200, CA, USA), with Vt=10 ml/kg; RR=12 b/min; I/E=1: 2.5; PEEP=5 cm H_2O, FiO_2=0.7). Twelve hours later, oxygenation values did not improve (PaO_2/FiO_2=142.3; pH=7.33; $PaCO_2$=47.0 mmHg) and airway obstruction worsened due to abundant secretions; one cycle of HFPV (VDR®4) was given at an operating pressure (27 cm H_2O) equal to $^2/_3$ the peak CMV pressure.

A radiographic examination at the end of treatment demonstrated an increase in lung volumes, a reduction in the upper $^2/_3$ and a better visibility of the vascular system, without significant changes in the left lung base infiltration. A blood gas analysis performed 12 hours after the end of treatment, with CMV at constant volume, confirmed the maintenance of PAO_2/FiO_2=206, which remained stable over the following days. Figure 2 shows the trend of PaO_2/FiO_2 before, during, after and at 12 hours after treatment after return to CMV.

Case 2.—A 75-year-old man, body weight 80 kg, (SAPS II 33), was admitted to the ICU after surgery for a phlegmon of the neck complicated by mediastinitis. The patient suffered from ischemic heart disease; a control ECG demonstrated inferior necrosis of undetermined onset.

A postoperative chest X-ray demonstrated reduced expansion of the lung fields. Over the following days, the clinical picture worsened due to formation of parenchymal thickening of the left lung base; bronchoscopy was repeated to remove the large quantities of thick secretions.

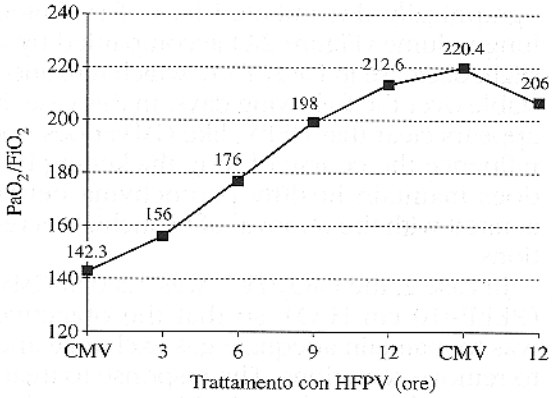

Figure 2.—Trend of PaO_2/FiO_2 before (CMV) and over the consecutive 12 hours of HFPV treatment. Note that the recovery of blood oxygenation is maintained also after return to CMV and over the following 12 hours (12 hours post-CMV).

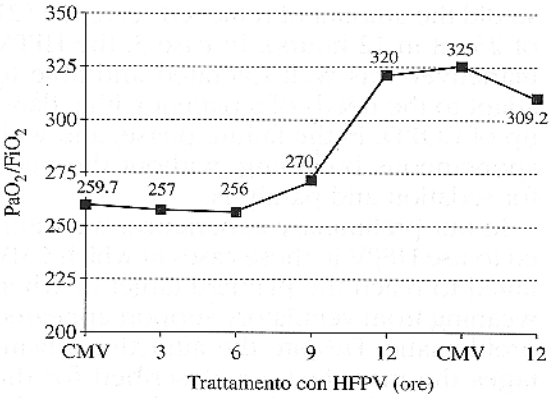

Figure 3.—PaO_2/FiO_2 before the start of CMV treatment, during treatment (hours of HFPV) and at the end and at 12 hours after the return to CMV. Note that in the first 9 hours of treatment the PaO_2/FiO_2 value remained substantially unchanged but improved considerably after removal of secretions.

After 9 days on CMV (Puritan Bennet 7200, CA, USA), with Vt=8 ml/kg; RR=12 b/min; I/E=1: 3; positive and-expiratory pressure (PEEP)=10 cm H_2O; FiO_2=0.4, although the gas exchange values remained acceptable (PaO_2/FiO_2=259.7), continued abundant secretions that were difficult to remove made weaning from the CMV impossible. The patient received a cycle of HFPV (VDR®4) at an operating pressure set to $^2/_3$ the initial value, with a percussion frequency of 800 cycles/min and FiO_2=0.3. Initially, the PaO_2/FiO_2 ratio remained near the starting values, but after several hours of treatment, the secretions were effectively liquefied and removed, leading to improved gas exchange, which was maintained 12 hours after the end of treatment (PaO_2/FiO_2=309.2) (Figure 3).

Case 3.—A 76-year-old man, body weight 50 kg, (SAPS II 30), affected with chronic obstructive pulmonary disease (COPD), left fibrothorax and sequelae of a cerebral ictus that had occurred 6 months earlier, was admitted to the ICU because of lack of recovery of spontaneous breathing following Miles' operation. Ventilatory support was continued using pressure support ventilation (PSV=19 cm H_2O) with PEEP=5 cm H_2O (Puritan Bennet 7200, CA, USA); a radiographic chest examination demonstrated the presence of an opacity in the left lung base. The following day, despite the increased pressure support (PSV=22 cm H_2O), the patient developed dyspnea and tachypnea with the effort of accessory respiratory muscles; blood gas analysis revealed PaO_2/FiO_2= 117.8, pH=7.48, $PaCO_2$=36.3 mmHg. Ventilation with HFPV (VDR®4) was instituted.

The HFPV parameters were set to ensure that the habitual $PaCO_2$ values for a patient were reached. After a brief initial period of adaptation, the clinical conditions gradually improved as did oxygenation, with a trend to stabilization of blood gas values: PaO_2/FiO_2=319; pH=7.46; $PaCO_2$=41.4 mmHg. Throughout treatment (12 h), the patient did not manifest intolerance to HFPV, as shown in Figure 4. After

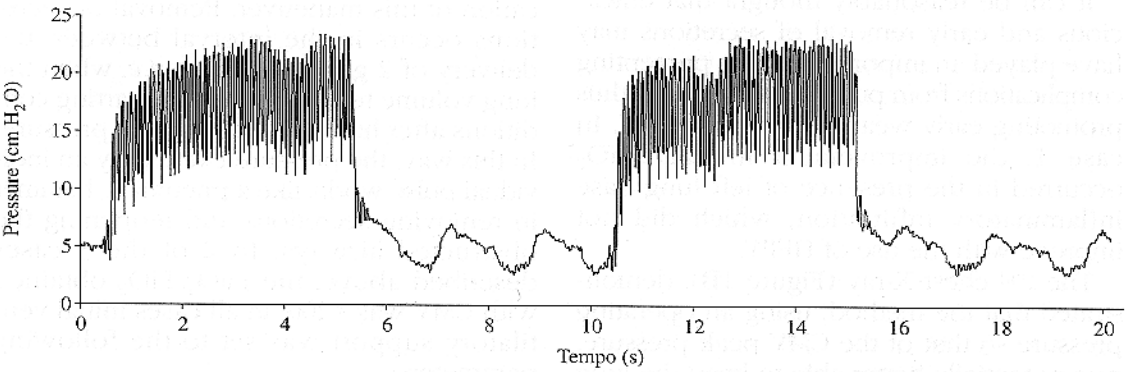

Figure 4.—Note that the pressure curve during HFPV is changed by spontaneous breathing. As the circuit is open to room air, the patient can enter in any phase of the respiratory cycle delivered by the device, without causing overpressurization in the system.

return to PSV, the need for pressure support diminished significantly (PSV=15 cm H_2O), so that the patient could gradually be weaned from ventilatory support.

Discussion and conclusions

In the cases described above, the most salient result is the improvement in gas exchange over the 12 hours of treatment with HFPV (VDR®4); the result remained substantially unchanged at 12 hours and after return to CMV. The short-term duration of treatment was chosen with the intent to limit interference from confounding factors that could have masked the effect of HFPV. Two of the 3 patients were placed on CMV after sedation or curarization, which was maintained during the HFPV cycle. The only patient with spontaneous breathing was gradually adapted to the VDR®4 system ventilator (Figure 4) without sedation. No patients experienced hemodynamic alterations and/or barotrauma.

Numerous past studies have underlined the positive effect of high frequency ventilation and rapid chest wall compression on mucociliary transport and mucus clearance [10-14]. This mechanism may also be hypothesized in our patients, in agreement with the results of the pilot study on patients with chronic bronchopathologies who had been treated in spontaneous breathing with a simpler HFPV system (intrapulmonary percussive ventilation, IPV) [15].

It can be reasonably thought that efficacious and early removal of secretions may have played an important role in preventing complications from pulmonary infection, thus promoting early weaning from the CMV. In case 1, the improvement in PaO_2/FiO_2 occurred in the presence of left lung base inflammatory infiltration, which did not improve with the use of HFPV.

The 2nd chest X-ray (Figure 1B), demonstrated that the method, using an operating pressure 2/3 that of the CMV peak pressure, was potentially better able to keep the lung parenchyma better aerated.

With the return to CMV, there was radiographically documented loss of recruited lung volume (Figure 2A) accompanied by a slight decrease in PaO_2/FiO_2, which remained stable over the following days. In this case, it appears clear that HFPV, like CMV, does not influence the course of lung thickening but does maintain healthy parenchyma better aerated with the removal of bronchial secretions.

In case 2, the PaO_2/FiO_2 was >200 in CMV (PEEP=10 cm H_2O), so that the objective was to maintain adequate gas exchange and to remove secretions. The response to treatment can be seen from the biphasic trend of PaO_2/FiO_2, which in the first 9 hours remained substantially unchanged since the attempts to remove secretions were ineffective. Later, as the PaO_2/FiO_2 increased, so did the amount of removed secretion (21 of 25 ml in 12 hours). In case 3, the HFPV maneuver was well tolerated and able to adapt to the needs of a patient with a flare-up of COPD, in the failure phase, and with spontaneous breathing, without the need for sedation and paralysis.

In our preliminary experience, we wanted to use HFPV in those cases in which CMV failed to reach the planned target or when weaning from ventilatory support appeared problematic. Despite the attractive advantages the manufacturer described for the mechanisms by which gas exchange can be improved,[16] in practice none of them alone was sufficient; nonetheless, the effective removal of secretions, evident after the first hours of treatment, in itself represents an element of clinical importance in the application of this maneuver. Removal of secretions occurs in the interval between the delivery of 2 gas mini-bursts, *i.e.* when the lung volume tends to return to starting conditions after having reached peak pressure. In this way, the pressure exerted by an individual pulse works like a pneumatic hammer in removing secretions and reopening the obstructed airways. In 2 of the 3 cases described above, the PaO_2/FiO_2 obtained with CMV was <200; in all cases initial ventilatory support was set to the following parameters:

— PaO_2 >100 mmHg using $FiO_2 \leq 0.5$ and Vt 8-10 ml/kg;

— $PaCO_2$ between 30 mmHg and 40 mmHg;

—pH >7.35.

The failure to reach these values and, as explained above, the difficulty in weaning the patients from CMV were the reasons why we decided to use HFPV.

A good correlation exists between improvement in PaO_2/FiO_2 and the quantity of secretions removed through an endotracheal tube or through the oropharyngeal cavity. This shows that, because the expiratory phase is activated by oscillation, mucus transport can be achieved also above the cuff and therefore mucociliary clearance can be increased. Considering these elements, it can be reasonably stated that HFPV constitutes not only an alternative mode of ventilatory support but that the maneuver combines the therapeutic effect provided by nebulization of mucolytic agents and bronchodilators in the circuit with the maintenance of adequate humidification. In this way, the secretions are first liquefied and then easily removed. In our experience, capnographic and blood gas monitoring proved vital for the correct use of the device. In fact, we found a considerable capacity for removal of CO_2, most likely linked to augmented alveolar ventilation and to the creation of vortexes at the level of the airways. This feature could significantly influence pathologic conditions (*e.g.* acute respiratory distress syndrome, ARDS) in which hypercapnia represents the "price to pay" for maintenance of adequate blood oxygenation, while limiting the risk of pulmonary barotrauma.[17] The method was found to be devoid of major side effects; the risk of barotrauma, which has been reported for other modes of high-frequency ventilation, is, in fact, limited because the circuit is open, so that airway pressure never exceeds the set pressure. Furthermore, no significant hemodynamic alterations were observed.

This preliminary experience suggests that in patients with altered gas exchange also due to secretions treatment with alternating brief cycles of HFPV and CMV was shown to be effective. Further studies will be needed to determine the conditions for routine use of this mode of ventilatory support.

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