High frequency percussive ventilation: An asset to existing ventilation modi in intraoperative care?

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Abstract

High frequency percussive ventilation (HFPV) is a ventilation mode that combines positive pressure ventilation with some advantages of high frequency ventilation. During HFPV, a pulsatile flow is generated with high frequency and low volumes. HFPV has been used in the intensive care unit (ICU) for several decades, in case of insufficient conventional positive pressure ventilation. However, literature on its use in intraoperative care is scarce. We hypothesize that HFPV might be a better alternative to existing ventilation modi during selected operative procedures or in patients with severely compromised pulmonary and/or cardiac function. In this paper, we explain the HFPV system, we zoom in on the physiological effects of HFPV, and we describe its potential role in the intraoperative setting. Results of existing studies show that, compared to other conventional ventilation modes, HFPV improves oxygenation and ventilation without jeopardizing hemodynamics. However, because of the low quality evidence regarding physiological effects and clinical effectiveness, and due to the complicated design and set-up of the HFPV ventilator, the use of HFPV in intraoperative care is currently very limited. We conclude that HFPV could potentially be an interesting ventilation mode for procedures requiring minimal respiratory motion or low airway pressures, however larger (comparative) study trials are required to evaluate its usability in the operating room in patients with compromised pulmonary and/or cardiac function.

Keywords: **High Frequency Percussive Ventilation, Intraoperative ventilation, Respiration, artificial, VDR-4.**

Introduction and objective

Most procedures under general anesthesia require artificial ventilation to some degree. Depending on the depth of anesthesia and whether or not the patient has received neuromuscular blocking agents, breathing has to be supported or completely taken over by a ventilator. Conventional modes of artificial ventilation include volume controlled and pressure controlled ventilation, and the newer dual controlled ventilation[1]. Alternatively, high frequency ventilation (HFV) can be used in specific situations. These ventilation modes are characterized by high respiratory rate (>60 times per minute) and sub-dead space tidal volumes (Vt). Different types of HFV exist, including jet ventilation, oscillatory ventilation, flow interruption and high frequency percussive ventilation (HFPV). Theoretically, intraoperative HFPV might be a better alternative to conventional ventilation modes. However, indications, advantages and disadvantages have insufficiently been studied.

In this paper, we explain the HFPV system, we zoom in on the physiological effects of HFPV, and we describe potential applications in the intraoperative setting.

Methodology

We performed a review of the current literature on HFPV, through a search of Pubmed. Search strategy was [(VDR-4) OR HFPV] OR percussive ventilation. No study design limits were applied, but only publications using original data and written in English were withheld. No publication date or publication status restrictions were imposed.

Retrieved articles were screened based on title and abstract, by two independent reviewers (LR, MV). Withheld articles were read in full. Bibliographies of articles were screened for additional useful literature

High frequency percussive ventilation

High frequency percussive ventilation (HFPV) combines conventional positive pressure ventilation with some advantages of high frequency ventilation. HFPV is delivered through the VDR-4 ventilator. It combines a low pressure circuit with a high pressure circuit.

VDR-4 ventilator

The VDR-4 has a complex looking appearance, that requires extensive study of the manual to set up (Fig. 1). The core unit however, that gives the VDR-4 its unique properties, is the phasitron (Fig. 2). The air from the high and low pressure circuits comes together in the 'phasitron', which is positioned just before the patient connection piece. It is a hollow cylinder in which the pulsatile airflow from a high pressure circuit causes a spring-controlled piston to move back and forth, opening or closing the entrainment port or the exhalation port. At one end of the phasitron, high pressure pulsatile flow is pushed in the narrow cylinder, where pressure drop and flow increase cause entrainment of ambient air through the entrainment port, based on the Venturi effect. In this way, air from the low pressure circuit is mixed with air from the high pressure circuit[2-4].

The volume from the phasitron is pushed into progressively smaller airways, increasing its speed and decreasing its pressure. High pressure micro Vt with low flow are converted into low pressure with high flow volumes. Any increase in airway pressure causes a decrease in the amount of ambient air drawn into the phasitron. In this way, the system automatically adapts to variation of lung resistance. There is a continuous communication with the external environment, thereby preventing hyperinflation and baro- or volutrauma. Pressures are continuously monitored through the monitoring sampling port at the patient connection piece[2-4].

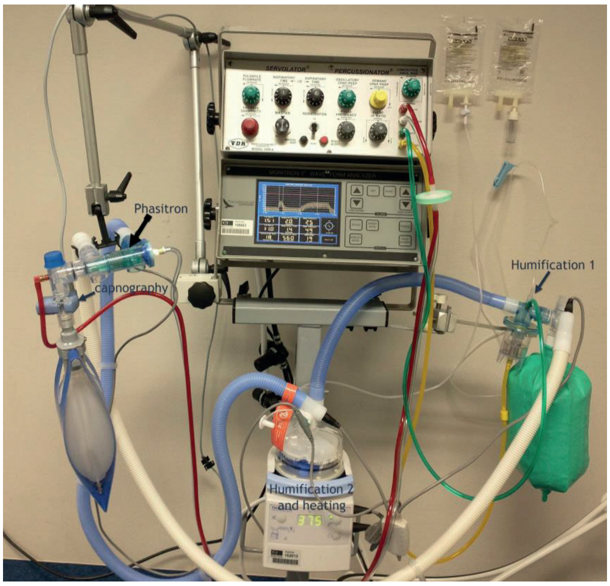

Fig. 1 — VDR-4 ventilator setup.

Pressure curve of HFPV

FPV combines high frequency ventilation with time-cycled pressure-limited controlled mechanical ventilation. Two different pressure levels are defined, around which ventilation oscillates. During HFPV, a high pressure pulsatile flow is generated with 'high frequency' and low volumes. The small high frequency pulses of gas accumulate to form a 'low frequency' Vt. The anatomy of the pressure wave is shown in figure 3. During the inspiratory phase, the lung is inflated in a pulsatile fashion to a peak inspiratory pressure (PIP), much like in intermittent positive pressure ventilation (IPPV). The time of the inspiratory phase and the time of the expiratory phase are preset, like in IPPV, and are described as the I/E ratio. Once the inspiratory time cycles off, the lung is allowed to passively deflate to a preset CPAP or PEEP. In addition to these 'low frequency' settings, 'high frequency' rate and i/e ratio of the gas pulses can be adjusted[3].

Adjusting ventilation

The control panel of the VDR-4 (Fig. 4) allows to set several variables of ventilation: pulsatile flowrate, inspiratory and expiratory time, demand PEEP/CPAP, oscillatory PEEP/CPAP, pulse frequency and i/e ratio, and FiO_2[5].

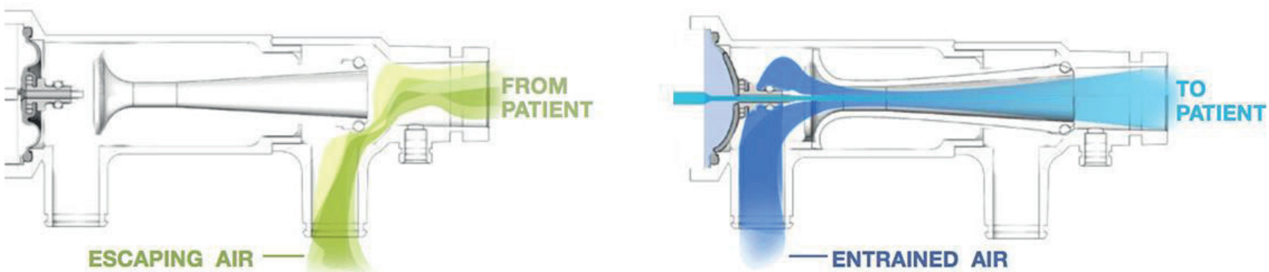

Fig. 2 — Phasitron detail and air flow[5].

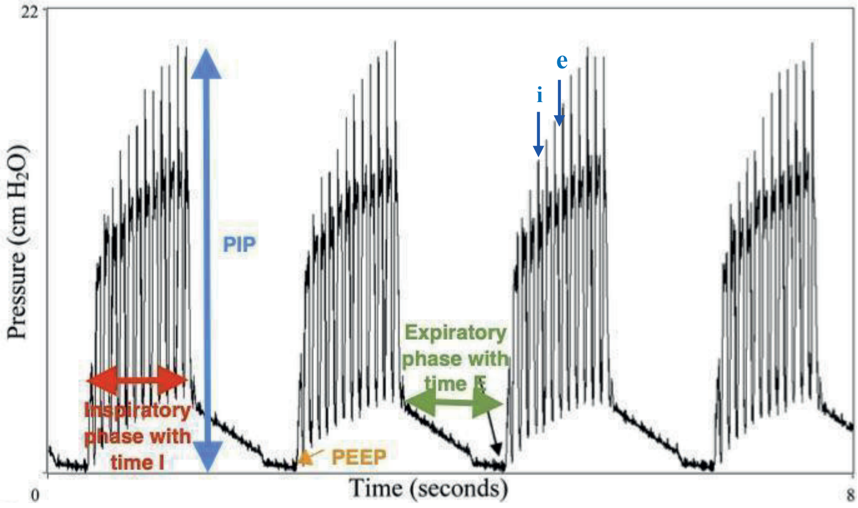

Fig. 3 — Pressure curve of HFPV[4]. E: expiratory, I: inspiratory, PEEP: positive end-expiratory pressure, PIP: peak inspiratory pressure.

Proposed guidelines for blood gas manipulation can be found in figure 5[5]. To increase oxygenation, several actions can be taken. The order in which these actions are taken, depends on the operator. Increasing PIP by increasing pulsatile flowrate or increasing inspiratory time will increase Vt. Also, just as in IPPV, increasing FiO_2 and both oscillatory and demand PEEP will increase oxygenation. Finally, a convective pressure rise can be given for better recruitment of the lung[2,4,6].

To decrease $PaCO_2$, ventilation should be increased. Ventilation is mainly increased by augmenting PIP. Oscillatory PEEP and demand PEEP can be lowered if oxygenation is good, thus increasing the driving pressure (the difference between airway pressure during inspiration and expiration; PIP-PEEP) for convective ventilation. If this is insufficient, percussive rate and both i:e and I:E ratio should be lowered. In this way, more expiratory time comes available and CO_2 elimination is increased[2,6].

Fig. 4 — VDR-4 ventilator with monitor[5].

Physiological changes during HFPV

Since the development of the VDR-4 ventilation system by Bird et al. in the eighties, several observational studies and some smaller sample size clinical trials have been published.

Before discussing these studies, a major issue should be acknowledged: after publication of the ARDSNet study, in the year 2000, low Vt protective lung ventilation (6 ml/kg) has been implemented. Before 2000, general practice of IPPV included the use of large Vt of 10-20 ml/kg[7]. This practice caused greater stretching of lung tissue, thought to be one of the causes of lung injury. Hence, the relevance of these older results is disputable.

Although high quality evidence is scarce, accumulating results of existing studies suggest benefits of HFPV on several organ systems and important advantages over conventional mechanical ventilation. We will discuss the effect of HFPV on the pulmonary system, hemodynamics, the brain, and on hard end-points.

1. Effects of HFPV on the pulmonary system

Gas exchange

In burn patients and patients with ARDS, HFPV has shown to improve gas exchange at lower peak and mean airway pressures. However, as mentioned before, some of the earlier studies date from before the implementation of lung protective low Vt ventilation[8-15]. Eight studies on gas exchange were found that used lung protective low Vt ventilation in the IPPV-arm.

In a randomized controlled trial in 64 burned children, a significant higher PaO_2/FiO_2 (P/F) ratio at lower airway pressures was observed with HFPV compared to IPPV (6-8 ml/kg)[16]. Several years later, similar results were found in an observational study

Decrease CO_2 only	Increase Oxygenation with $PaCO_2$ in Range	Increase PaO_2 and Lower $PaCO_2$
a. ↑Pulsatile flow by 2 cmH_2O up to maximum AIP 40-46 cmH_2O b. ↓Pulse Frequency by 50-100 bpm to a minimum of 400 c. Lengthen I time to 3.0 seconds and shorten E time to 1 second. d. Turn on convective pressure rise. Gradient between convective pressure rise and pulsatile flow = (3-10). If all of the above have not achieved desired CO_2 level, creating a mild to moderate cuff leak per auscultation can be used depending on your infection control/ VAP guidelines.	a. ↑FIO_2 if at low levels b. ↑Oscillatory CPAP/PEEP by 2 cmH_2O max (16-20) c. If maximum Oscillatory CPAP/PEEP is reached: • ↑Pulse Frequency by 50-100 ppm to a maximum of 700 • May cause some increase in CO_2 d. ↑Time at P High • ↑IT by 0.5-1.0 seconds up to 3.5 seconds max e. Turn on convective pressure rise. Gradient between convective pressure rise and pulsatile flow = (3-10) ⇨ **BE PATIENT**-it can take up to 2-4 hours for recruitment to take place.	a. ↑Pulsatile flow by 2 cmH_2O up to maximum AIP 40-46 cmH_2O b. ↑Increase Oscillatory CPAP/PEEP by 2, but keep the gap between pulsatile flow and Oscillatory CPAP/ PEEP the same with adjustment. If the gap is decreased, then CO_2 removal may not be as effective.

Fig. 5 — Proposed guidelines for blood gas manipulation[5].

of 31 pediatric patients with acute respiratory failure, failing conventional ventilation. The improved oxygenation continued throughout 48 hours after transition back to IPPV. Also, a reduction in pCO_2 occurred 6 hours after initiation of HFPV[17].

In the adult population, results on gas exchange during HFPV are not so straightforward. Most studies in various populations (burn patients with and without inhalation injury, acute lung injury (ALI) and ARDS patients with multiple etiologies, obese patients with respiratory failure and postoperative cardiac surgery patients) show a significant rise in P/F ratio[18-23], but results on sustainability of this improved P/F ratio are conflicting. Also, effect of HFPV on pCO_2 was unaltered in some and improved in other studies[17,21] and there was no difference in oxygenation index between the two groups[18]. Another study found that oxygenation improved more in non-septic ARDS and that ventilator dependency and mortality at 30 days were higher in pneumonia related ARDS[19].

A retrospective analysis on HFPV as rescue therapy in patients failing IPPV, found that it was a successful strategy to preclude patients from ECMO in morbidly obese patients with respiratory failure and in postoperative cardiac surgery patient[22,23]. Comparative studies between different HFV modes were not found, except for one. A retrospective study compared HFPV (n=27) with high frequency oscillatory ventilation (HFOV) in a population of 16 pediatric patients with acute respiratory failure[24]. HFPV patients showed increased P/F ratios and decreased $PaCO_2$ levels 6 hours after initiation of HFPV, whereas HFOV patients showed no significant differences. Mortality was 15% in the HFPV group and 50% in the HFOV group[24].

Lung compliance

A study that compared lung computed tomography (CT) in 8 patients before initiation of HFPV and after one hour of HFPV, showed alveolar recruitment without relevant hyperinflation and better lung compliance[20]. Also a case control study that used low Vt in the IPPV-arm (n=35), demonstrated an improvement in lung compliance after initiation of HFPV[21].

Pulmonary barotrauma

Early comparative studies between HFPV and IPPV found a significant decrease in incidence of barotrauma in several populations[10,25]. However, these studies did not use low Vt lung protective ventilation.

A previously mentioned randomized controlled trial in burned children (n=64), who did use lung protective strategy in the IPPV arm, reported that no patient in the VDR group had evidence of barotrauma compared with two in the IPPV group[16]. Also a randomized controlled trial in adult burn patients (n=62) found significantly less barotrauma in the HFPV group compared to the IPPV group[18].

Evacuation of mucus

Improved clearance of mucus is a generally accepted property of HFPV. The high frequency causes a vibration of mucosa and secretions, and the variation of percussive frequency causes turbulence in the airway, which is, together with the elevated flow, thought to improve mucus clearance[3]. However, objective data on this topic are lacking. Some authors report a subjective improvement of secretion clearance[6,24], whereas others found no difference between the overall volume of sputum[26].

Incidence of ventilator associated pneumonia (VAP)

In contrast to an early historical case-control study in inhalation injury patients (n= 54)[10], no difference in infectious complications was observed in a randomized controlled trial of patients with inhalation injury (n=35)[8]. However, both studies did not use low Vt protective lung ventilation in the IPPV arm.

A randomized controlled trial in 62 burn patients, that did use low Vt lung protective ventilation, could not find a significant difference, however there was a trend towards less incidence of VAP in the HFPV group[18].

2. Effect of HFPV on hemodynamics

Several smaller studies in different pathologies (ARDS, obesity and burns) showed no effect on hemodynamic parameters during HFPV[12,13,15, 21,27].

In 24 cardiac surgery patients (mean left ventricular ejection fraction 49%), HFPV was initiated at arrival on the ICU, followed by transitioning to conventional mechanical ventilation. No difference in mean arterial pressure, cardiac index and mixed venous PaO_2 was found[28].

Another study in a population of 8 patients with early non-focal ARDS, showed an increase in mean arterial pressure and reduced doses of norepinephrine during HFPV. These benefits disappeared after resuming IPPV. However, it has to be acknowledged that except for one patient, who was admitted after cardiac arrest, none of the study patients had cardiovascular comorbidity[20].

3. Effect of HFPV on the brain

Two studies were found on the effect of HFPV on intracranial pressure in patients with traumatic brain injury: one prospective controlled trial (n=38) and one retrospective chart review (n=10)[29,30]. Both studies came to the same conclusion that there was a significant decrease in intracranial pressure after initiation of HFPV. However, both studies did not use low Vt lung protective ventilation, and therefore the results of these studies are less relevant today.

4. Effect of HFPV on hard end-points

Some older retrospective studies in burn patients, that did not include lung protective low Vt ventilation, found an improvement in survival rate and decrease of incidence of pneumonia[10,31].

A cohort study in inhalation injury patients retrospectively compared patients who were treated with HFPV (n=95) with patients who were treated with IPPV (n=130)[14]. The authors found a significant decrease in both overall morbidity and mortality in a subset of patients with 40% or less burned total body surface area treated with HFPV. Of note in this study is the very heterogeneous Vt, since patients who were included before 2000 were ventilated with 10 ml/kg while patients who were included after 2000 received low Vt lung protective ventilation in accordance with the findings of the ARDSnet study[14].

A study of biomarkers of lung injury (interleukin 6 & 8, and tumor necrosis factor alpha (TNF-α)), that have shown to be predictors of morbidity and mortality, found that these biomarkers did not increase after initiation of HFPV[32].

However, a more recent randomized controlled trial in 62 adult burn patients, that did use low Vt lung protective ventilation in the IPPV arm, did not find a significant difference in ventilator free days nor in mortality[18].

Applications in the intraoperative setting

Growing interest in minimizing invasiveness of surgical procedures has increased the need for alternative ventilation strategies in the operating room. Because of the minimal respiratory motion in HFV, its use has been explored in cardiac ablations, urology, solid organ tumor ablations and radiology[33]. HFPV could potentially have advantages over the other HFV modes in intraoperative care.

Only four papers discussed the intraoperative use of HFPV: one randomized controlled trial[26], one retrospective case-control study[34] and two case reports[35,36].

The first record of the use of HFPV in the operating room was a case report on a bronchial repair in a patient with one lung, in 2006[35]. HFPV was initiated after opening of the chest wall, in order to minimize air leakage. To overcome emerging hypoxia and hypercarbia with acidosis, driving pressure of the convective ventilation was increased and percussion was added to the expiratory phase. Following these interventions, FiO_2 could be reduced from 100 to 50%, pCO_2 normalized, and air leakage was resolved before the bronchus repair was completed. Hemodynamics remained stable throughout the procedure. Patient recovered in the ICU under conventional ventilation and was extubated 24 hours later. The authors concluded that the CO_2 washout depended mainly on the pressure gap created by the bilevel ventilation mode. They highlighted the ability to improve gas exchange as a result of both convective and diffusive mechanism, and this with safe peak and mean airway pressures. Furthermore, the surgeon was permitted to work in an almost immobile field.

Three years later, the same authors published the first and so far the sole randomized controlled study. Forty-four patients undergoing an elective partial pulmonary resection by thoracotomy in the lateral decubitus position were included[26]. The dependent lung was ventilated with IPPV. Patients received either HFPV (n=22) or CPAP (n=22) on the nondependent lung, initiated after 20 minutes of one lung ventilation or if SpO_2 dropped below 90% at any time. The authors found that the HFPV group had higher pO_2 just before re-expansion than those in the CPAP group. $PaCO_2$, heart rate and mean arterial blood pressure did not differ between the groups nor during the procedure. Postoperative sputum clearance was more efficient in the HFPV group (72% of total volume of sputum at day 4, compared to 46% in the CPAP group). More patients in the CPAP group had postoperative fever and two patients were diagnosed with postoperative pneumonia compared to none in the HFPV group. Hospital discharge was significantly earlier for patients in the HFPV group (68% at day 5, compared to 27% in the CPAP group).

In 2016, Inoue and colleagues retrospectively studied 24 neonates operated for congenital diaphragmatic hernia[34]. The focus of their paper was mainly on the surgical technique and HFPV was merely mentioned as an aid strategy, introduced after two cases of severe respiratory acidosis during thoracoscopic repair. In this case, an alternative for the VDR-4, a combination of intrapulmonary percussive ventilation and a respirator, was used. The authors observed no severe hypercapnia after implementation and concluded that HFPV is a countermeasure against intraoperative respiratory acidosis and allows acceptable and safe conditions for surgery[34].

The most recent paper is a case report which describes the use of HFPV during whole lung lavage for pulmonary alveolar proteinosis in a 47-year old woman with oxygen dependent COPD[36]. The non-operated lung was ventilated with a conventional ventilator, while the operated lung received HFPV with the VDR-4. The authors highlighted that the amount of lavage fluid needed was far less than the average amount needed during whole lung lavage, which they attributed to the improved clearance of pulmonary secretions due to HFPV. They stated that some known difficulties causing hypoxemia during whole lung lavage were avoided, such as fluid spillage in the ventilated lung, double lumen tube displacement during repositioning, and ventilation/perfusion mismatch.

Interestingly, HFPV has also been used in non-intubated, non-sedated patients. Intrapulmonary percussive ventilation (IPV) is derived from HFPV and has been used in patients with excessive respiratory secretions and atelectasis, for example COPD and cystic fibrosis. It uses a face mask or mouthpiece as an interface, instead of an endotracheal tube. Similar to HFPV, there is a lack of evidence regarding physiological effects and clinical effectiveness[37]. Recently, IPV has been explored in small prospective observational studies as a strategy to facilitate breath holding in awake patients, aiming to minimize respiratory movement during radiotherapy, MRI scanning and PET-CT scanning[38-41]. In radiotherapy (n=4), a reduction of the mean heart dose was observed as compared to free breathing, and slight improvement of the dosimetric surrogates for lung toxicity were observed, potentially reducing collateral damage[40]. In the PET-CT (n=4) and MRI setting (n=2), the authors found that it was feasible to induce apnea for several minutes (5-10 min), allowing improved image acquisitions[38,39].

Discussion

Although current literature suggests that HFPV might have advantages over existing ventilation modi, its use in intraoperative care is at present still very limited.

Studies in the ICU suggest that, as compared to IPPV, HFPV improves oxygenation and ventilation, without augmenting pulmonary pressures. Also, unchanged or improved hemodynamics were seen in patients ventilated in the ICU with HFPV compared to IPPV.

HFPV might also overcome some of the drawbacks of the other HFV modi. In most types of HFV, the expiration is passive. Air can only leave the lung by ‘escaping’ between the jet cannula, surgical instruments, mucus, and airway debris. Build-up of auto-PEEP is a feared complication that can lead to hyperinflation and subsequent barotrauma, compromised venous return and increased intracranial pressure. Therefore, chronic obstructive pulmonary disease, severe cardiovascular compromise and elevated intracranial pressure are relative contraindications for HFV. Recent jet ventilators partially meet these concerns by featuring the possibility of continuous pressure measurement through double lumen jet cannula's, and automatically abort ventilation when preset pressures are reached[33].

Because of the unique properties of the phasitron in the VDR-4 ventilator, any increase in airway pressure causes a decrease in the amount of ambient air drawn into the phasitron. In this way, the system automatically adapts to variation of lung resistance. The advantage is that heterogeneous areas of the lung are ventilated thanks to accumulating mini-burst of airflow that are 'tailored' to the mechanical characteristics of the thoracopulmonary system[3]. Because of the continuous controlled communication with the external environment through three safety valves, hyperinflation and barotrauma are prevented[42]. On the other hand, small changes in patient lung characteristics can lead to major changes in ventilation. This could be of particular importance in the intraoperative setting, e.g. during surgical manipulation. Furthermore, minimal intended or unintended adjustments of controls can lead to major changes in ventilation.

Another advantage of HFPV over other types of HFV in the intraoperative setting is the possibility of monitoring end-tidal CO_2. Due to high flow, small Vt and the open circuit, capnography in HFV is not reliable[33]. Although this can be overcome by transcutaneous measurement or intermittent blood gas analysis, these alternatives have a delay in reading.

Potential applications of HFPV in the operating room include procedures requiring minimal respiratory motion (eg ablations) or low airway pressures to minimize air leakage such as during bronchial repair. Currently, one lung ventilation is being used in these settings. Increasing FiO_2 up to 1.0 and initiating CPAP on the non-dependent lung are used as a salvage strategy in case of desaturation. Lucangelo et al. proposed HFPV as an alternative escape strategy for CPAP when desaturation occurs during one lung ventilation. They demonstrated that HFPV of the non-dependent lung could prevent desaturation and hypercapnia and diminished the need for early escape return to two lung ventilation. Also, the more efficient evacuation of mucus might be related to the lower chance of postoperative fever and postoperative pneumonia, and to earlier hospital discharge in the HFPV group[26].

Despite many theoretical potential advantages, the physician has to face major drawbacks when she/he considers to use the VDR-4. The set-up of the VDR-4 is complicated and takes some time, yet correct assembly of the components is paramount for optimal functioning[6]. Setting up the VDR-4 requires an extensive study of the manual, and thorough training of anesthesiologists, nursing staff, as well as technicians is needed. The ventilator can be set up by adjusting buttons and need to be reset again once the patient is connected to the ventilator. Also, the VDR-4 ventilator does not have a user friendly design: control buttons are mechanical (not digital) and very sensitive to (accidental) touch, and important controls such as FiO_2 are not logically displayed. The settings are far less intuitive than in IPPV and require more frequent adjustments in response to lung characteristics and observed parameters. Additionally, its use comes with a higher financial burden than conventional ventilation. All these disadvantages might account for the limited application of the HFPV in the intraoperative setting.

Conclusion

We conclude that HFPV might be a better alternative to existing ventilation modi during selected operative procedures. However, due to lack of evidence regarding physiological effects and clinical effectiveness, larger (comparative) study trials are required to evaluate its usability in selected patients with compromised pulmonary and/or cardiac function.

Conflict of interest: None.

References

1. Ball, L., Dameri, M. and Pelosi, P. 2015. Modes of mechanical ventilation for the operating room. Best Pract. Res. Clin. Anaesthesiol., 29, 285-299.
2. Salim, A. and Martin, M. 2005. High-frequency percussive ventilation. Crit. Care Med., 33, S241-245.
3. Lucangelo, U., Fontanesi, L., Antonaglia, V., Pellis, T., Berlot, G., Liguori, G., et al. 2003. High frequency percussive ventilation (HFPV). Principles and technique. Minerva Anestesiol., 69, 841-851.
4. Allan, P. F., Osborn, E. C., Chung, K. K. and Wanek, S. M. 2010. High-frequency percussive ventilation revisited. J. Burn Care Res., 31, 510–520.
5. VDR-4 Operator's Manual. VDR-4 Operator's Manual; Percussionaire Corporation, 2020.
6. Kunugiyama, S. K. and Schulman, C. S. 2012. High-frequency percussive ventilation using the VDR-4 ventilator: an effective strategy for patients with refractory hypoxemia. AACN Adv. Crit. Care, 23, 370–380.
7. Brower, R. G., Matthay, M. A., Morris, A., Schoenfeld, D., Thompson, B. T. and Wheeler, A. 2000. Ventilation with Lower Tidal Volumes as Compared with Traditional Tidal Volumes for Acute Lung Injury and the Acute Respiratory Distress Syndrome. N. Engl. J. Med., 342, 1301–1308.
8. Reper, P., Wibaux, O., Van Laeke, P., Vandeenen, D., Duinslaeger, L. and Vanderkelen, A. 2002. High frequency percussive ventilation and conventional ventilation after smoke inhalation: a randomised study. Burns, 28, 503-508.
9. Cioffi, W. G., Graves, T. A., McManus, W. F. and Pruitt, B. A., Jr. 1989. High-frequency percussive ventilation in patients with inhalation injury. J. Trauma, 29, 350-354.
10. Cioffi, W. G., Jr, Rue, L. W., 3rd, Graves, T. A., McManus, W. F., Mason, A. D., Jr and Pruitt, B. A., Jr. 1991. Prophylactic use of high-frequency percussive ventilation

in patients with inhalation injury. Ann. Surg., 213, 575-580; discussion 580-582.
11. Cortiella, J., Mlcak, R. and Herndon, D. 1999. High frequency percussive ventilation in pediatric patients with inhalation injury. J. Burn Care Rehabil., 20, 232-235.
12. Velmahos, G. C., Chan, L. S., Tatevossian, R., Cornwell, E. E., 3rd, Dougherty, W. R., Escudero, J., et al. 1999. High-frequency percussive ventilation improves oxygenation in patients with ARDS. Chest, 116, 440-446.
13. Paulsen, S. M., Killyon, G. W. and Barillo, D. J. 2002. High-frequency percussive ventilation as a salvage modality in adult respiratory distress syndrome: a preliminary study. Am. Surg., 68, 852-856; discussion 856.
14. Hall, J. J., Hunt, J. L., Arnoldo, B. D. and Purdue, G. F. 2007. Use of high-frequency percussive ventilation in inhalation injuries. J. Burn Care Res., 28, 396-400.
15. Reper, P., Van Bos, R., Van Loey, K., Van Laeke, P. and Vanderkelen, A. 2003. High frequency percussive ventilation in burn patients: hemodynamics and gas exchange. Burns, 29, 603-608.
16. Carman, B., Cahill, T., Warden, G. and McCall, J. 2002. A prospective, randomized comparison of the Volume Diffusive Respirator vs conventional ventilation for ventilation of burned children. 2001 ABA paper. J. Burn Care Rehabil., 23, 444-448.
17. Rizkalla, N. A., Dominick, C. L., Fitzgerald, J. C., Thomas, N. J. and Yehya, N. 2014. High-frequency percussive ventilation improves oxygenation and ventilation in pediatric patients with acute respiratory failure. J. Crit. Care, 29, 314.e1–7.
18. Chung, K. K., Wolf, S. E., Renz, E. M., Allan, P. F., Aden, J. K., Merrill, G. A., et al. 2010. High-frequency percussive ventilation and low tidal volume ventilation in burns: a randomized controlled trial. Crit. Care Med., 38, 1970-1977.
19. Spapen, H., Borremans, M., Diltoer, M., Van Gorp, V., Nguyen, D. N. and Honoré, P. M. 2014. High-frequency percussive ventilation in severe acute respiratory distress syndrome: A single center experience. J. Anaesthesiol. Clin. Pharmacol., 30, 65-70.
20. Godet, T., Jabaudon, M., Blondonnet, R., Tremblay, A., Audard, J., Rieu, B., et al. 2018. High frequency percussive ventilation increases alveolar recruitment in early acute respiratory distress syndrome: an experimental, physiological and CT scan study. Crit. Care, 22, 3.
21. Lucangelo, U., Zin, W. A., Fontanesi, L., Antonaglia, V., Peratoner, A., Ferluga, M., et al. 2012. Early short-term application of high-frequency percussive ventilation improves gas exchange in hypoxemic patients. Respiration, 84, 369–376.
22. Korzhuk, A., Afzal, A., Wong, I., Khusid, F., Worku, B. and Gulkarov, I. 2020. High-Frequency Percussive Ventilation Rescue Therapy in Morbidly Obese Patients Failing Conventional Mechanical Ventilation. J. Intensive Care Med., 35, 583-587.
23. Wong, I., Worku, B., Weingarten, J. A., Ivanov, A., Khusid, F., Afzal, A., et al. 2017. High-frequency percussive ventilation in cardiac surgery patients failing mechanical conventional ventilation. Interact. Cardiovasc. Thorac. Surg., 25, 937-941.
24. Tawfik, D. S., Bennett, T. D., Welch, B. and Poss, W. B. 2016. Use of High-Frequency Ventilation in the Pediatric Intensive Care Unit. Pediatr. Crit. Care Med., 5, 12-20.
25. Rodeberg, D. A., Maschinot, N. E., Housinger, T. A. and Warden, G. D. 1992. Decreased pulmonary barotrauma with the use of volumetric diffusive respiration in pediatric patients with burns: the 1992 Moyer Award. J. Burn Care Rehabil., 13, 506-511.
26. Lucangelo, U., Antonaglia, V., Zin, W. A., Confalonieri, M., Borelli, M., Columban, M., et al. 2009. High-frequency percussive ventilation improves perioperatively clinical evolution in pulmonary resection. Crit. Care Med., 37, 1663-1669.
27. Tsuruta, R., Kasaoka, S., Okabayashi, K. and Maekawa, T. 2006. Efficacy and safety of intrapulmonary percussive ventilation superimposed on conventional ventilation in obese patients with compression atelectasis. J. Crit. Care, 21, 328-332.
28. Oribabor, C., Gulkarov, I., Khusid, F., Ms, E. F., Esan, A., Rizzuto, N., et al. 2018. The use of high-frequency percussive ventilation after cardiac surgery significantly improves gas exchange without impairment of hemodynamics. Can J Respir Ther, 54, 58-61.
29. Hurst, J. M., Branson, R. D. and Davis, K., Jr. 1988. High-frequency percussive ventilation in the management of elevated intracranial pressure. J. Trauma, 28, 1363-1367.
30. Salim, A., Miller, K., Dangleben, D., Cipolle, M. and Pasquale, M. 2004. High-frequency percussive ventilation: an alternative mode of ventilation for head-injured patients with adult respiratory distress syndrome. J. Trauma, 57, 542-546.
31. Rue, L. W., 3rd, Cioffi, W. G., Mason, A. D., McManus, W. F. and Pruitt, B. A., Jr. 1993. Improved survival of burned patients with inhalation injury. Arch. Surg., 128, 772-778; discussion 778-780.
32. Reper, P. and Heijmans, W. 2015. High-frequency percussive ventilation and initial biomarker levels of lung injury in patients with minor burns after smoke inhalation injury. Burns, 41, 65-70.
33. Galmén, K., Harbut, P., Freedman, J. and Jakobsson, J. G. 2017. The use of high-frequency ventilation during general anaesthesia: an update. F1000Res., 6, 756.
34. Inoue, M., Uchida, K., Otake, K., Nagano, Y., Mori, K., Hashimoto, K., et al. 2016. Thoracoscopic repair of congenital diaphragmatic hernia with countermeasures against reported complications for safe outcomes comparable to laparotomy. Surg. Endosc., 30, 1014-1019.
35. Lucangelo, U., Zin, W. A., Antonaglia, V., Gramaticopolo, S., Maffessanti, M., Liguori, G., et al. 2006. High-frequency percussive ventilation during surgical bronchial repair in a patient with one lung. Br. J. Anaesth., 96, 533-536.
36. Kinthala, S., Liang, M., Khusid, F. and Harrison, S. 2018. The Use of High-Frequency Percussive Ventilation for Whole-Lung Lavage: A Case Report. A A Pract, 11, 205-207.
37. Reychler, G., Debier, E., Contal, O. and Audag, N. 2018. Intrapulmonary Percussive Ventilation as an Airway Clearance Technique in Subjects With Chronic Obstructive Airway Diseases. Respir. Care, 63, 620-631.
38. Prior, J. O., Péguret, N., Pomoni, A., Pappon, M., Zeverino, M., Belmondo, B., et al. 2016. Reduction of Respiratory Motion During PET/CT by Pulsatile-Flow Ventilation: A First Clinical Evaluation. J. Nucl. Med., 57, 416-419.
39. Beigelman-Aubry, C., Peguret, N., Stuber, M., Delacoste, J., Belmondo, B., Lovis, A., et al. 2017. Chest-MRI under pulsatile flow ventilation: A new promising technique. PLoS One, 12, e0178807.
40. Péguret, N., Ozsahin, M., Zeverino, M., Belmondo, B., Durham, A.-D., Lovis, A., et al. 2016. Apnea-like suppression of respiratory motion: First evaluation in radiotherapy. Radiother. Oncol., 118, 220-226.
41. Audag, N., Van Ooteghem, G., Liistro, G., Salini, A., Geets, X. and Reychler, G. 2019. Intrapulmonary percussive ventilation leading to 20-minutes breath-hold potentially useful for radiation treatments. Radiother. Oncol., 141, 292-295.
42. Lucangelo, U., Fontanesi, L. and Bird, F. 2002. High Frequency Percussive Ventilation. High Frequency Percussive Ventilation. Anaesthesia, Pain, Intensive Care and Emergency Medicine — A.P.I.C.E. ,163-171.

doi.org/10.56126/73.1.03