HIGH FREQUENCY VENTILATION AND REGIONAL COMPLIANCE

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Persistent clinical demand and recent industrial developments show that High Frequency Ventilation is well established, although the method was introduced prematurely into clinical use, with incomplete knowledge, primitive instrumental development and inadequate monitoring standards. The confusion [1–6] in the literature regarding the clinical use of high frequency ventilation and its merits indicates that there is a lack of understanding of the method.

The user must understand that high frequency ventilation does not follow the usual rules of ventilation. In addition, new high frequency ventilators and monitoring systems are required.

Basic mechanisms

In order to understand the nature of high frequency ventilation, it is necessary to consider two different mechanisms of gas exchange:

Periodic ventilation. With spontaneous breathing, alveolar gas exchange occurs in aliquots. The alveolus is never completely devoid of carbon dioxide. The forces which cause periodic washin and washout of carbon dioxide arise from periodic changes in the dimensions of the alveoli (and the airways).

The upper part of the airways functions solely as a conductor, and under physiological conditions deadspace volume does not limit ventilatory function. Variations in deadspace are well documented and, during conventional mechanical ventilation, it is easy to adjust tidal volume and frequency to match the patient’s age, body weight and temperature. However, during high frequency ventilation, the concept of deadspace—taken as a space, a gas volume or a diagnostic or therapeutic model compartment—creates particular problems [7] and satisfactory control of ventilation can only be achieved by repeated blood-gas measurement.

“Diffusion respiration” or “apnoeic gas exchange”. This was an early attempt to replace periodic ventilation by continuous washout of carbon dioxide [8]. Oxygen was insufflated continuously, and was taken up by the erythrocytes in the lung capillaries, ensuring oxygenation of blood. However, washout of carbon dioxide was inadequate. With the resulting continuous increase in alveolar concentration, carbon dioxide transfer was diminished and, within 1 h, arterial $P_{CO_2}$ increased to lethal values.

Experimental and clinical investigations into the “continuous flow method” confirmed that at low fresh gas flow rates, diffusion of carbon dioxide from alveoli into airways does not remove sufficient carbon dioxide; direct flushing of alveoli does not appear to contribute to alveolar ventilation (with the exception of some very short respiratory units); oxygen uptake is guaranteed, provided the lung has been washed out with oxygen and the alveolar space is adequately inflated. So we have to conclude that a small periodic ventilatory movement of the alveolus remains an indispensable precondition for expulsion of carbon dioxide from the alveoli. Carbon dioxide may be flushed effectively from the bronchioles with adequate volume flow.

Historically, confusion began here: Some investigators reported successful “diffusion respiration” when the insufflated gas flow was large. According to Meltzer and Auer [9], this was accompanied by a roaring noise behind the sternum. However, “alveolar ventilation” under “continuous high flow ventilation” may be a
mechanism which differs in principle from that of "continuous low flow ventilation" (if this exists):

If gas flow is increased to more than 200 times the minute volume of the actual oxygen demand, the pulmonary parenchyma may be made to oscillate. Continuous insufflation may act on bronchial bifurcations in a manner similar to that of air blown into organ pipes (fig. 1) [10]. Removal of carbon dioxide then depends on the extent of the oscillatory agitation and the extent of the airway system which is included in this type of excitation. This is conceptually similar to high frequency oscillation.

With periodic insufflation at frequencies exceeding 7 Hz, a frequency-dependent excitation of individual gas conduits and the attached parenchyma appears to occur [11, 12]. With each change in the excitation frequency, the distribution of the oscillating bronchi changes, and it has been suggested that all the regions of the lung could be ventilated by systematically varying the exciting frequency [13].

Unfortunately, the term "high frequency ventilation" has been applied to all types of ventilation at frequencies greater than 80 b.p.m. However, the adult airways system is able to follow periodic pressure and volume changes up to 3–5 (even 7) Hz, and the natural frequency of the airway system has been shown to be in the range of 3–7 Hz [14]. At frequencies less than 7 Hz and in conventional respiration, regional alveolar ventilation has to be assumed to depend solely on segmental compliance and airway resistance. Only at higher exciting frequencies, beyond 10 Hz, does ventilation become independent of compliance [15–19].

The use of HFV at frequencies less than 7 Hz has not been shown to confer any marked advantages in clinical practice, although jet ventilation has proved useful in ENT surgery and in maintenance of ventilation by cricothyroid puncture during difficult tracheal intubation [4]. The reduced tidal volume was compensated for by an increase in ventilatory frequency at 2–5 Hz, but minute volume had to be increased to compensate for the increased proportion of deadspace ventilation. Klain [4] and Rouby [5] recommend a reduction in the exciting frequency if elimination of carbon dioxide is inadequate.

Another advantage of the jet is that it tends to produce preferential ventilation of those bronchial compartments which are in line with the axis of the tube, with later distribution determined by compliance and airways resistance.

The high jet flow is able to inflate the lungs with shorter inspiratory time than that needed for conventional ventilation. However, expiration is passive and is thus limited by the elastic properties of the airways, lungs and the chest wall and by the
“High Frequency Ventilation” one has to reconsider much of the literature in this field [4–6]. However, to make research consistent and to prevent further confusion, results from “Rapid” (up to 7 Hz) and true “High Frequency Ventilation” should not be compared as if the basic mechanisms were similar.

For any mode of true “High Frequency Ventilation” (that is, frequencies greater than 10 Hz), direct ventilation of very proximal alveoli is possible. In babies undergoing high frequency oscillations, direct ventilation of alveoli may occur throughout the major portion of the lungs. However, in adults the question has to be answered: How does an alternating pressure induce spatial oscillations of the airway system—that is, frequency-dependent, segment-specific mobilization? There are three possible mechanisms:

1. Periodic changes in the length and width of the airways probably occur in response to pressure alterations (fig. 2). [20]. The elastic resistance to stretch and the inertance of the bronchi and of the lung tissue determine the natural frequency of the oscillatory movements of each bronchial and parenchymal segment.

2. Curved or angular bronchi execute whiplash-like movements (fig. 3). As in the “garden hose phenomenon” in coronary vessels [21–24], the “erectile forces” become greater as the radius of curvature of a bronchial bend becomes smaller or the angle of bifurcation becomes greater.

3. Gas blowing across the carinae (fig. 1) of the bronchial bifurcations, which is comparable to the blowing of the lip of an organ pipe [10], may produce wave motions in the bronchi and also alter their gas content.

The first two mechanisms result in a mutual shaking and squeezing of neighbouring parenchymal lung segments, and thus may contribute to intra- and interparenchymatous gas mixing. The “organ pipe mechanism” is thought to vary during the cyclic variation in pressure because the altered geometry of the bronchial system exposes different numbers of bifurcations to the gas stream during the phasic modulation of gas flow.

There are five possible ancilliary mechanisms:

1. There may be gas transfer from “high pressure” to “low pressure” areas (collateral flow) [13]. Interalveolar windows, ascribed to (but not described first by) Kohn (1893), may function as one pathway for “one-way” gas flow.

expiratory airway resistance. At frequencies greater than 1–2 Hz an increase in end-expiratory lung volume occurs.

When classifying some variants of clinical jet ventilation as “Rapid Ventilation” rather than as
(ii) Oscillatory flow or “pendelluft” [25] may be induced between respiratory units with different mechanical properties and time constants may result in intersegmental gas mixing. As the oscillating gas flow passes the well flushed central bronchus twice per period, it may be renewed with fresh gas and freed from carbon dioxide.

(iii) The markedly non-linear pressure-diameter relation of the bronchi contributes to a mixing procedure along the airways [17]. With increasing pressure, the bronchi dilate, rapidly at first, then more slowly. When the pressure decreases the bronchi collapse quickly at first, then slowly during the late phase of pressure change. The periodic pattern of changes in bronchial diameter over the exciting pressure cycle reflects an asymmetrically varying transverse flow component. The resulting radial gas movement, which is superimposed on the oscillatory axial flow, may contribute to mixing within the airways.

(iv, v) Two further mechanisms discussed widely in the literature [15, 18, 26, 27] (Taylor dispersion and asymmetrical flow profiles) are endobronchial flow and mixing processes. Investigation of these phenomena is hampered by lack of measuring methods which can be used within the airway system. Thus most of the data have been derived from investigations on rigid models interpreted in a broad mathematical approach [15, 27].

Techniques of measurement

What has been measured? What basic findings contribute to the particular gas mixing procedure during High Frequency Ventilation? Observations on lungs ventilated in the open chest and multifocal pressure measurements in the subpleural compartment [12, 13] confirm that not only does alveolar ventilation become non-homogeneous during monofrequent excitation above 10 Hz, but there is also a non-symmetrical distribution of the gas content within the lungs [28]. Some zones tend to remain motionless and collapse, whilst others are well inflated and follow the excitation.

We recently used core-to-surface impedance (fig. 4) to assess segmental length changes within the lung during ventilatory movements. When increasing the exciting frequency (fig. 5) from 3.14 to 62.4 Hz by a rotating valve tube ventilator [13] we obtained modulations of segmental length oscillations and in mean segmental length which were different between measuring sites.

Clinically, unequal distribution [28] of gas filling cannot be expressed in terms of FRC and cannot be assessed by measurement of chest circumference [5]. It is more likely to occur in the human adult lung and at high excitation frequencies, than in the lungs of a 20-30 kg dog during jet ventilation at 2–4 Hz. This suggests that data from experiments on dogs do not apply to clinical observations and data from “rapid jet ventilation” [4–6] cannot be compared with data from “high frequency ventilation” [1, 2, 11–13]. When dried (that is, rigid) lungs are being excited and when pressures or gas flow velocity in the subpleural compartment or in small epipleural chambers are being measured, local aerodynamics are found to be very non-homogeneous—that is, frequency dependent [13]. The same happens when the peripheral lung tissue is removed and pressures are measured in chambers mounted at the rigid bronchial ends. Lung parenchyma is thus
not a prerequisite for inducing inhomogeneities in a branching airway system and inhomogeneities occur in rigid airway systems in addition to freshly excised ones. This raises the question whether agitating the gas in the lungs or moving the tissue against its gas contents makes the greatest contribution to gas mixing in the airways.

Individual lung geometry is likely to affect the efficacy of alveolar ventilation as does bronchial length, curvature and angulation of branching and cross section of the airways (fig. 6). Geometrical factors are difficult to quantitate, but must be taken into account when evaluating experimental or clinical results.

**Transthoracic excitation**

Experiments on external excitation were performed originally in an attempt to excite the lung homogeneously [29, 30]. However, it became apparent that damping by the lung parenchyma was so effective that the energy transfer from the chest wall to the bronchial tree was almost completely abolished. Flushing of the bronchi and remodelling of the bronchial tree during cyclic excitation must obviously be less efficient than when the airway system is excited via the trachea. This approach, conceived as “home ventilatory assistance” for chronically ill patients, is unlikely to have a promising future.

**Use of the Bias Flow Connector in rapid and high frequency jet ventilation**

In clinical practice a bias flow is often used (fig. 7). This was designed to wash out carbon dioxide, but it has now been suggested that it may act also as a pressure limiting “valve” which prevents overinflation of the lungs. In the original concept, the loss of energy throughout the system was kept small by attaching a long tube which served as a low pass filter [1, 2]. In fact, the device tends to make the system behave in a manner similar to pressure-controlled ventilation. The inspiratory flow down the tracheal tube into the lungs ceases when a certain pressure is attained in the airways. Gas which continues to be delivered from the generator is released in a retrograde direction through the tube or through the bias flow connector to atmosphere.

Some systems are wide open to atmosphere, so that the volume insufflated into the lungs varies markedly and, in some patients with a low lung compliance, there may be little or no alveolar ventilation.

These devices obviate the need for complex automatic controls for prevention of barotrauma, and they facilitate use by less highly trained staff. However, it is not acceptable for imperceptible changes in lung function to be able to change the
ventilation pattern so that a critical reduction in alveolar ventilation occurs.

**Monofrequent excitation**

Monofrequent excitation is the most common mode of application of high frequency ventilation, although experimental data suggest that homogeneous alveolar ventilation requires a broad frequency spectrum [12, 13, 16, 20].

The rational application of high frequency ventilation aims at selective, segmental ventilation. This implies that the diseased lung should not be treated globally, as with conventional ventilation, but should be mobilized or immobilized segmentally, according to the distribution of the disease.

Thus the aim is to provide the required frequency range to any compartment of the lung in such a way that the local mean pressure is great enough to keep any segment inflated for uptake of oxygen, whilst alveolar vibration amplitude is kept small enough to prevent damage but not too small for adequate elimination of carbon dioxide. The best way to do this would be to produce excitation with a homogeneous power content of 5–40 Hz. Unfortunately, it appears technically impossible to achieve this in the airways. As an alternative, we have used a frequency sweep—that is, a periodic frequency change of 5–40 Hz, which may be achieved easily with a rotating valve tube. However, the time constants for deflating and inflating any compartment of the lung differ, especially in the non-homogeneous lung seen in intensive care patients. Even in experiments on

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**Fig. 6.** Comparison of the geometry of a human and a porcine lung in the blown and dried state shows the human lung to be more or less a cube, whereas the porcine lung represents an elongated rectangular body. As a result, the length of the bronchi (the distances from the bifurcation to the pleura) in different compartments of the human lung are more homogeneous than in the porcine lung.

**Fig. 7.** Bias flow coupling connectors between the high frequency ventilator and the tracheal tube are designed to deliver fresh gas, to eliminate carbon dioxide and to conserve energy. Some variants are also intended to produce autoregulation of tidal volume by generating a pressure controlled mode of ventilation. BF = Bias flow; Exs = Exciting system (high frequency ventilator); FG = fresh gas delivery (left) and elimination of carbon dioxide (right); LPF = low-pass filter (a tube up to 8 m long which allows carbon dioxide-contaminated exhaled gas to be washed out without too great a loss in exciting energy). Sources: A [1, 2]; B [4, 5, 26]; C [4, 5].
healthy dogs, when the exciting frequency was varied systematically, there was no homogeneous inflation of peripheral lung segments.

In this context, it might be useful to remember that measurement of chest circumference by strain gauges [5] merely indicates the mean lung volume but does not assess regional variations. It is important to know the mean lung volume in order to predict circulatory depression. However, during high frequency ventilation, venous return to the right heart is likely to be unchanged, rather than reduced, compared with conventional ventilation [31].

REFERENCES
