5. V. S. Kononenko, Fiziol. Zh. (Kiev), 25, No. 5, 529 (1979).
6. Ya. B. Maksimovich, E. R. Kukurichkin, S. S. Rybalova, and I. P. Chaikovskaya, Farmakol. Toksikol., No. 3, 22 (1985).
7. S. F. Semenova and A. P. Chuprikov, Zh. Nevropatal. Psikhiat., No. 12, 1798 (1975).
8. V. Yu. Urbakh, Statistical Analysis in Biological and Medical Research [in Russian], Moscow (1975).
9. E. I. Chazov and N. P. Bekhtereva, Vest. Akad. Nauk SSSR, No. 4, 14 (1986).
10. A. P. Chuprikov, Vest. Akad. Med. Nauk SSSR, No. 11, 53 (1974).
11. G. Heister and H. Schroeder, Cortex, 20, No. 2, 271 (1984).
12. S. D. Glick et al., Life Sci., 25, No. 4, 395 (1979).
13. S. D. Glick and D. A. Ross, Trends Neurosci., 4, No. 8, 169 (1981).
14. R. Waziri, Psychopharmacology, 68, No. 1, 51 (1980).

INTENSIFICATION OF GAS EXCHANGE DURING HIGH-FREQUENCY ARTIFICIAL VENTILATION

L. I. Nemerovskii, E. V. Vikhrov,
E. M. Levité, and M. M. Rainov

UDC 615.816.2.015.4:
612.22/23].07

KEY WORDS: high-frequency artificial ventilation; oscillations of the lungs; blood oxygenation.

The method of high-frequency (HF) artificial ventilation of the lungs (AVL) is being used increasingly in the treatment of acute respiratory failure, because of the possibility of intensifying the gas exchange and correcting the hypoxemia which develops as a result of disturbances of ventilation-perfusion ratios and an increase in arteriovenous shunting, which cannot be corrected by ordinary AVL. No satisfactory explanation of the causes of the intensification of gas exchange during HF AVL is yet forthcoming, and this limits the development and more widespread introduction of this type of AVL.

The mechanism of intensification of gas exchange during HF AVL is discussed in this paper, with the aim of determining its optimal parameters.

Traditionally research workers have devoted most attention to the mechanism of gas transport along the bronchial tree. The cause of improvement of ventilation, for example, during jet HF AVL, is considered to be the high initial velocity of the gas jet, which promotes direct carriage of oxygen into the distal portions of the bronchial tree [8]. It is also considered that the gas flows are separated — to the alveoli (along the axis of the bronchial lumen) and in the opposite direction (along the walls of the bronchus), as a result of which the velocity of gas exchange in the lungs is increased. Because of the increased velocity of the jet, turbulence of the gas mixture leads to its more effective mixing and, consequently, to acceleration of gas exchange at the level of bronchi of higher orders than in ordinary types of AVL [5]. These suggestions apply only to gas transport along the bronchi and have nothing to do with diffusion processes in the alveoli. Meanwhile hypoxemia can be overcome only by increasing the volume of oxygen absorbed by the blood, and in turn, this can take place through a reduction in the volume of arteriovenous shunting on account of an increase in the capacity of alveoli ventilation, acceleration of the diffusion of gases in the alveolus, or a combination of both processes. Consequently, the question arises: what determines acceleration of oxygen diffusion into the blood and an increase in the volume of alveolar ventilation in different methods of HF AVL [7]. One possible answer to this question can be formulated as follows.

During jet HF AVL, which consists essentially of injecting a pulsed jet of gas into the air passage of the lungs with a definite following frequency (1-5 Hz), the development of a double oscillatory process is observed in the lung

---

Institute of General Resuscitation, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 108, No. 8, pp. 142-144, August, 1989. Original article submitted June 21, 1988.

0007-4888/89/0008-1071$12.50

TABLE 1. Parameters of Acid-Base Balance and Gas Composition of Patients' Arterial Blood during AVL and a Combination of AVL with Vibration

Parameter	AVL	AVL + vibration
pH	7,53±0,01	7,52±0,02
P_aCO_2, mm Hg	28,72±0,98	30,78±1,38
BE, mmoles/liter	+2,32±0,33	+2,79±0,63
P_aO_2, mm Hg	76,82±3,00	95,61±6,96*

Legend. Asterisk indicates statistically significant difference.

parenchyma. One of them is due to the following frequency of the gas pulses. It can be postulated that if the above-mentioned frequency range of jet pulses is used, the following frequency of the pulses will be equal or approximately equal to the natural frequency of the respiratory apparatus of the particular individual (the frequency of air oscillations at which conditions of resonance are produced on injection into the respiratory passages), which causes resonance or near-resonance oscillations of the respiratory apparatus. Meanwhile, in the intervals between pulses of pressure, because of the elasticity of compressed air in parts of the lung parenchyma and inertia of adjacent areas of the chest wall, damped self-oscillations are organized in the chest wall which, in this case, is the motor transmitting oscillations to areas of the lung parenchyma. The frequency of damped self-oscillations is much higher than the following frequency of the pulses of gas pressure. It follows from the theory and practice of pulmonography [3] that resonance conditions are maintained in areas of the lung parenchyma for a wide range of frequencies (30-250 Hz). Damped self-oscillations of the chest wall lie within these limits.

The amplitude of oscillations of gas particles in the alveoli is commensurate with the size of the alveoli. If at the moment the jet of gas is injected into the lungs a pressure of about 200 mm water is created, the value of the maximal displacement of the chest wall (with a total capacity of the lungs of 4.5 liters) in intervals between pulses, when the pressure falls, will be 0.8 mm (the average diameter of the alveolus is 0.3 mm). This determines mixing of the gas with equalization of the oxygen concentration gradient both in the alveoli and in the small bronchi. Under these circumstances the oxygen concentration rises in the juxtamural layer of alveolar air, which accelerates its passage through the alveolar membrane into the blood.

An explanation of this effect can be given on the basis of the theory of spread of sound waves in the lungs [3]. Areas of lung parenchyma together with adjacent areas of the chest wall constitute natural oscillatory circuits in which resonance vibrations are excited during the application of oscillations of different frequencies: the higher the frequency of the oscillations, the smaller the area of parenchyma adjacent to the chest wall in which resonance arises, i.e., a sharp increase in amplitude of the vibrations is associated with a low amplitude of applied oscillations. For example, if oscillations with a amplitude of ±5 ml (at the entrance into the upper respiratory passages) are used, displacement of the chest wall in the absence of resonance will be only 0.04 mm. Because of resonance, the displacement is increased 20-fold [3] and now amounts to 0.8 mm. Thus the oscillatory HF AVL promotes intensification of the gas exchange through mixing of the gases in the alveoli space under the influence of an oscillatory process in the air column of the lungs.

The mechanism of intensification of the gas exchange is thus the same when both methods of HF AVL are used. The differences are that during jet HF AVL the oscillatory process takes place on account of free damped oscillations of the chest wall and lung parenchyma, whereas during oscillatory AVL it is caused by excitatory oscillations reaching resonance circuits, formed by areas of lung parenchyma and chest wall.

As regards pulses applied with a frequency of 1-5 Hz during jet HF AVL, they perform mainly a transport function of supplying the necessary quantity of gas mixture to allow alveoli ventilation. This relatively low-frequency process is less important for accelerating the diffusion of gases, for under these circumstances the rate of mixing of the gas particles is low and commensurate, just as with ordinary AVL, with the rate of diffusion of the gases.

There is evidently another cause of intensification of gas exchange in the lungs. Oscillations of the air column in the lungs, excited during HF AVL, disturb adhesion of the exudate with the epithelium of the bronchi and bronchioles, thus helping to remove the exudate into the lumen of lower-order bronchi. This opens access of air into previously closed areas of the lungs. As a result of this the number of unventilated alveoli is reduced and the volume of oxygen diffusing through the alveolocapillary membrane is increased.

The effect of this mechanism on improvement of the gas exchange depends on the concrete state of the patient's lungs and, in some cases, it can play a decisive role in the abolition of hypoxemia. It is important to emphasize that in both cases the factor inducing intensification of the gas exchange was oscillatory processes arising in the lung.

HF AVL, unlike other methods of AVL, probably not only supplies fresh portions of the respiratory mixture, but also, on account of the interrupted flow of gas and excitation of the oscillatory process in the lung tissue, reduces the arteriovenous shunting volume and increases the oxygen concentration gradient in the zone of the alveolocapillary membranes, which contributes to intensification of the gas exchange.

To confirm the hypothesis under discussion, clinical observations were made on 14 patients with acute respiratory failure of varied etiology (atelectasis, pneumonia, shock lung) and with developed hypoxemia, which could not be corrected by traditional AVL by the addition of a vibrator, connected pneumatically with the adapter of the respirator. The membrane vibrator worked off an electric motor with frequency of 65 Hz, the amplitude of oscillations of the membrane was 4 mm, and the area of the membrane was about 4 cm^2. AVL was applied under the following conditions: respiratory minute volume 10-16 liters/min, tidal volume 500-700 ml, inspiratory pressure not more than 35 cm water, final expiratory pressure 0 + 12 cm water, FiO_2 0.4 to 0.6. The results of the observations are given in Table 1.

Of 24 investigations, in 22 an increase in p_aO_2 on average by 20% (from 76 to 95 mm Hg) was recorded.

For instance, in patient I., a man aged 54 years, after subtotal resection of the stomach for tumor, extensive right-side pneumonia developed with respiratory failure and hypoxemia, requiring AVL. After temporary correction of the blood gas composition the hypoxemia progressively worsened and p_aO_2 fell to 58.6 mm Hg. The hypoxemia was abolished 1 h after connecting the vibrator (with AVL continued under the previous conditions) and p_aO_2 rose to 92.2 mm Hg.

In most cases reproducibility of the results of the action of oscillations on the value of p_aO_2 was obtained: during repeated activations of the vibrator (after an interruption in its work and a fall of p_aO_2) p_aO_2 was observed to increase again. In the same patient I., after a positive effect had been obtained, the vibration was stopped. After 1 day hypoxemia was observed again: p_aO_2 fell to 69.7 mm Hg. On reconnecting the vibrator p_aO_2 increased to 143 mm Hg. Continuous application of AVL with pneumovibration for 3 days led the gas exchange to stabilize and enabled the patient to resume unaided breathing.

Improvement of oxygenation as a result of the combined use of traditional and oscillatory HF AVL was not accompanied by an significant change in p_aCO_2 (Table 1). This state of affairs can be used as an argument in support of the hypothesis under discussion. Hypoxemia accompanying respiratory failure is due mainly to an increase in the arteriovenous shunting volume. Correction of hypoxemia, as already stated, is therefore possible only by reducing the shunting, by increasing the respiratory surface of the lungs (freeing the air passages, reducing the dead space) or by increasing the p_AO_2-p_aO_2 gradient (more precisely, the oxygen concentration gradient in the juxtamural alveolar layer and capillary blood) and acceleration of transmembrane diffusion. The value of the gradient is of no special importance for CO_2 diffusion, for even if the gradient is 0.03 mm Hg, the exchange of this gas between blood and alveolus is adequate [1, 2]. For the elimination of CO_2, the volume of the tidal air and not the intra-alveolar diffusion is of decisive importance. Accordingly, acceleration of diffusion by oscillations is effective only on the oxygen exchange.

That this explanation is correct is confirmed by the fall in p_aCO_2 due to an increase in alveolar ventilation under certain conditions of jet HF AVL [4] and, conversely, the increase in p_aCO_2 during oscillatory HF AVL alone [7].

In the course of observations on two patients, no increase in p_aO_2 was found after connection of the pneumovibrator. Both patients were found to have a pneumothorax, after closure of which vibration again became effective.

It can be tentatively suggested that when pneumothorax develops, besides the reduction in the respiratory surface of the lung, another relevant factor may be disturbance of the conditions of resonance in the acoustic circuit, for the formation of an air gap between the lung parenchyma and the chest wall prevented resonance formation.

To sum up, it can be stated with a fair degree of confidence that intensification of the gas exchange takes place due to oscillatory processes in the alveolar space against the background of adequate lung ventilation. Accordingly, a combination of traditional volume AVL with oscillatory AVL can be regarded as the most promising procedure for clinical use.

LITERATURE CITED

1. C. Blaja and S. Krivda, Theory and Practise of Resuscitation in Surgery [in Russian], Bucharest (1967).
2. V. L. Kassil', Artificial Ventilation of the Lungs in Intensive Care [in Russian], Moscow (1987).
3. L. I. Nemerovskii, Pulmonography [in Russian], Moscow (1981).
4. A. N. Charnyi, Pathophysiology of Hypoxic States [in Russian], Moscow (1961).
5. A. Bancalari, T. Gerhardt, E. Bancalari, et al., J. Pediat., 110, No. 4, 617 (1987).
6. A. Ben-Jebria, Int. J. Biomed. Comput., 15, No. 1, 35 (1984).
7. J. M. Harst and R. D. Branson, Curr. Surg., 44, No. 1, 7 (1987).
8. P. W. Scherer, F. R. Haselton, and J. R. Seybert, Ann. Biomed. Eng., 12, No. 4, 385 (1984).