Effect of High-Frequency Ventilation of the Lungs on the Pulmonary and Systemic Circulation in Pulmonary Artery Microembolism

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UDC 616.131-005.7-02:615.816.2]-07:616.1-008.1

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol.116, № 11, pp. 466-469, November, 1993
Original article submitted April 21, 1993

Key Words: *high-frequency artificial ventilation of the lungs; pulmonary artery microembolism; pulmonary circulation; systemic circulation; ultrasound*

Pulmonary microembolism may be caused by massive transfusions of preserved blood, by operations with extracorporeal perfusions, by locomotor system injuries, and other events [9]. There are reports on the use of various regimens of traditional artificial ventilation of the lungs (AVL) [7,10,11] and of high-frequency (HF AVL) ventilation [12-15] in pulmonary microembolism and edema; however, the relationships between lesser and greater circulation hemodynamics in pulmonary artery microembolism during various types of AVL are still to be researched. Some attempts have been made to study the pulmonary macrocirculation under such conditions [2,9]. We investigated the potentialities of ventilation therapy of pulmonary artery microembolism using both traditional AVL and HF AVL. Previously we examined the hemodynamic status during traditional AVL replacement by HF AVL in intact animals [8].

In the present study we compare the effects of traditional and high-frequency AVL on the pulmonary and systemic circulation in pulmonary artery microembolism and edema associated with it.

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MATERIALS AND METHODS

Linear and volumetric blood flow velocity were measured by the ultrasonic method [4] in the ascending aorta and pulmonary artery cone in acute experiments on 22 male cats weighing 2.3 to 3.2 kg with an open chest under nembutal anesthesia (30-40 mg/kg intraperitoneally). The balance between the right and left ventricular output was assessed as the ratio of mean blood flow values in the pulmonary artery cone and ascending aorta using an analog computer. Blood pressure (AP) was measured in the pulmonary and femoral arteries with a microelectromanometer [5]. Experiments were carried out on the open chest under AVL. Traditional AVL was carried out using a DAM EPM-2 respiratory device for laboratory animals with a 30-40% fraction of inhaled oxygen (F_iO_2=0.3-0.4). Ventilation parameters were as follows: respiratory volume 50-80 ml, minute respiratory volume 1.1-2.1 ml/min, respiration rate 15-21/min. HF AVL was carried out with a SPIRON-601 device. Ventilation was carried out in a jet mode at 100/min frequency, 1/2 inspiration to expiration ratio, minute respiratory volume 1.9-2.5 ml/min, and F_iO_2 0.8-0.9.

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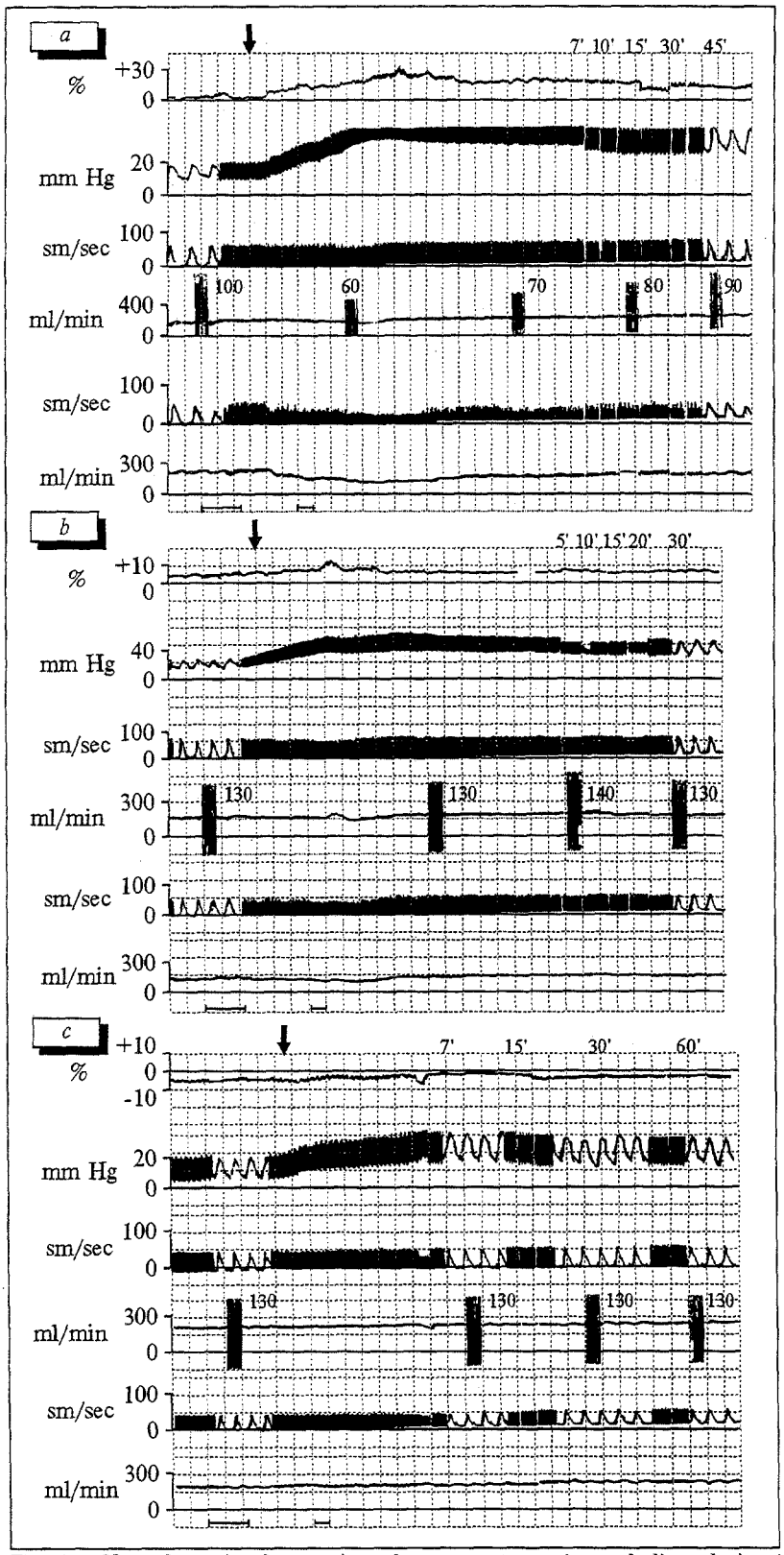

Fig. 1. Hemodynamic changes in pulmonary artery microembolism during traditional AVL (*a*) and HF AVL (*b*, *c*). From top to bottom: balance between right and left ventricular output in relative units (curve directed upward corresponds to blood flow increase in the ascending aorta in relation to blood flow in pulmonary artery cone), AP in pulmonary artery, blood flow linear velocity in ascending aorta, blood flow volumetric velocity in ascending aorta, blood flow linear velocity in pulmonary artery cone, and blood flow volumetric velocity in pulmonary artery cone. Thin lines under each of the curves: zero levels. Arrow shows onset of pulmonary artery microembolism. Time scale 1 and 10 sec. Numbers at top: time from microembolism onset, min. Bars: Ap in femoral artery.

Tracheal pressure during both types of ventilation was assessed using an indicator pressure gauge in cm H_2O through a thin catheter introduced into the trachea to the point of bifurcation. HF AVL was adapted to the cat minute respiratory volume by regulating the working pressure of the SPIRON-601 device.

The pulmonary microcirculation was examined by a modified biomicroscopy method [1]. The capillary perfusion index was calculated as the length of blood-perfused alveolar capillaries per 10,000 μ^2 of pulmonary area.

Cardiac output values in the aorta and systemic AP were used to estimate greater circulation parameters; for lesser circulation assessment values of cardiac output to the pulmonary artery and pulmonary AP were taken.

Pulmonary artery fatty microembolism was induced by intravenous infusion of olive oil (1.2 ml/kg, 1 ml/min) [2,7]. The intensity of developing pulmonary edema was assessed by the pulmonary coefficient and dry residue value [3]. The data were statistically processed using an RDR computer (USA) by the paired comparison method with the Student *t* test.

RESULTS

Pulmonary artery microembolism results on a drastic increase of the pulmonary vascular resistance by 487% on average during traditional AVL and by 252% during HF AVL by the third minute of the experiment. This causes the mean pressure to rise in the pulmonary artery, by 167% in traditional AVL (Fig. 1, *a*) and by 127% in HF AVL (Figs. 1, *b*, *c*; 2, *a*, *b*).

The appearance of blood flow obstruction presenting as microemboli results in a reduction of the blood flow volumetric velocity in the pulmonary artery: by 60% in traditional AVL and by 34% in HF AVL. The time course

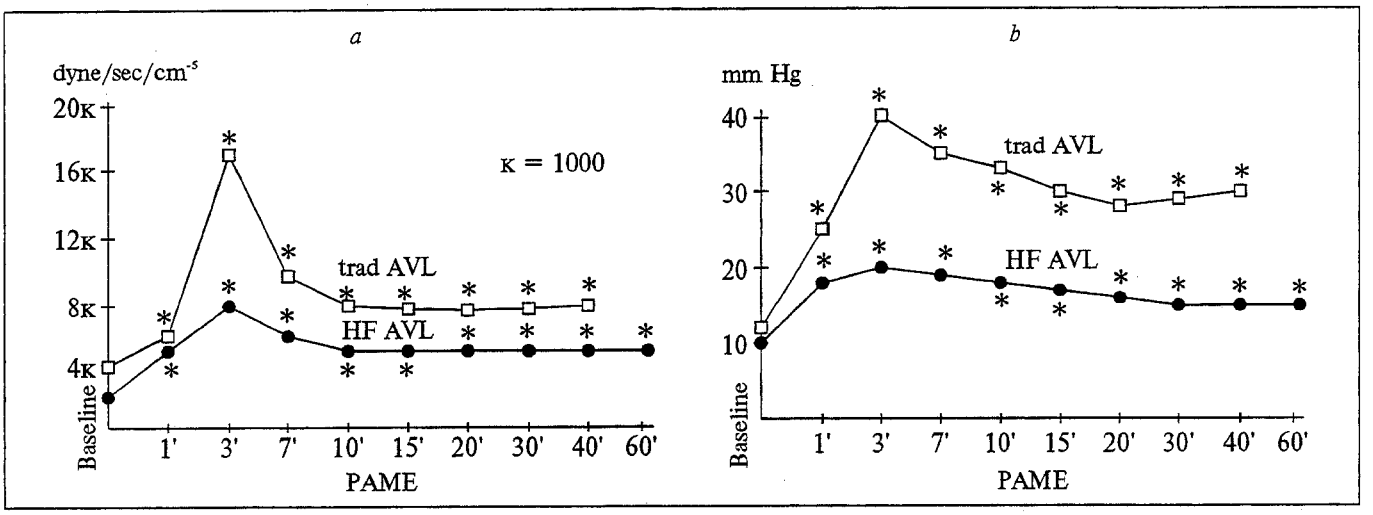

Fig. 2. Changes in pulmonary resistance (*a*) and mean pressure in pulmonary artery system (*b*) in pulmonary artery microembolism during traditional AVL and HF AVL. PAME: pulmonary artery microembolism. *: reliability of changes ($p<0.05$).

of blood flow linear velocity in the pulmonary artery is different: this parameter is unchanged in HF AVL in the majority of cases over the entire experiment ($p>0.05$), whereas in traditional AVL a 68.7% reduction of linear blood flow velocity in the pulmonary artery is observed in the third minute of the experiment (from 52.71±1.67 to 16.11±5.23 cm/sec). By the 20th min the linear velocity of the pulmonary artery blood flow increases to 46.3±5.72 cm/sec and then irreversibly drops.

The balance between the right and left ventricular output is altered in microembolism, with an intensive redistribution of blood into the greater circulation (Fig. 3, *a*). During HF AVL the imbalance period ($p<0.05$) is 5 to 7 min, after which this parameter normalizes. During traditional AVL the imbalance stabilizes at high values with 30% blood redistribution into the greater circulation. Under such conditions in traditional AVL, evidently due to the Parin-Schwigk reflex [6], the systemic AP drops to 51% of its initial level in the third minute after embolism onset (from 135±3.41 to 66.7±7.32 mm Hg). This is paralleled by a 36.2% reduction of the total peripheral vascular resistance in the 7th min ($p<0.05$). During HF AVL changes in the systemic AP and total peripheral vascular resistance are less expressed or absent ($p<0.05$).

Right ventricular work increases in pulmonary artery microembolism. Lower values of right ventricular work in HF AVL ($p<0.05$) result from a lesser load on the right heart than in traditional AVL, that is, from a smaller increase of resistance and pressure in the pulmonary artery system.

Acute fatty microembolism did not cause irreversible changes in hemodynamics under conditions of HF AVL. This resulted in prolongation of the life of animals exposed to HF AVL by 4 times and longer in comparison with those subjected to

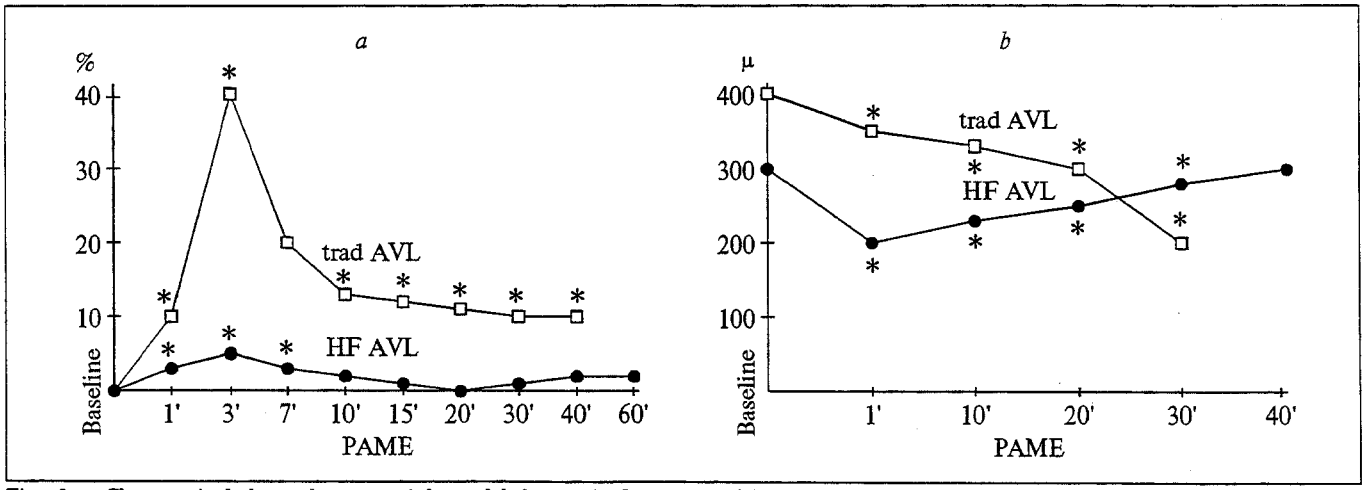

Fig. 3. Changes in balance between right and left ventricular output (*a*) and pulmonary capillary perfusion index (*b*) in pulmonary artery microembolism during traditional and HF AVL.

traditional AVL. In traditional AVL microembolism results in a marked imbalance between the lesser and greater circulation which is incompatible with life. Assessment of the degree of pulmonary tissue edema after the experiment demonstrated that the pulmonary coefficient (8) and dry residue (19%) after HF AVL did not differ from the normal values ($p>0.05$) and were reliably better than the values after traditional AVL: pulmonary coefficient = 12.5 and dry residue = 12%.

The time course of interstitial edema and degree of microcirculation obstruction are similar from the first to the tenth minute of microembolism whatever the type of ventilation, although hemodynamic differences in animals exposed to traditional and HF AVL are evident as early as the third minute. The capillary perfusion index is reduced ($p<0.05$) in this period for both types of AVL (Fig. 3, *b*). Differences in pulmonary microcirculation status under conditions of traditional and HF AVL become evident starting from the 10th min of embolism. Traditional AVL is associated with fatty blockage of pulmonary microvessels with poor development of a bypass. The interstitial edema signs augment as the capillary perfusion index progressively decreases. Later, alveolar edema develops in the presence of a massive interstitial edema. Death occurs within 40 to 60 min after embolism onset.

The functioning of compensatory mechanisms including blood flow bypass is much better expressed during HF AVL than during traditional AVL. Fragmentation of fatty elements occurs starting from the very first minutes of embolism in HF AVL. The interstitial edema phase is present in HF AVL but is much less manifest than in traditional AVL. An alveolar edema phase was not observed in HF AVL.

The decompensation stage characteristic of traditional AVL, which was previously identified as the third stage of pulmonary embolic edema development [7], was absent in animals exposed to HF AVL. It was replaced by a pulmonary edema resolution stage, with capillary perfusion index recovery to the initial level ($p>0.05$) (Fig. 3, *b*).

Fatty emboli are fragmented in HF AVL due to the effect of respiratory jet high-frequency resonance oscillations on the pulmonary microcirculation, followed by clot passage into the pulmonary veins system. The pulmonary microcirculation under conditions of HF AVL may be largely responsible for the greater and lesser circulation hemodynamics and pulmonary water balance, be the principal condition for microembolic block resolution, and cause an appreciable prolongation of life under conditions of fatty microembolism.

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