PHYSIOLOGY

Influence of Changes in Intrathoracic and Central Venous Pressure on Cardiac Filling Dynamics

Zh. A. Donina, I. N. Lavrova, M. A. Tikhonov*, A. N. Kotov*, V. I. Kolesnikov*, and V. M. Baranov*

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Changes in the ratio between intrathoracic and central venous pressure were studied in narcotized cats under conditions of constant positive or negative pressure ventilation. Transformation of elastic characteristics in the respiratory system caused by changes in intrathoracic pressure led to inversion of the ratio between transpulmonary intrathoracic and central venous pressure determining right atrial filling pressure.

Key Words: *right atrial filling pressure; intrathoracic pressure; central venous pressure*

Changes in respiratory mechanics produced by correction of the transpulmonary or transthoracic pressure (water immersion; negative pressure ventilation; various types of artificial ventilation; and prevention of breathlessness during professional activity under extreme conditions, including deep-water diving, gravitational overload, and emergency situations in high-altitude flights) affect intrathoracic hemodynamics and modulate venous return, cardiac filling pressure, and cardiac output [1,2,5]. Most invasive studies of these factors were performed after thoracotomy under conditions of artificial ventilation [4]. Therefore, the role of intrathoracic pressure (ITP) as the factor providing respiratory mechanics and intrathoracic hemodynamics was misinterpreted. During spontaneous breathing cyclic changes in the right atrial filling pressure are mainly related to respiratory phase-dependent variations in the ratio between ITP and central venous pressure (CVP). Changes in the ratio between physical parameters (elasticity, resistance, and internal strain of tissue structures) determining respiratory mechanics and intrathoracic hemodynamics under conditions of constant positive or negative pressure in the thoracic cavity can modulate cardiac filling pressure [5].

Here we evaluated right atrial transmural pressure as the function of the ratio between ITP and CVP during respiration under conditions of positive or negative pressure.

MATERIALS AND METHODS

The study was conducted according to principles of biological ethics. Experiments were performed on 15 tracheostomized cats weighing 2.5-3.0 kg and narcotized with ketamine (40 mg/kg) during spontaneous breathing under positive or negative pressure. The animals were in the horizontal position. We used the method of body plethysmography. ITP was measured through a probe with a rubber balloon introduced into the lower third of the esophagus. Blood pressure in the common carotid artery and CVP in the anterior vena cava at the level of the heart were estimated via catheters connected to PDP-300 and PDP-1000 pressure transducers. ECG was recorded using needle electro-

Laboratory for Physiology of Respiration, I. P. Pavlov Institute of Physiology, Russian Academy of Sciences, St. Petersburg; *Russian State Research Center, Institute of Medical and Biological Problems, Russian Academy of Sciences, Moscow. ***Address for correspondence:*** isaev@kolt.infra.ru. Donina Zh. A.

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des. Positive and negative intrathoracic pressure was modeled by elevation or reduction of pressure in the plethysmographic chamber (from 5 to -5 cm H_2O). Right atrial transmural pressure (CVP_{TM}) was calculated as the difference between anterior vena cava pressure and ITP. The respiratory volume (RV) and functional residual capacity were determined after a plethysmographic study. Pulmonary distensibility (C) was calculated as: $C=\Delta RV/\Delta ITP$. The indexes were recorded during inspiration and expiration. The measurements were performed by the end of the 1st minute at each stage of observations.

The study was performed by the following scheme: control (3-min breathing atmospheric air); positive transthoracic pressure (3-min breathing under negative pressure) and negative transthoracic pressure (3-min breathing under positive pressure); and negative transpulmonary pressure (3-min breathing under negative pressure). The results were analyzed by Student's *t* test.

RESULTS

The intrapulmonary pressure remained unchanged after pressure in the plethysmographic chamber was increased to 5 cm H_2O (positive transthoracic pressure, Fig. 1, *2*), but the animal breathed under a negative pressure (relative to pressure in the chamber). Negativeness of ITP during expiration increased from -0.2±0.3 to -4.3±0.5 cm H_2O ($p<0.001$). Inspiratory ITP decreased from -5.7±1.1 to -10.5±1.0 cm H_2O ($p<0.001$). Expiratory pressure in the anterior vena cava decreased from -0.9±0.6 to -5.6±0.6 cm H_2O ($p<0.001$). Inspiratory pressure in this vein decreased from -4.1±1.1 to -12±1 cm H_2O ($p<0.001$). Expiratory CVP_{TM} decreased from -0.5±0.5 to -0.9±0.8 cm H_2O ($p>0.05$). Inspiratory CVP_{TM} decreased from 1.6±0.5 to -1.6±0.5 cm H_2O ($p<0.05$). RV decreased from 41.5±4.3 to 25.2±5.4 cm^3 ($p<0.05$). Functional residual capacity was shifted by 57.4±9.1% towards expiration. Pulmonary distensibility decreased from 8.8±1.0 to 4.3±1.0 ml/cm H_2O ($p<0.001$). Cardiovascular indexes remained practically unchanged.

The negative transthoracic pressure (-5 cm H_2O, Fig. 1, *3*) increased expiratory ITP (from -0.2±0.3 to 5.3±0.8 cm H_2O, $p<0.001$), inspiratory ITP (from -5.7±1.1 to 0.6±0.8, $p<0.001$), expiratory CVP (from -0.9±0.6 to 4.1±0.9 cm H_2O, $p<0.001$), and inspiratory CVP (from -4.1±1.1 to -0.6±1.0 cm H_2O). We observed a decrease in expiratory CVP_{TM} (from -0.5±0.5 to -0.9±1.4 cm H_2O, $p>0.05$) and inspiratory CVP_{TM} (from 1.6±0.5 to -1.2±1.2 cm H_2O, $p<0.05$). RV increased insignificantly (from 41.5±4.3 to 50.9±5.0 cm^3, $p>0.05$). Functional residual capacity was shifted by 41.7±4.6% towards inspiration. Pulmonary distensibility increased from 8.8±1.0 to 12.6±1.4 ml/cm H_2O ($p<0.05$). Breathing under negative transpulmonary pressure (-5 cm H_2O, Fig. 1, *1*) decreased expiratory ITP (from -0.2±0.3 to -3.0±0.5 cm H_2O), inspiratory ITP (from -5.7±1.1 to -10.5±0.8 cm H_2O, $p<0.05$), expiratory CVP (from -0.9±0.6 to -4.4±0.7 cm H_2O, $p<0.05$), and inspiratory CVP (from -4.1±1.1 to -11.2±1.0 cm H_2O, $p<0.05$). We observed a decrease in expiratory CVP_{TM} (from -0.5±0.5 to -1.0±0.8 cm H_2O) and inspiratory CVP_{TM} (from 1.6±0.5 to -0.7±0.9 cm H_2O, $p<0.05$). RV decreased from 41.5±4.3 to 21.6±4.1 cm^3 ($p>0.05$). Functional residual capacity was shifted only by 14.0±2.1% towards expiration. Pulmonary distensibility decreased from 8.8±1.0 to 3.4±0.8 ml/cm H_2O ($p<0.05$) and was lower than during breathing under positive transthoracic pressure. Blood pressure did not differ from the control.

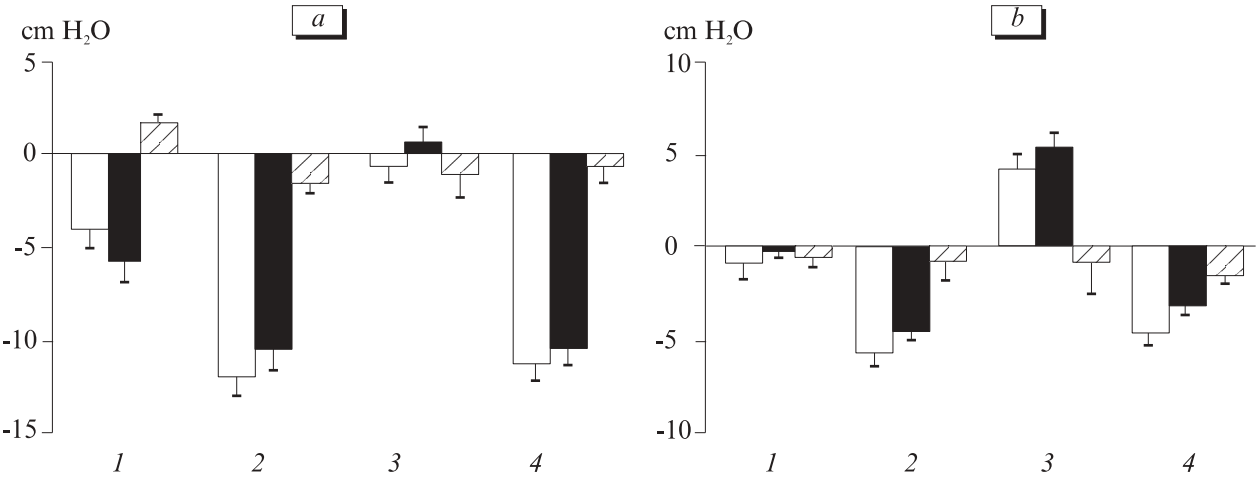

Fig. 1. Effect of positive or negative pressure ventilation on changes in central venous (light bars), intrathoracic (dark bars), and cardiac filling pressure (hatched bars) during inspiration (*a*) and expiration (*b*): control (*1*) and positive transthoracic (*2*), negative transthoracic (*3*), and negative transpulmonary pressure (*4*).

These data show that breathing under positive transthoracic and negative transpulmonary pressure is accompanied by similar changes in respiratory mechanics, anterior vena cava pressure, and cardiac filling pressure. Despite differences in the nature of mechanical factors affecting the respiratory system, these treatments were followed by an increase in negative ITP. In both cases the animals breathed under constant negative pressure. Therefore, biomechanical characteristics of breathing and hemodynamics underwent the same changes. The decrease in RV, shift in functional residual capacity towards expiration, and reduction of pulmonary distensibility attest to similarity of these states. ITP and CVP decreased during inspiration and expiration. Despite increased "pumping" activity of the thorax, the observed changes were not followed by the increase in the right atrial transmural pressure.

Negative transthoracic pressure (-5 cm H_2O) caused a shift in functional residual capacity towards inspiration and increased RV and pulmonary distensibility. Inspiration and expiration occurred in the expanded lungs. Therefore, the process of breathing proceeded under elevated ITP. Under these conditions we observed an increase in bothITP and CVP. However, cardiac filling pressure in both respiratory phases remained below the control level.

Phasic changes in ITP and CVP were studied under control conditions (inspiration of atmospheric air in the horizontal position). CVP_{TM} in the anterior vena cava increased, while CVP and ITP decreased during inspiration (Fig. 1, *1*). We compared indexes of breathing under negative or positive pressure and control conditions. Since ITP decreased more significantly than CVP, cardiac filling pressure increased only during the inspiratory phase of normal breathing. CVP_{TM} did not increase under conditions of negative and positive pressure ventilation. In the inspiratory and expiratory phase the absolute value of ITP was more positive than that of CVP. Moreover, CVP_{TM} is the algebraic sum of venous pressure and ITP. During inspiration ITP decreases, while CVP_{TM} in vessels increases. These changes promote vasodilation. It should be emphasized that during inspiration intraabdominal pressure increases, but transmural pressure, diameter, and capacitance of abdominal vessels decrease [2]. The rise in the pressure gradient between abdominal and thoracic veins is followed by an increase in blood supply to thoracic veins. Blood supply to abdominal veins increases during expiration. Thus, ITP (difference between atmospheric pressure and pressure near lungs, intrathoracic vessels, and heart) contributes to respiratory mechanics and pressure gradient between the thorax and abdominal cavity determining venous return [2,3].

Despite the decrease in CVP, right atrial filling pressure increased only in the inspiratory phase of normal breathing (control). Changes in biomechanical characteristics of the respiratory system, thoracic veins, and heart chambers during positive or negative pressure ventilation led to inversion of the ratio between ITP and CVP determining right atrial CVP_{TM}.

Our results indicate that right atrial filling pressure during positive or negative pressure ventilation is determined by the ratio between transpulmonary ITP and CVP.

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