Does the System of Pleural Layers Determine Paradoxical Phenomena in Respiratory Mechanics?

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Spirogram, transpulmonary pressure, intraesophageal, intrapleural pressure, and pressure in a closed bronchus were recorded in 16 healthy rabbits under thiopental anesthesia (first series). Dead rabbits were placed in a chamber, where respiratory movements were simulated by changing pressure, and all the parameters were recorded (second series). The lungs were then isolated and placed in the Donders bell (third series). Deformation of the plateau in transpulmonary pressure during a 0.5-sec interruption of air flow was observed only in the first series of experiments. The amplitude of pressure in a closed bronchus exceeded intrapleural pressure only in the first series. It is concluded that pleura does not contribute to the paradoxical mechanics of respiration.

Key Words: *respiration; pleural layer; intrapleural pressure*

Deformation of a plateau of transpulmonary pressure (TPP) upon interruption of air flow and the higher amplitude of respiratory fluctuations of pressure in a bronchus than the amplitude of intrapleural pressure are a fundamental paradoxical phenomena contradicting the modern concepts on the respiratory mechanics [4]. An attempt was made to explain this paradox with the help of the theory of alveolar interdependency [5]. This theory admits the prevalence of negative pressure in alveoli over intrapleural pressure due to transmission of the respiratory muscle force to the lung surface via the system of pleural layers.

Our objective was to check up the hypothesis that the paradoxical phenomena in the mechanics of respiration are caused by the function of the system of pleural layers. The most obvious paradoxes have been chosen: the deformation of TPP plateau upon interruption of air flow with a valve and the prevalence of amplitude of pressure in a closed bronchus over the amplitude of intrapleural pressure.

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MATERIALS AND METHODS

Experiments were performed on 16 rabbits (body weight 2.3-3.4 kg) under intravenous thiopental anesthesia. Tracheostomal cannula was connected with a miniature pneumotachograph tube of a miniature spirograph. The movements of the spirograph drum were converted into electric signal with a rheostat. Pressure gradient in the pneumotachograph tube was measured with a PDP-12MD manometer. An automatic intermitting unit of air flow was placed between the pneumotachograph tube and the spirograph. This unit was switched on during inspiration and expiration for 5 sec. Special probe with a thin latex balloon was inserted in the lower third of the esophagus for measuring of intraesophageal pressure. The probe was connected to a PDP-1000MD manometer. For TPP measurements, the trachea was connected to the opposite outlet of a differential manometer via the outlet localized on the pneumotachograph tube prior to a pressure sensor. For measurements of intrabronchial pressure a thin probe was inserted into the lungs to the position of complete closure of the bronchus. The probe was connected to another PDP-1000MD manometer. A multichan-

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nel recorder was used for recording spirogram, pneumotachogram, TPP, and intrabronchial pressure. Three series of experiments were performed.

In the first series, spirogram, TPP, and intrabronchial pressure were recorded in live rabbits. Air flow was interrupted upon inspiration and expiration. The forces from the thorax were controlled by the change in intrabronchial pressure and by spirogram. The degree of the TPP plateau deformation was measured from the level of its appropriate position to the maximum deviation during interruption of air flow. Spirogram and intraesophageal pressure, as the analog of intrapleural pressure, and intrabronchial pressure were then recorded. In 6 animals, we measured intraesophageal and intrapleural pressure. The results of these measurements where similar. Measurements of intraesophageal pressure were preferable, since this technique preserved the integrity of pleural cavity.

In the second series, the rabbits were killed by overdose of anesthetic. After 60 min, the animal was placed in a chamber, where inspirations and expirations were imitated by changing the pressure. The system for the volume and pressure measurements was the same. The apparatuses were connected to the animal via outlets on the chamber wall and rigid tubes, which did not react to pressure changes in the chamber. First, we recorded TPP with interruption of air flow upon "inspiration" and "expiration." Then intraesophageal and/or intrapleural pressure and intrabronchial pressure were measured. The frequency and depth of artificial respiration were similar to the respiration of live rabbits.

In the third series, the lungs with the cannula and catheter were isolated and placed under the Donders bell, and the pressure in the bell was measured instead of intraesophageal and intrapleural pressure. Transpulmonary pressure was calculated as the difference between the pressure in the Donders bell and in the trachea. The air flow was interrupted as in previous series, and the pressures in the Donders bell and in the bronchus were then compared. Artificial ventilation of isolated lungs was maintained at the level of natural breathing.

RESULTS

The results of measurement of traditional parameters of respiratory mechanics (work of breathing, its fractions, and lung compliance) were published previously [1]. The minute respiratory volume was the same in all experimental series (Table 1).

Deformation of TPP plateau upon inhalation was observed in 12 out of 16 animals. It varied from 0.44 to 1.14 kPa (0.81±0.082 kPa) (Fig. 1, *a*). During exhalation, the TPP plateau deformation was pronounced only in 5 animals, amounting for 0.42-0.82 kPa (0.55±0.064 kPa) (Fig. 1, *b*). Thus, the occurrence of plateau deformation was significantly higher during inspiration than during expiration ($p<0.01$). The deformation was so great that it exceeded the amplitude of respiratory movements upon spontaneous respiration: 0.46±0.075 kPa ($p<0.01$). Nevertheless, it did not occur in all rabbits, and its degree varied from animal to animal. It was impossible to determine the contribution of anesthesia and various regulatory factors. In the present study we did not plan to estimate the incidence of paradoxes or their causes. We investigated the most pronounced manifestations of plateau deformation. There were no difficulties in determining the appropriate position of a plateau. Previously [4], it was shown that the plateau appears immediately at the moment of interruption, and its dynamic component is distracted on the TPP curve. The exponential course of the curve upon plateau formation does not result from the equalizing of pressure in different sites of the lungs [6], but is due to deformation of plateau. Various variants of plateau deformation were described elsewhere [4].

Phenomenon of plateau deformation is paradoxical by itself, since, according to the generally accepted views, TPP should not change if the lung volume does not change, for example, upon interruption of air flow. During inhalations, the plateau

TABLE 1. Amplitudes of Intraesophageal (Pleural Cavity) and Intrabronchial Pressure in Experimental Animals (*M±m*)

Parameter	Experimental series		
	first	third	second
Minute respiratory volume, liter/min	0.94±0.17	0.90±0.16	1.0±0.2
Pressure amplitude, kPa:			
pleura	0.64±0.066**	0.81±0.088*	0.77±0.150***
bronchus	0.94±0.065	0.42±0.048*	0.16±0.041*,**

Note. *$p<0.001$ compared with the first series, **$p<0.01$ compared with the second series; *$p<0.001$, **$p<0.01$, ***$p<0.02$ compared with the amplitude of intrabronchial pressure.

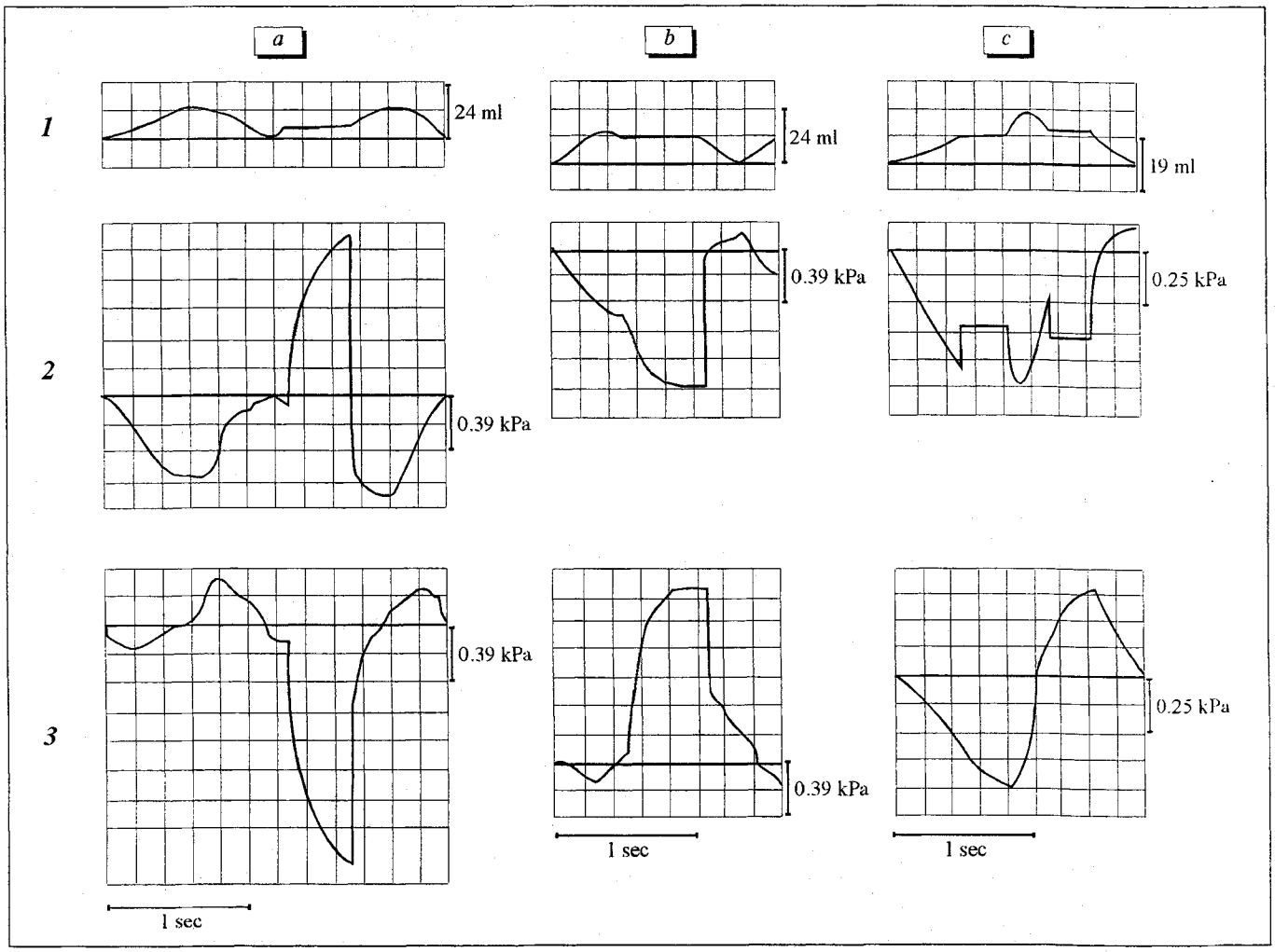

Fig. 1. Spirogram (*1*) and transpulmonary (*2*) and intrabronchial pressure curves (*3*) upon interruption of air flow during inhalation (*a*), exhalation (*b*) and in the second series of experiments (*c*); *c*) there is no deformation of the transpulmonary pressure plateau.

deformation looked like a pointed wave oriented to the positive pressure region. This indicates that intraalveolar pressure became more negative than intrapleural pressure when a rabbit tried to overcome the valve. Intrabronchial pressure became negative; its drop was a mirror reflection of the plateau and its magnitude was greater. The ratio between pressures characterizing plateau deformation and intrabronchial pressure upon interruption of air flow during exhalation was analogous. Similar results were obtained by us previously [3,4]. It was shown that there is no plateau deformation in isolated lungs. In the present study, plateau deformation was not observed in the second series of experiments (Fig. 1, *c*), when passive ventilation was carried out under conditions of preserved integrity of pleural layers. Thus, the function of pleural layers could not be the cause of TPP plateau deformation. The paradox was observed only in live animals, being regarded as a manifestation of pulmonary activity in the whole.

The amplitude of the intrabronchial pressure fluctuations was much greater than that in pleural cavity only upon spontaneous respiration (Table 1). Such a phenomenon has been already described [7], but was not analyzed as a paradox. Remembering that this phenomenon is not observed in isolated lungs, it can be classified as mechanical activity of a lung with a closed bronchus [2,4]. The present study has shown that the paradoxical ratio between the amplitudes of pressures in a bronchus and pleural cavity was not observed in experiment with isolated lungs and upon artificial ventilation of dead lungs with preserved integrity of pleural layers. Consequently, this paradox is not associated with the function of pleural layers. A decrease in the intrabronchial pressure amplitude compared with that in dead lungs with preserved pleura indicates a more effective transmission of pressure to the lung surface via the system of pleural layers. Thus, this system does not determine paradoxical phenomena in the mechanics

of respiration. Such a conclusion holds true only for pronounced paradoxical phenomena.

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Effect of Preliminary Adaptation to Transauricular Electrostimulation on the Content of Catecholamines and Met-Enkephalin in Rat Heart and Adrenals in Stress and Acute Myocardial Infarction

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Adaptation to transauricular electrostimulation decreases the content of epinephrine in the adrenal glands and norepinephrine in the heart. Immobilization stress has no appreciable effect on the content of catecholamines in the heart and adrenal glands. In animals with myocardial infarction, the content of norepinephrine in the heart decreases 2-fold, while the content of epinephrine in the adrenals decreases inconsiderably. Adaptation to transauricular electrostimulation is associated with a rise in met-enkephalin concentration. Preadaptation induces a more pronounced rise of met-enkephalin and promotes normalization of epinephrine in the adrenals, without changing the content of norepinephrine in the heart.

Key Words: *transauricular electrostimulation; stress; myocardial infarction; catecholamines; met-enkephalin*

Recent experimental data suggest that elimination of the stress component of the damage partially underlies the protective effect of transauricular electrostimulation (TES) in acute myocardial infarction [2]. This assumption is confirmed by the fact that TES can limit the stress reaction through suppression of adrenoreactivity and activation of the opioid systems. Indeed, similarly to adaptation to stress, preadaptation to TES reduces heart sensitivity to toxic concentrations of epinephrine [4] and considerably elevates the plasma content of immunoreactive β-endorphin in rats [3]. However, the role of the interplay between catecholaminergic and opioid systems in protective effect of TES is poorly understood. The effect of TES on the interaction between these systems in the adrenal glands is also little studied.

In the present study we evaluate the effect of TES on the content of catecholamines and met-enkephalin (ME) in rat heart and adrenal glands in immobilization stress and acute myocardial infarction.

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