PULMONARY ELASTIC HYSTERESIS UNDER NORMAL CONDITIONS AND IN EMPHYSEMA

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The static elastic pressure was investigated in healthy subjects, patients with emphysema, and in normal and emphysematous cadaveric lungs. Hysteresis of the elastic structure of the lungs is regarded as the result of expenditure of the work of breathing on overcoming tissue friction. The tissue friction is less in emphysematous than in normal cadaveric lungs. In healthy subjects there is no pulmonary elastic hysteresis, showing that tissue friction is overcome by the lungs themselves. In patients with pulmonary emphysema considerable pulmonary elastic hysteresis is found; this shows that even the reduced tissue friction in such patients can be only partly overcome by the lungs themselves.

Elastic hysteresis is manifested by the fact that the diagram of the static elastic pressure usually does not coincide on expiration and inspiration. It is caused by the expenditure of the work of breathing on overcoming the nonelastic resistance of the lung tissues. Elastic hysteresis, while negligible in healthy persons, is appreciably increased in emphysema [4, 5, 8]. The absence of pulmonary elastic hysteresis in healthy subjects has been explained by the ability of the smooth muscle of the lungs to overcome the tissue friction [2-4].

If this hypothesis is true, an increase in elastic hysteresis must be expected in unchanged cadaveric lungs because of the appearance of nonelastic tissue resistance under these conditions. It was also decided to compare elastic hysteresis in normal and emphysematous cadaveric lungs.

EXPERIMENTAL METHOD

The method used to study elastic hysteresis in healthy subjects and in patients with pulmonary emphysema was described previously [3, 4]. The lungs were removed en bloc with the trachea on the 1st-3rd day after death and fixed in a Donders' bell. A pneumotachographic tube, automatic airflow interrupter, and water spirometer were connected to the trachea. The respiratory fluctuations of intrapleural pressure were simulated by means of rubber bellows. The transpulmonary pressure was determined by a differential manometer. The pressure drop in the pneumotachographic tube was measured by another manometer. Mechanical movements of the spirograph drum were converted into electric waves by means of a linear variable resistor. The transpulmonary pressure, pneumotachogram, and lung volume were recorded simultaneously on a 4-channel electrocardiograph. The airflow was interrupted hermetically for 0.4-0.7 sec to determine the elastic pressure under static conditions. The "respiratory loop" (the loop of total pulmonary hysteresis) was plotted from pressure and volume curves. From the values of the static elastic pressure a loop of pulmonary elastic hysteresis was drawn inside the respiratory loop. The magnitude of the total and elastic hysteresis was determined from the work of "breathing" per liter of ventilation, calculated from the corresponding areas. The cadaveric lungs also were studied morphologically.

Altogether eight lungs from persons dying accidentally and five lungs from persons dying from pulmonary emphysema, in whom the elastic hysteresis was investigated during life also, were investigated.

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TABLE 1. Total and Elastic Hysteresis of the Lungs in Healthy and Emphysematous Subjects and in Normal and Emphysematous Cadaveric Lungs ($M \pm \sigma$)

Object tested	Age (in years)	MVR (in liters/min)	Hysteresis (in kg·m/min/liter)	
			total	elastic
Lungs of healthy subjects (n = 10)	45	7,0	0,034±0,013	—0,004±0,011
Normal cadaveric lungs (n = 8)	48	2,1	0,149±0,047	0,100±0,042
Lungs of patients with emphysema	50	2,0†	0,114±0,030	0,035±0,009
Cadaveric emphysematous lungs (n = 5)	50	2,7	0,108±0,042	0,067±0,017

*Value of MVR corresponds to rate of airflow in respiratory cycle during determination of total and elastic hysteresis (in kg·m/min/liter).

†MVR, mean value 2 liters/min, obtained at a mean respiration rate of 4/min and a mean depth of respiration of 0.5 liter. Work of breathing measured in 2-3 respiratory cycles.

Since the mechanical properties of the lungs could vary after death, the pulmonary elastic hysteresis also was measured in 10 cats during life, immediately after death, and 1-3 days later. The experiments were carried out under intravenous urethane anesthesia.

EXPERIMENTAL RESULTS AND DISCUSSION

The results of the measurements are given in Table 1. During the investigation of the healthy subjects and patients with emphysema it was impossible to obtain equal values of the minute volume of respiration (MVR). However, the lower MVR in the patients with emphysema was accounted for entirely by the reduced depth of breathing. This indicates that their elastic hysteresis was sharply increased.

The MVR of the normal and emphysematous cadaveric lungs was the same as in the patients with emphysema. It evidently could not be increased because forced "expiration" led to operation of the valve mechanism.

Elastic hysteresis in the normal cadaveric lungs was much greater than in healthy subjects despite the high MVR. This is in agreement with data in the literature [9] and it is evidently explained by inactivation of the smooth muscle of the lungs.

The elastic hysteresis in the cadaveric emphysematous lungs also was high, but significantly ($P < 0.001$) less than in normal cadaveric lungs. The decrease in tissue friction during emphysema was probably connected with atrophy of the lung tissue and with the large volume of the unventilated parts of the lungs as a result of a disturbance of bronchial patency.

The elastic hysteresis in the patients with emphysema was on the average only half that in the cadaveric emphysematous lungs. Even the reduced tissue friction in the patients with emphysema was thus overcome by only 50%.

The experiments showed that the pulmonary elastic hysteresis increased immediately after the animal's death and then remained unchanged for 1-3 days. This showed that the length of time after death, within these limits, did not affect the magnitude of the hysteresis in the cadaveric lungs.

The results of these investigations support the views of other workers [6, 7] on the importance of tissue friction in the mechanics of respiration. The writer's interpretation of pulmonary elastic hysteresis and the function of the smooth muscle of the lungs under normal conditions and in emphysema are in harmony with the morphological data [1] on fatty degeneration of the smooth-muscle fibers of the lungs in emphysema.

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