PRESSURE IN THE OCCLUDED BRONCHUS IN EXPERIMENTAL PULMONARY EMPHYSEMA

F. F. Tetenev

UDC 616.24-007.63-092.9-07:616.233-008.718-073.178

The work of respiration and its components and the dynamic elasticity of the lungs were investigated in 8 healthy rabbits and in 15 animals with emphysema induced by intravenous injection of lycopodium spores. The pressure in the occluded bronchus and the intrapleural and intraesophageal pressures were measured relative to atmospheric. In healthy rabbits the pressure in the occluded bronchus fluctuated with the mean amplitude twice as great as that of the intrapleural pressure. This contradicts the existing view of the passive role of the lungs in the mechanics of respiration. The existence of a source of mechanical energy in the lung structures is postulated.

KEY WORDS: pressure; occluded bronchus; pulmonary emphysema.

Evidence of the existence of contractility of the lungs can be found in the literature [1, 2, 4, 6-8, 14]. The ability of the lungs to contract actively was studied in this investigation.

EXPERIMENTAL METHOD

Experiments were carried out on 8 healthy rabbits (group 1) and 15 rabbits in which emphysema was induced by Mavrin's method [3] by intravenous injection of lycopodium spores. Ten animals were studied 4.5 months (group 2) and 5 animals 9 months (group 3) after the beginning of the experiment. The weight of the healthy and emphysematous rabbits varied from 2.5 to 3 kg and the mean weight was the same for all three groups.

Tracheostomy was performed under intravenous thiopental anesthesia. The trachea was connected to a minature spirograph. The transpulmonary pressure (the difference between the pressure in the trachea and esophagous) was measured by a differential electromanometer. Respiratory fluctuation of transpulmonary pressure and the spirogram were recorded simultaneously on a four-channel electrocardiograph, using a dc amplifier. The work of respiration, its various fractions, and the dynamic elasticity were calculated from the respiratory loop.

A catheter 2 mm in diameter was passed through the tracheal cannula until it occluded the bronchus. The spirogram, the pressure in the occluded bronchus, and the intraesophageal and intrapleural pressures relative to atmospheric were recorded simultaneously by identical electromanometers. The position of the intraesophageal catheter, of the catheter occluding the bronchus, and of the intrapleural needle was verified roentgenologically. After the investigation the lungs were removed together with the trachea and catheter and their volume determined. In every case the catheter occluded the bronchus in the lower lobe of the right lung. The intrapleural pressure was measured at the same place. Pieces were taken from different parts of the lungs for morphological examination, after which the lungs were fixed in a Donders' bell jar. The respiratory fluctuation of intrathoracic pressure were simulated by means of bellows and the spirogram, the pressure in the Donder's bell jar and the pressure in the occluded bronchus were recorded simultaneously.

Department of Propaedeutics of Internal Diseases, Central Research Laboratory, Tomsk Medical Institute. Department of Pathological Anatomy, Patrice Lumumba Peoples' Friendship University, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR D. D. Yablokov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 81, No. 1, pp. 30-32, January, 1976. Original article submitted April 1, 1975.

TABLE 1. Indices of Mechanics of Respiration in Healthy Rabbits and in Emphysematous Rabbits 4.5 and 9 months after Beginning of Experiment (M ± m)

Group of rabbits	MVR (in liters/min)	Total work		Elastic work		Viscous work (in g·cm/min)			Dynamic stretching (in ml/cm water)	Volume of lungs (in ml)
		g·cm/min	g·cm/liter	g·cm/min	percent of total	inspiration	expiration	total viscous work		
1st ($n=8$)	2,25 ± 0,204	0,76 ± 0,163	0,324±0,061	0,70 ± 0,159	94,0 ± 3,49	0,03 ± 0,039	0,08 ± 0,060	0,11 ± 0,089	10,85 ± 1,208	22,0 ± 0,840
2nd ($n=10$)	2,01 ± 0,302	1,72 ± 0,275	0,87 ± 0,078	1,14 ± 0,195	68,4 ± 3,41	0,52 ± 0,104	0,85 ± 0,114	1,37 ± 0,228	3,45 ± 0,513	41,0 ± 5,385
3rd ($n=5$)	1,66 ± 0,136	1,47 ± 0,201	0,908 ± 0,89	0,58 ± 0,092	39,5 ± 1,92	0,76 ± 0,103	0,59 ± 0,099	1,35 ± 0,201	3,52 ± 0,611	50,6 ± 3,762
P_{1-2}	—	<0,01	<0,001	<0,2	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001
P_{1-3}	<0,02	<0,02	<0,001	—	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001
P_{2-3}	—	—	—	<0,02	<0,001	<0,1	<0,2	—	—	<0,1

TABLE 2. Amplitude of Respiratory Fluctuations of Pressure in the Occluded Bronchus and the Intrapleural and Intraesophageal Pressures in Rabbits During Life and in the Isolated Lungs (M ± m)

Groups of rabbits	During life							Isolated lungs		
	MVR (in liters/min)	amplitude of pressure (in cm water)				P		MVR (in liters/min)	amplitude of pressure (in cm water)	
		bronchus	pleura	esophagus	bronchus—pleura difference	bronchus—pleura	pleura—esophagus		bronchus	Donders' bell jar
1st ($n=6$)	1,6 ± 0,254	40,9 ± 6,239	19,6 ± 3,798	19,4±4,170	21,3 ± 3,64	<0,01	—	0,88 ± 0,263	16,3±1,419	53,1 ± 5,152
2nd ($n=4$)	2,9 ± 0,627	30,2 ± 1,512	21,7 ± 3,764	20,5±3,732	8,5 ± 1,598	<0,05	—	0,59 ± 0,236	10,0±4,377	55,6 ± 19,65
3rd ($n=5$)	2,5 ± 0,453	29,3 ± 4,041	26,2 ± 1,854	22,1±5,093	3,1 ± 2,62	—	<0,2	—	—	—
P_{1-2}	<0,1	<0,1	—	—	<0,001	—	—	—	—	—
P_{1-3}	<0,1	<0,1	<0,1	—	<0,001	—	—	—	—	—
P_{2-3}	—	—	—	—	<0,1	—	—	—	—	—

EXPERIMENTAL RESULTS AND DISCUSSION

Various degrees of atelectasis and emphysema were found in the lungs of the experimental animals. The emphysema was more marked in the later stages of the experiments (group 3). Parallel with the emphysema, an increase in the degree of severity of precapillary pulmonary hypertension was observed (hypertrophy, vacuolation of the smooth muscles of arteries of the muscular type, and overstretching of the longitudinal muscular layers in the intima of the vessels). When emphysema was severe, the elastic fibers in the framework of the respiratory bronchioles and alveoli were replaced by collagen. Various degrees of lymphostasis and lymphoplethora were established.

The volume of the lungs was increased in the animals of group 2 and still more in those of group 3 (Table 1). The total and, in particular, the specific work were significantly increased in both the second and third groups on account of the viscous work. The elastic work as a percentage of the total work fell considerably in both experimental groups, indicating a predominant increase in the nonelastic resistance; this increase was greater in inspiration, mainly in the animals of group 3. The elasticity of the lungs was considerably and, on average, equally reduced. The increase in the nonelastic resistance of the lungs can be explained by an increase in the bronchial resistance. The decrease in elasticity of the lungs can be explained by an increase in pressure in the system of the pulmonary artery, lymphostasis, fibrous changes in the lungs, and a shift of the respiratory volume toward the inspiratory reserve on account of an increase in the residual lung volume. On the whole, therefore, the mechanical properties of the lungs in experimental emphysema corresponded to those in emphysema in man [5].

Pressure in the occluded bronchus in the healthy rabbits varied with an amplitude 2.5 times greater than that of the intrapleural and intraesophageal pressure, and on average twice as great as that for the whole group of healthy animals (Table 2). These results agree with data in the literature [13], although there is no explanation there of this fact. This phenomenon cannot be explained on the basis of the generally accepted concept of the passive role of the lungs in the mechanics of respiration. It can be postulated that the greater amplitude of the respiratory fluctuations of pressure in the occluded bronchus occurred because of an additional source of mechanical energy in the structures of the lungs.

The possibility of obtaining a more negative pressure in the occluded bronchus than in the pleural cavity, as a result of the phenomenon of "alveolar interdependence," has been examined theoretically and by the use of models [9-12]. In the opinion of the authors concerned, this phenomenon can be observed whereever ventilation of the alveoli is uneven, especially under pathological conditions.

The amplitude of the pressure fluctuation in the occluded bronchus in the rabbits of groups 2 and 3 was smaller on average, but in the pleural cavity it was much greater than in the healthy animals. Consequently, the predominance of amplitude of the respiratory fluctuations of pressure in the occluded bronchus over that of the intrapleural pressure was smaller on average in the rabbits of groups 2 and 3 ($P < 0.001$), although the emphysema in these animals, especially in group 3, was considerable and led to a disturbance of the uniformity of ventilation. The predominance of amplitude of the respiratory pressure fluctuation in the occluded bronchus cannot therefore be explained by the interdependence effect, whereas the assumption of an additional source of mechanical energy has a logical continuation: Under pathological conditions, in experimental emphysema, the activity of this hypothetical source is reduced.

In the isolated lungs of the healthy and emphysematous animals fluctuations of pressure in the occluded bronchus during stimulation of respiratory movements was several times smaller in amplitude than in the Donders' bell jar. This can be explained by the expenditure of a considerable part of the mechanical energy applied to the surface of the lungs in the bell jar in overcoming the total nonelastic tissue resistance. It is logical to suggest that the hypothetical source of mechanical energy which, during life, overcomes the nonelastic tissue resistance [6], and which, when the bronchus is occluded, exerts an effort aimed at overcoming the obstruction, does not function in the isolated lungs.

LITERATURE CITED

1. V. I. But, Fiziol. Zh. SSSR, No. 7, 853 (1969).
2. A. I. Verpintseva, in: Problems in Curative and Preventive Medicine [in Russian], Leningrad (1963), p. 39.
3. V. K. Mavrin, Arkh. Pat., No. 3, 42 (1970).
4. I. V. Makarov, Vopr. Okhr. Mat., No. 8, 87 (1967).
5. F. F. Tetenev, Ter. Arkh., No. 11, 29 (1968).
6. F. F. Tetenev, Byull. Éksperim. Biol. Med., No. 6, 21 (1974).
7. R. Hughes et al., J. Physiol. (London), 146, 85 (1959).
8. A. Luisada, Beitr. Klin. Tuberk., 73, 657 (1930).
9. P. Macklem et al., Am. J. Med., 57, 371 (1974).
10. J. Mead et al., J. Appl. Physiol., 28, 596 (1970).
11. H. Menkes et al., J. Appl. Physiol., 32, 675 (1972).
12. H. Menkes et al., J. Appl. Physiol., 32, 681 (1972).
13. W. Webb et al., Ann. Surg., 153, 650 (1961).
14. C. Williams, cited by J. C. Widdicombe, Physiol. Rev., 43, 1 (1963).