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SURFACE ACTIVITY OF SURFACTANT IN EXPERIMENTAL COMPRESSION ATELECTASIS

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Among investigations to study the state of the lung surfactant (LS) in atelectasis [1-4, 9, 10] there have been none to study the dynamics of changes in LS at different periods of atelectasis. It is not clear how the qualitative composition of the phospholipids of LS, on which its surface-active properties depend, changes under these circumstances, or whether aeration of the lung tissue can be restored after removal of the cause of the atelectasis.

In the investigation described below a combined study was undertaken of the time course of changes in the surface activity of LS at different stages of compression atelectasis.

EXPERIMENTAL METHODS

Material for the investigation consisted of lungs of 35 guinea pigs of both sexes and weighing 150-250 g, in which compression atelectasis was modeled by the creation of hydrothorax (injection of 15-20 ml of sterile isotonic sodium chloride solution into the pleural cavity). The animals were decapitated 30 and 60 min, and 3, 12, and 24 h after injection of the solution. In each of the five groups corresponding to the above-mentioned times, six animals were killed, and five animals served as the control group. To confirm the presence of atelectasis of the lung tissue, paraffin sections of lung tissue were examined histologically, using survey stains. To investigate LS, 10% saline extracts were prepared from the lungs, from which

TABLE 1. Surface Tension of LS, Total Lipid and Phospholipid Content and Qualitative Composition of Phospholipids during Experimental Compression Atelectasis

Duration of atelectasis	ST_{min}, mN/m	CI	Content of total lipids, g/liter	Content of phospholipids		
				total, mmole/liter	phosphatidylcholine	phosphatidylethanolamine
					%	
Control	14,2±1,0	1,1±0,1	0,92±0,02	0,078±0,010	37,3±2,1	19,6±2,2
30 min	27,1±2,9	0,5±0,05	0,99±0,03	0,027±0,007	30,8±6,8	43,7±6,6
60 min	19,8±2,8	0,5±0,11	0,99±0,03	0,044±0,018	None	61,7±6,0
3 h	22,7±1,5	0,6±0,08	0,98±0,04	0,048±0,015	32,8±9,0	49,4±9,4
12 h	31,8±3,7	0,5±0,06	1,06±0,01	0,035±0,006	47,2±14,3	39,1±7,4
24 h	29,9±4,3	0,5±0,05	0,98±0,03	0,034±0,015	39,2±10,9	61,8±3,5

Legend. Results significant compared with control ($P < 0.05$) except phosphatidylcholine content and value of ST_{min} for atelectasis with a duration of 60 min ($P > 0.05$).

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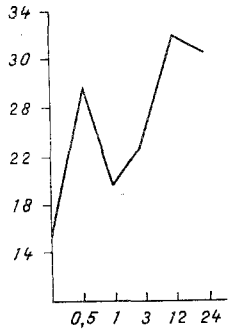

Fig. 1

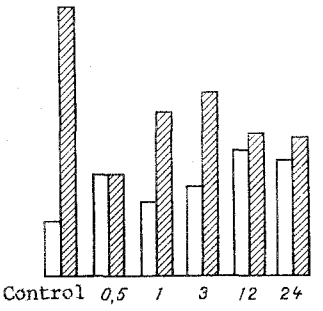

Fig. 2

Fig. 1. Time course of changes in ST_{min} during compression atelectasis. Abscissa, time (in h), ordinate, value of ST_{min} (in mN/m).

Fig. 2. Dependence of phospholipid level of LS on ST_{min}. Abscissa, time (in h). Unshaded columns – ST_{min}; shaded – phosphlipid content.

the surface-active fraction was isolated by differential centrifugation [5]. The state of the surface activity of LS was studied by physicochemical methods (determination of surface tension of the surface-active fraction of the lung extracts from the animals on Wilhelmy scales). Total lipids of the surface-active fraction were determined by oxidation with perchromic acid [6]. Phospholipids were extracted by the method in [7]. The isolated lipids [8] were fractionated by thin-layer chromatography on Silufol UV-254 plates (Czechoslovakia).

EXPERIMENTAL RESULTS

At different times of compression atelectasis surface activity of LS was inhibited, as shown by the high values of minimal surface tension (ST_{min}) and also the low level of phospholipids in LS (Table 1). At different times of atelectasis inhibition of the surface-active properties of LS differed in magnitude: whereas 30 min after the creation of hydrothorax ST_{min} reached 27.1 ± 2.9 mN/m, after 60 min it fell to 19.8 ± 2.8 mN/m, and remained at this level for a short time longer (after 3 h it was 22.7 ± 1.5 mN/m), after which ST_{min} rose again so that, after 12 h, it was 31.8 ± 3.7 mN/m. This character of the change in surface activity of LS was evidently connected with the fact that after a period of increased secretion of LS by type II alveolocytes, which was observed 60 min and 3 h after the creation of hydrothorax, the LS reserves became exhausted, and this led to an increase in ST_{min}. The time course of changes in ST_{min} at different times of compression atelectasis is illustrated in Fig. 1.

When the results of the biochemical investigation of LS are compared, the very small fluctuation in the content of total lipids at the different times of the experiment will be noted. The difference between the greatest (1.06 ± 0.01 g/liter) and the least 0.98 ± 0.03 g/liter) values on this parameter is only 0.08 g/liter, although on the whole in compression atelectasis the level of total lipids was higher than in the control. This fact confirms the views of other workers that the total lipid level has no significant effect on the surface activity of LS.

Analysis of the phospholipid contentin LS and the value of ST_{min} shows that the highest values of ST_{min} correspond to a low phospholipid content and vice versa, i.e., the phospholipid content is inversely proportional to ST_{min} (Fig. 2). On the whole, however, during compression atelectasis, a significant decrease in the phospholipid level of LS is observed compared with the control

The results of thin-layer chromatography of the phospholipids of LS show that changes in the surface-active properties of LS in compression atelectasis are not accompanied by a disturbance of its qualitative composition. The absence of phosphatidylcholine in the composition of the phospholipids of LS 60 min after induction of hydrothorax may be due to exhaustion of the phosphatidylcholine reserves during this period in the course of intensified synthesis and secretion of LS, aimed at restoring surface tension. The increase in the content of phosphatidylethanolamine (61.7 ± 6.0%), which is known to be a precursor of phosphatidylcholine, at this time can be regarded from the same point of view. Increased LS syn-

thesis leads subsequently to an increase in the content of phosphatidylcholine, whose level after 12 h was higher than in the control. This fact is evidence that the qualitative composition of the phospholipids of LS is preserved in compression atelectasis. However, since the cause of the atelectasis had not been removed, the surface tension level remained high. In compression atelectasis the decrease in surface activity of LS is connected with a disturbance of secretion of LS into the lumen of the alveoli, and not with disturbance of its synthesis, as was confirmed by the low level of phospholipids of LS, in whose composition a high content of phosphatidylcholine still remained.

In experimental compression atelectasis inhibition of the surface activity of LS is thus observed, and is connected with disturbance of LS secretion into the lumen of the compressed alveoli. The qualitative composition of the phospholipids of LS is preserved under these circumstances, and this may play an important role in the re-expansion and reaeration of the lung tissue, especially in the early stages of development of atelectasis, if the cause of the atelectasis has been removed at this period.

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