EFFECTS OF VAGOTOMY AND INCREASED BLOOD PRESSURE ON THE INCIDENCE OF DECOMPRESSION-INDUCED PULMONARY HEMORRHAGE

(Research Note)

H. S. FANG and C. M. TANG

Dept. of Physiology, College of Medicine, National Taiwan University and Aerospace Bio-Medical Division, CAF General Hospital, Taipei, Taiwan

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Abstract. Sixty eight male rats were divided into four groups. They were rapidly decompressed from one atmospheric pressure (760 mmHg) to an ambient pressure of 30 mmHg in 0.4 s. It was found that about half of the control animals (47%) revealed mild pulmonary hemorrhage, while all of the cervical vagotomized rats (100%) showed mild to severe pulmonary hemorrhage. Besides, 16 of 17 epinephrine injected rats (94%) and 15 of 17 carotid arteries occluded rats (88%) also exhibited decompression-induced pulmonary hemorrhage. The differences between control and experimental groups were statistically significant. The results suggest that during rapid decompression, the overexpansion of the alveoli accompanied by an increased pressure in the pulmonary vessels may be responsible for the production of pulmonary hemorrhage.

1. Introduction

Because of the delicate nature of the gas-containing pulmonary tissue, the lungs are the most vulnerable part of the body during rapid decompression. It is known that the ability of the lung to withstand a sudden reduction in the environmental pressure is influenced by the volume of the air in the lungs and the rate and range of decompression (Bancroft, 1961; Fang, 1966; Hitchcock, 1953). The purpose of the present study was to ascertain whether cervical vagotomy, intravenous injection of epinephrine and occlusion of carotid arteries would influence the incidence of pulmonary hemorrhage following rapid decompression.

2. Methods

Sixty eight adult inbred rats (Long Evans Strain) were used for these experiments. They were anesthetized with 1.6 g per kg of urethane given intraperitoneally. In each experiment, four rats, i.e., (a) one control, (b) one bilateral cervical vagotomized, (c) one epinephrine injected (12 μ per kg, i.v.), and (d) one carotid arteries occluded animal, were decompressed together. Because of the short duration of epinephrine effect, it was necessary to measure the blood pressure of epinephrine injected rats. The blood pressure was measured from the carotid artery with a Stathem model PR23-15D-300 transducer on a Grass polygraph. Rapid decompression was made when the epinephrine-induced transient hypertension just reached maximal level.

Rapid decompression was accomplished by the rupture of a sheet of X-ray film separating a small animal chamber at one atmospheric pressure and a large tank at

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very low pressure. The animal chamber was kept open to room air until immediately before the rapid decompression. The rate of decompression was measured by means of a Staham model PM6±15–350 pressure transducer on the same Grass polygraph. The range of decompression was performed from initial pressure of 760 mmHg to final pressure of 30 mmHg in 0.4 s. In order to avoid hypoxia acting as a complicating factor, all experimental animals were recompressed immediately following decompression. The lungs were then carefully examined for gross evidence of hemorrhage within a few minutes thereafter.

3. Results

The frequency of occurrence and the severity of the decompression-induced pulmonary hemorrhage in 68 rats are given in Table I. It will be noted that 8 of 17 control animals showed mild pulmonary hemorrhage. However, in the cervical vagotomized animals, lung hemorrhages varying from mild to severe were revealed without exception. These involved both the right and the left lungs except in one case in which the hemorrhage was observed in the right lung only. Epinephrine administered to the

TABLE I

Occurrence of pulmonary hemorrhage in vagotomized, epinephrine injected and carotid arteries occluded male rats following a single rapid decompression from one atmospheric pressure to an ambient pressure of 30 mmHg in 0.4 s.

Control			Cervical vagotomy			Epinephrine injection			Occlusion of carotid arteries		
Body wt g	Pulmonary hemorrhage		Body wt g	Pulmonary hemorrhage		Body wt g	Pulmonary hemorrhage		Body wt g	Pulmonary hemorrhage	
	Rt lung	Lt lung		Rt lung	Lt lung		Rt lung	Lt lung		Rt lung	Lt lung
346	0	0	330	++	++	430	++	++	370	0	0
380	0	0	360	++	++	340	+	++	318	++	0
370	+	0	350	+	+	340	++	++	345	++	+
380	0	0	400	++	++	300	+	+	335	++	++
360	+	0	400	++	++	335	++	+	375	++	+
245	+	0	350	++	+	370	++	++	373	+	0
280	0	0	335	+	0	300	++	++	337	++	0
340	+	+	330	+	+	390	++	++	417	0	0
270	+	0	270	++	++	330	+	0	318	+	+
260	0	+	280	+	+	385	++	++	355	+	++
270	0	0	280	+	+	330	+	++	360	+	+
260	+	+	300	++	++	320	++	++	363	+	0
250	+	0	260	++	+	290	0	0	350	++	++
205	0	0	255	++	+	345	++	++	313	++	+
195	0	0	200	+	+	290	+	+	280	++	++
208	0	0	211	++	++	290	++	++	338	++	++
212	0	0	220	++	+	285	++	++	363	++	+

0: No hemorrhage.
+: Mild hemorrhage.
++: Severe hemorrhage.

anesthetized rats intravenously produced an average increase in blood pressure of 54 mmHg (from 82 to 136 mmHg). Such rats showed an increased frequency of pulmonary hemorrhage following decompression. This is indicated by the fact that 16 of 17 rats exhibited hemorrhage of the lungs on autopsy, among which hemorrhage was mild in 3 and severe in 13. With both carotid arteries occluded, the occurrence of pulmonary hemorrhage was also increased. 15 of 17 such rats had mild to severe pulmonary hemorrhage. According to the method of the chi-square test, the differences between control and different experimental groups were statistically significant.

4. Discussion

In this investigation, it was shown that cervical vagotomy caused an increased frequency of pulmonary hemorrhage following rapid decompression. It is known that if both vagus nerves in the neck are cut, a change in respiratory rhythm takes place immediately; the movements become less frequent, but are increased in amplitude resulting in an increased amount of air in the lungs. With such an increased amount of air, there will be overexpansion of the lungs during rapid decompression, by which the alveoli will become so much overstretched that rupture of the alveolar structures with resultant hemorrhage may occur. On the other hand, another explanation may be offered. When both vagi are divided, the blood pressure rises. As a result, the pulmonary arterial pressure is also increased. It seems reasonable to assume that, during rapid decompression, the elevation of pulmonary pressure will cause distention of the vessels, facilitating the occurrence of pulmonary hemorrhage. Epinephrine injection or occlusion of common carotid arteries, either one of which will be accompanied by an increase in blood pressure, may facilitate the decompression-induced pulmonary hemorrhage in the same sense.

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