

PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

RESPIRATORY ARRHYTHMIA IN MAN UNDER CONDITIONS OF HYPOXEMIC HYPOXIA

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L. A. Mirzabaeva and V. N. Alifanov

Clinical Physiology Laboratory, Institute of Normal and Pathologic Physiology,
Academy of Sciences of the USSR

(Presented by Academician V. V. Parin)

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Most authors consider that a decrease in the partial pressure of oxygen in the arterial blood produces, via the chemoreceptors of the sino-carotid and aortic zone is a reflex tachycardia caused by a decrease in the tonus of the vagus nerve nuclei [4-6, 8, 11-16, and others].

The tonus of the vagus nerve nuclei may be judged not only by the number of cardiac contractions but also by respiratory arrhythmia which produces a shortening of the cardiac cycle during inspiration and a lengthening of the cycle at the end of expiration and during the respiratory pause.

The literature holds comparatively scant and conflicting data concerning the effect of hypoxia on the respiratory arrhythmia. Some investigators [3] find a more pronounced arrhythmia during hypoxia, while others note that it lessens or disappears under these conditions [7, 14].

Certain authors consider the appearance of respiratory arrhythmia in hypoxic conditions an advantageous phenomenon attesting to the high resistance of the organism [7], but this fact has not been confirmed in studies on humans [2].

We have attempted to study the dynamics of the respiratory arrhythmia during hypoxia, using more modern methods of investigation.

METHODS

Twenty studies on healthy men aged 18-25 years were performed. Hypoxia was induced by "elevating" the subjects in a pressure chamber to an "altitude" of 5000 meters where they remained for 30 min., or by breathing of a gas mixture containing 11% oxygen and 89% nitrogen for half an hour under normal atmospheric pressure.

The cardiac and respiratory rhythms were recorded.

During the investigation the electrocardiogram was recorded on Standard Lead II with a "Biofizpribor" make cardiocyclograph [1]. The principle of this machine is such that increase in the R wave of the electrocardiogram controls the light of a cathode oscillograph, transmitting it to a new "line".

For quantitative expression of the degree of respiratory arrhythmia the arrhythmia coefficient was calculated consisting of the percent ratio of the sum of the difference in length of each preceding and following cardiac cycle to the summed lengths of a certain number (not less than 50).

RESULTS

The data presented in the table show that under conditions of hypoxia on a background of intensified tachycardia and unchanged respiratory rate there occurs a regular decrease in respiratory arrhythmia. Similar data were also obtained with respiration of hypoxemic gas mixtures.

Changes in Heart Rate and Respiration during Hypoxemic Hypoxia

Index	Original value	After staying at "height" of 5000 m for 3 min			After staying at "height" of 5000 m for 20 min			After "descent"		
		Average value	Difference from start	True difference in %	Average value	Difference from start	True difference in %	Average value	Difference from start	True difference in %
Average number of cardiac systoles per minute	64.2	85.6	+21.4	99.9	92	+28.2	99.9	63.4	-0.8	Less than 90
Respiratory frequency per minute	13.2	13.6	+ 0.4	Less than 90	14.0	+ 0.8	Less than 90	12.8	-0.4	Less than 90
Coefficient of respiratory arrhythmia	11.3	4.7	-6.6	98	1.7	-9.6	95	11.3	0	0

The results obtained may be explained as the consequence of decrease in vagal tone under hypoxic conditions.

Recently it was discovered in experiments on dogs [9, 10] that change in the number of cardiac contractions in hypoxia depends on two opposite reactions. The chemoreceptors of the sino-carotid zone produce the first reflex inhibition of the sympathetic effect on the cardiovascular system with a slowing of the pulse and increase in peripheral vascular resistance. A reflex increase in respiration under these conditions is the opposite action.

Our data confirm that in man the indicated reactions to hypoxic conditions occur differently. During the hypoxic period we could not discover signs of a decrease in sympathetic tone and an increase in vagal effect. On the contrary, in hypoxia tachycardia occurred and the respiratory arrhythmia was almost unchanged and undiminished with small changes in respiratory rate. We suggest that in man hypoxia first produces a reflex inhibition of vagus tone. This is also confirmed by the fact that in the transition to breathing oxygen respiratory arrhythmia again appears, indicating the restoration of vagal tone in the cardiac branches of the vagus nerves.

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