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Effects of Anoxemia on Intrapleural Pressure in Dogs.*

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(Introduced by C. D. Leake.)

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The theory has been advanced that changes in intrapleural pressure following alterations in bronchial caliber are mediated by carbon dioxide.¹ Thus in broncho-constriction, a gradual increase in carbon dioxide tension in the alveoli and blood seems to stimulate the inspiratory mechanism so that a relatively increased average thoracic girth results, and intrapleural pressure becomes more negative. If, however, stimulation of the respiratory center, by the direct administration of relatively high concentrations of carbon dioxide, becomes great enough to initiate active expiratory effort, then the intrapleural pressure becomes more positive than usual during expiration.² Since oxygen want in itself is, as was shown by Gasser and Loevenhart,³ initially stimulating to the respiratory center, we thought it might be interesting to determine directly whether or not anoxemia would alter intrapleural pressure in the same way as increased carbon dioxide tensions.

Anoxemia was produced by rebreathing into a spirometer through a tracheal cannula. Carbon dioxide was absorbed by soda lime placed in the circuit. Gas analyses were made by means of a Henderson-Haldane-Bailey⁴ apparatus in order to determine the degree of anoxemia produced and also to determine whether or not carbon dioxide was adequately absorbed. Blood gas analyses were also made in certain experiments. Details of our technique for estimating intrapleural pressure have been previously described.⁵

Anoxemia produced by this method (18 experiments) was found uniformly to result in an initial gradual decrease in intrapleural

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¹ Prinzmetal, M., Brill, S., and Leake, C. D., *PROC. SOC. EXP. BIOL. AND MED.*, 1931, **28**, 836.

² Prinzmetal, M., Brill, S., and Leake, C. D., *PROC. SOC. EXP. BIOL. AND MED.*, 1931, **28**, 832.

³ Gasser, H. S., and Loevenhart, A. S., *J. Pharmacol. Exp. Therap.*, 1914, **5**, 239.

⁴ Bailey, C. V., *J. Lab. Clin. Med.*, 1921, **1**, 657.

⁵ Brill, S., and Leake, C. D., *PROC. SOC. EXP. BIOL. AND MED.*, 1930, **27**, 518.

pressure accompanied by an increased frequency and amplitude of respiration, and by a mean increase in thoracic girth. This primary effect corresponds to Loevenhart's initial stimulating action of oxygen want on the respiratory center, and seems to be exerted chiefly on the inspiratory mechanism.

TABLE I.
Effect of Anoxemia on Intrapleural Pressure and Related Functions in a Dog of 12 kg. Anesthetized with 625 mg. of Sodium Amytal.

	Intrapleural Pressure in Cm. Water	Thoracic Girth in Cm.	Respiratory Rate per Minute	O ₂ Tension mm. Mercury
Normal	-2.0 to -5.0	46.8 to 46.88	39	156
1 minute	-2.0 " -6.0	46.9 " 47.0	42	105
2 minutes	-1.0 " -8.0	47.1 " 47.2	46	75
2½ minutes	+2.0 " -6.0	47.0 " 47.1	23	53

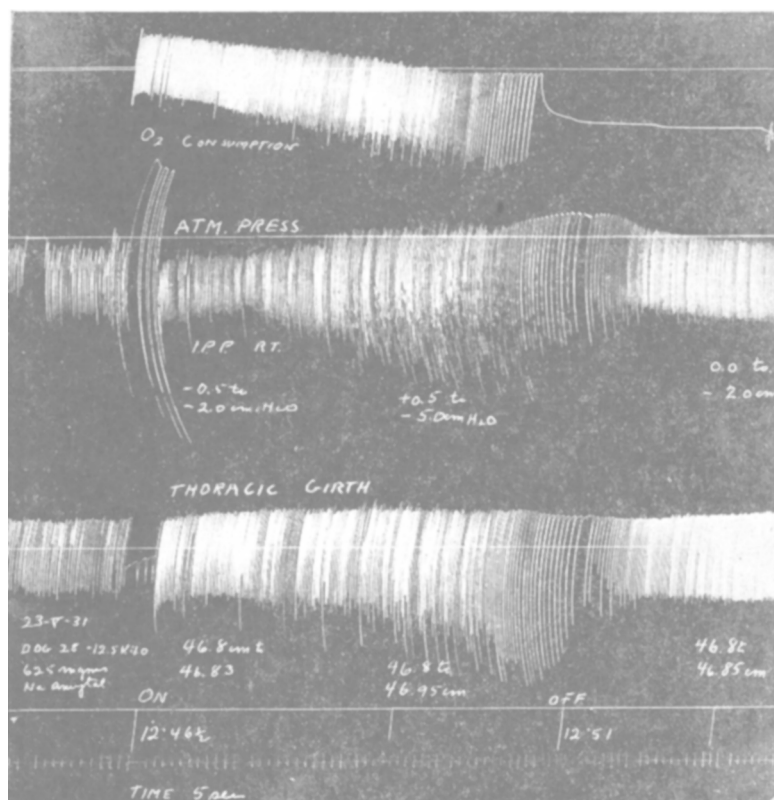


FIG. 1.

Typical kymographic record of an experiment showing the effects on intrapleural pressure (IPP) and related functions of gradually induced anoxemia, produced by rebreathing into a spirometer with carbon dioxide absorption.

The duration of anoxemia was seldom more than 4 minutes. Analysis of respiratory air showed that within this period carbon dioxide was absorbed and that oxygen tension (corrected) was progressively reduced to between 52 and 90 mm. of mercury. Blood gas analysis indicated marked anoxemia. After about 4 minutes of this gradually induced anoxemia a secondary increase in intrapleural pressure was usually observed, accompanied by a mean decrease in thoracic girth and marked slowing of respiration. This corresponds to Loevenhart's secondary depression of the respiratory center as a result of continued oxygen want. It is to be noted that anoxemia like excess carbon dioxide tension seems first to stimulate the inspiratory mechanism and then later that concerned with expiration.

The initial increase of thoracic girth with more negative intrapleural pressure under the influence of mild anoxemia offers an explanation of the mechanism by which emphysema develops during continued residence at high altitudes.

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Phenomenon of Local Skin Reactivity to Serum Precipitates.

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Phenomenon of local skin reactivity to serum precipitates is elicited as follows:

The abdominal skin of a rabbit is injected with 0.25 cc. of undiluted *B. typhosus* "agar washings" filtrate.¹ Twenty-four hours later the rabbit receives a single intravenous injection of a suspension of serum precipitate prepared in the manner about to be described. Four to 5 hours after the intravenous injection there appears at the prepared skin site a severe hemorrhagic and necrotic lesion characteristic of the phenomenon of local skin reactivity to bacterial filtrates.²

The serum precipitates with which the phenomenon could be consistently reproduced were obtained from the following mixtures:

¹ Shwartzman, G., *PROC. SOC. EXP. BIOL. AND MED.*, 1929, **26**, 843.

² Shwartzman, G., *PROC. SOC. EXP. BIOL. AND MED.*, 1928, **25**, 560; *J. Exp. Med.*, 1928, **48**, 247.