A relatively high acidity and high carbon dioxide tension of the arterial blood may be indicative of a relatively high efficiency of the transport of acid from the tissues and its liberation in the lungs.

The experiments support the view of the lack of correspondence between changes in acidity in the blood and in the tissues.

The results agree with the theory that the acidity of the respiratory center is important in the control of pulmonary ventilation. They are not in disagreement with a direct effect of oxygen on pulmonary ventilation by changes in the state of oxidation of the respiratory center.

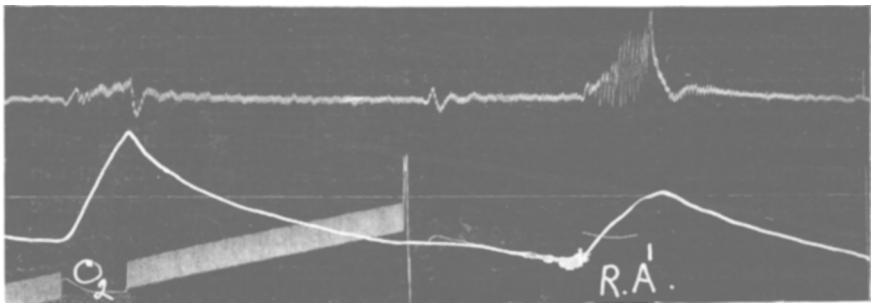


FIG. 1.

Record showing the effects of mechanical asphyxia during artificial ventilation on the acidity of the arterial blood and mean blood pressure. With the lungs filled with oxygen the increase in acidity of the blood is greater than in the second observation, in which the lungs were filled with room air. Note: the increase in mean blood pressure was considerably less.

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## Tissue and Blood Acidity: Coördination of the Dual Function of Hemoglobin During Low Oxygen Administration.

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This paper deals primarily with effects of low alveolar oxygen on oxidation, and resulting changes in acid metabolism, coupled with the impairment of transport of acid and its elimination in the lungs.

Low oxygen mixtures (3 to 12 per cent) were administered by normal ventilation, which permits control of ventilation by the animal, and by artificial ventilation, which maintains a constant

movement of air. Changes in acidity of the arterial and venous blood were studied with the continuous method, and with the quinhydrone and hydrogen electrodes.

Short administration of low oxygen (4 per cent) by normal ventilation elicited a sudden increase in alkalinity (0.1 to 0.2 pH) of the arterial and venous blood, followed by a rapid recovery on the re-administration of room air. Low oxygen administration elicited the usual increased pulmonary ventilation, which was followed by decreased ventilation on the re-administration of room air.

Similar administration of low oxygen by artificial ventilation, equal in volume to the preceding ventilation of room air, elicited markedly increased alkalinity of the arterial and venous blood. The increase in alkalinity, however, was smaller than that elicited on the administration of low oxygen by normal ventilation. The increased alkalinity of the blood resulting from administration of low oxygen may, therefore, be attributed to the washing out of carbon dioxide by excessive ventilation, and to the liberation of alkali in the process of reduction of the hemoglobin. These conclusions are supported by the recovery curves on the re-administration of room air. Whereas the normal acidity of the blood was only slowly approached on re-administration of room air with normal ventilation, with artificial ventilation it was rapidly approached. Overshooting occurred with subsequent recovery to the previous normal acid value. Such experiments indicate the accumulation of acid in the body during the administration of low oxygen, if increased ventilation is prevented.

The administration of pure oxygen, following low oxygen, led to a more rapid increase in blood acidity, and a greater suppression of respiratory movement.

Prolonged administration of low oxygen led to greater increased alkalinity of both the arterial and venous blood than short administration. This held for normal and artificial ventilation. With still longer administration, increasing alkalinity changed to increasing acidity. The directional change occurred earlier with artificial ventilation. The degree of acid change varied with the duration of administration. When markedly prolonged, the arterial and venous blood became more acid than normal. Subsequent administration of room air increased the acidity still more. Then followed a slow recovery, ending in an acid value above the preceding normal value.

With severe administration, disagreement between the manganese dioxide and hydrogen electrode occurred, indicating the appearance

of reducing metabolites in the blood stream, as a consequence of marked impairment of oxidations.

The increase in pulmonary ventilation, with increasing alkalinity of the blood on the administration of low oxygen, and the decrease or stoppage of ventilation with increasing acidity of the blood associated with re-administration of room air is in agreement with the common finding of an inverse relation between blood acidity and pulmonary ventilation.

Granting an increased total acid metabolism (carbon dioxide and lactic acid) and impairment of acid transport from the tissues by a broken coördination of the dual function of hemoglobin during oxygen deficiency, and granting the reverse, a decreased total acid production and improved transport of acid on the re-administration of oxygen, the results are in agreement with a direct relation between activity and acidity of the respiratory center.

The results support the view of the importance of acidity in the control of ventilation. They do not, however, preclude the direct effects of lack of oxygen on oxidation.

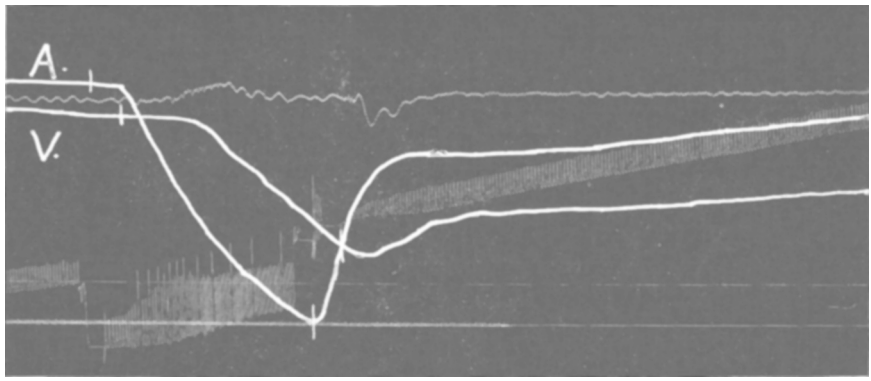


FIG. 1.

Record showing effects of administration of room air, 3 per cent oxygen, and pure oxygen on the acidity of the arterial and venous blood, on mean blood pressure and on pulmonary ventilation. The acidity curves are marked A and V, respectively. Note the cessation of respiration as the arterial blood turns acid. The recovery of the normal acidity values is incomplete at the end of the record. The blood is still turning acid in both the artery and vein.