discharge is not a factor in the accelerated pulse which accompanies muscular activity. The accelerator influence is acting in any case.

SUMMARY AND CONCLUSION

Intravenous injections of epinephrin under conditions closely simulating adrenal discharge cause not only increased blood-pressure, but, generally, also accelerated pulse. Acceleration of the pulse, therefore, is one of the adaptive functions of the adrenal glands.

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THE TIME THAT THE BREATH CAN BE HELD AS AN INDEX FOR ACIDOSIS

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The importance of acidosis in connection not only with diabetes but also with renal, cardiac and other diseases is being increasingly recognized. For practical clinical purposes it is essential, however, not merely to recognize the condition but also to be able to measure its intensity and to follow its variations—both those spontaneously occurring and those therapeutically induced. For this purpose, determination of the carbon dioxid content of the pulmonary air is rapidly coming to be recognized as the most accurate available method. In principle such a determination may be roughly said to assume that the sensitiveness of the respiratory center is normal and constant, and that the blood stimulus to it is composed of two factors, namely, carbonic acid (CO_2), and other acids (or deficiency of alkali). Whenever the former is found to be automatically decreased below normal, the latter is thereby indicated as above normal; in other words, there is a condition of acidosis.

Unfortunately the determination of the alveolar carbon dioxid is a procedure which, although not difficult for one who has acquired its technic, is quite different from most of the operations to which clinicians are accustomed. Its difficulty varies inversely with the amount of intelligent assistance which the patient can give. For these reasons there appears to be a need for some method of attaining a similar quantitative indication of the degree of acidosis which, even if somewhat less accurate, would be universally and more readily employable.[1]

I believe that such an index of the degree of acidosis is afforded by the length of time that the breath can be held voluntarily. The point of view now coming to be accepted regarding the relation of acidosis to the rate of pulmonary ventilation and the alveolar carbon dioxid is based to a considerable extent on data accumulated by the Pike's Peak expedition.[2] During our stay at the summit and after our descent we had frequent occasion to note that the length of time the breath could be held was shorter as the alveolar carbon dioxid was lower—that is, according to the intensity of the acidosis which causes the increased respiration of great altitudes. The relation was not, however, a direct proportion. While the alveolar carbon dioxid was reduced about one-third, most of the members of the party could hold their breath for less than half as long as at sea-level.

At that time the bearing of such observations on acidosis was not so clear as it is now becoming. We now recognize that one and the same underlying condition in the blood determines the resting alveolar carbon dioxid, the degree of dyspnea on slight exertion, and the duration of voluntary apnea. Lewis, Ryffel, Wolf, Cotton and Barcroft[3] have recently shown that the dyspnea of nephritis is due to an acidosis essentially like that developed in normal persons at great altitudes. Kennaway, Pembrey and Poulton[4] have found that by following the alveolar carbon dioxid in diabetics a warning drop in its tension indicates the approach of coma as long as forty-eight hours beforehand and earlier than any other method. Recent developments indicate that there are a number of distinct forms of acidosis.[5] It is quite possible that the effects on the alveolar air and voluntary apnea of the different forms of this, as yet little understood, condition may show specific variations; but if this proves to be the case, such differences would be of diagnostic advantage rather than otherwise. It will suffice for this preliminary notice to say that a few observations made for me by some of my clinical colleagues have shown that inability to hold the breath at all is an accompaniment of some at least of the forms of acidosis when approaching an acute stage.

While arrangements for a thorough investigation of this topic were under way, there appeared an abstract of a paper by V. A. Stange[6] in which voluntary apnea is recommended as a test of a patient's fitness to undergo general anesthesia. Stange regards the ability to hold the breath as depending on, and an indication of, the condition of the heart-muscle. He states (correctly) that the normal period is between thirty and forty seconds. He considers any period under twenty seconds as contra-indicating general anesthesia. While he appears to have no suspicion as to what it is, as we believe, which the test really depends on and indicates, namely, acidosis, he nevertheless reports observations on a number of chronic diseases in which he finds the duration of voluntary apnea to be abbreviated in about the degree in which acidosis is known from the results of other observers to occur.

At my suggestion a full investigation of this topic has been undertaken by Miss M. P. FitzGerald, who has an extensive experience in this field of work and who made important contributions in connection with the Pike's Peak expedition. The test used consists in directing the patient to sit quiet for at least five minutes; then to draw a full but not abnormally deep inspiration; and to hold it with the mouth closed and the nostrils compressed with the fingers, while the observer notes the time.

The practical utility of this test appears so considerable that it seems advisable to publish it even in its present incompletely studied condition. This is done in the hope also that some of those who may use it will favor us with a report of their observations to supplement and control our own.

1. Compare Boothby, W. M., and Peabody, F. W.: A Comparison of Methods of Obtaining Alveolar Air, Arch. Int. Med., March, 1914, p. 497.
2. Douglas, Haldane, Henderson and Schneider: Phil. Tr. Roy. Soc., 1913, Series B, cciii, 185. FitzGerald, M. P.: Ibid., p. 351.
3. Lewis, Ryffel, Wolf, Cotton and Barcroft: Heart, 1913, v, 45.
4. Kennaway, Pembrey and Poulton: Jour. Physiol., 1914, xlvii; Proc. Physiol. Soc., p. 10.
5. Compare A. W. Sellards: Bull. Johns Hopkins Hosp., 1914, xxv, 101, 141.
6. Stange, V. A.: Russk. Vrach, 1914, xiii, 72; abstr., THE JOURNAL A. M. A., April 4, 1914, p. 1132.