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It may now be considered established that the mechanism of chemoreception in the carotid sinus and intestinal reflexogenic zones is closely connected with the carbohydrate metabolism in the tissue of the receptors [1, 2, 4, 5, 6]. However, the role of the C₃ acid cycle in the mechanisms of chemoreception has not yet been explained. It was shown in a previous communication [3] that disturbance of the glycolytic cycle by sodium fluoride, or of the C₃ acid cycle by hydroxylamine leads to a decrease in the sensitivity of the intestinal receptors to acetylcholine. The depressing action of sodium fluoride on reflexes caused by acetylcholine may be abolished by the addition to the perfusate of the end product of glycolysis, namely pyruvic acid, but the depressing action of hydroxylamine is not abolished by this acid. In this connection it has been postulated that the role of the various links in the chain of tissue metabolism in the mechanisms of chemical stimulation of the intestinal receptors is not identical, and that in fact the C₃ acid cycle is more closely connected with the mechanism of reception of acetylcholine than is the glycolytic cycle.

It was considered of interest to investigate whether similar relationships also applied to the chemical stimulation of the receptors of the carotid sinus reflexogenic zone.

EXPERIMENTAL METHOD

In 29 cats under urethane anesthesia the isolated carotid sinus was perfused with oxygenated Ringer-Locke's solution. All vessels leading from the carotid bifurcation were ligated with the exception of the external and common carotid arteries into which were inserted efferent and afferent cannulas, and the carotid sinus nerve was preserved intact. As stimulus we used acetylcholine in a dilution of 10⁻⁴ g/ml, which was injected into the perfusion fluid with a syringe in a volume of 0.5 ml. The reflex changes in arterial pressure and respiration were recorded by the usual methods.

It must be pointed out that when the receptors of the carotid sinus were exposed to the action of acetylcholine, besides a deepening of respiration we often observed an increase in the arterial pressure in some experiments and a decrease in others. Since a control injection of 0.5 ml of Ringer-Locke's solution did not cause such a fall in the blood pressure, the depression caused by the acetylcholine could not be associated with the increased pressure within the sinus itself as a result of injection of the acetylcholine solution. It must also be mentioned that after division of the carotid sinus nerve, acetylcholine in the same dose did not cause changes in the respiration and blood pressure, which undoubtedly suggests that the depressor reaction was reflex in nature.
Fig. 1. Reaction of the circulation and respiration to stimulation of the carotid sinus receptors of a cat with acetylcholine (0.5 ml, 10^{-4} g/ml). A) Perfusion of the carotid sinus with Ringer-Locke's solution without sodium pyruvate. Reflex to acetylcholine: a) 5 minutes before injection of sodium fluoride; b, c, d, e) 5, 15, 40 and 50 minutes after injection of sodium fluoride into perfusion fluid (1 ml 10^{-2} g/ml). B) Perfusion of carotid sinus with Ringer-Locke's solution containing sodium pyruvate in a concentration of 10^{-4} g/ml. Reflex to acetylcholine: f) 5 minutes before injection of sodium fluoride; g, h, i, j) 5, 15, 40 and 50 minutes after injection of sodium fluoride (1 ml 10^{-2} g/ml). Significance of curves (from above down): respiration; arterial pressure; zero line; velocity of perfusion; time marker (5 seconds); stimulation marker.

**Experimental Results**

Identical results were obtained in eight experiments, as shown in Fig. 1.

It will be seen from Fig. 1, a-e that the reflexes to acetylcholine were at first depressed by the action of sodium fluoride, and were subsequently restored 40-50 minutes after injection of sodium fluoride. After the reflexes had been restored the carotid sinus was perfused with Ringer-Locke's solution containing the sodium salt of pyruvic acid in a concentration of 10^{-4} g/ml. Against this background sodium fluoride was again injected, and at various intervals of time after the injection the magnitude of the reflexes was determined. It is clear from Fig. 1, f-j that the respiratory reflex was not restored at all under these conditions, but the depressor reflex was restored after the same period of time as in the absence of pyruvic acid.