SPECIFIC MECHANORECEPTOR ZONES OF THE CIRCULATORY SYSTEM IN FROGS

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Discharges of the nerve of the carotid labyrinth in amphibians, which is considered to be homologous with the mammalian carotid sinus, are intensified if the intralabyrinthine pressure rises [6, 10], but the arterial pressure (BP) and heart rate are unchanged under these circumstances [5, 7, 12]. At the same time, if the pressure rises in the pulmo-cutaneous artery of toads, innervated by the laryngeal branch of the vagus nerve, marked bradycardia develops and BP falls [5, 7]. Dieter [3, 4] found that if filling of the vascular system (FVS) increases or decreases in frogs, the efferent tonic impulsation (ETI) in the renal nerve weakens or strengthens respectively. He suggested that the intensity of ETI is determined by filling of the heart, and showed that it is sufficient to increase the filling of the right atrium only to produce detectable depression of ETI. Information on the state of the frog's heart can be transmitted by mechanoreceptors of the atria and ventricle [8, 9, 11], volleys of which are intensified during an increase in filling of the heart [8, 9]. After bilateral vagotomy changes in FVS do not change impulsation in the renal nerve [3, 4]. In amphibians the latter contains vasoconstrictor fibers [14] and is therefore representative of the vasoconstrictor system. The results of previous investigations [3-5, 7] suggest that afferent innervation of specific mechanoreceptor zones of the cardiovascular system, controlling its state as a whole in amphibians, is effected only by the vagus nerves, i.e., there is as yet no functional analog of the carotid sinuses in amphibians, which in higher vertebrates are innervated by a branch of the glossopharyngeal nerve [1].

Since data in the literature [3, 4] are very important for an understanding of the evolutionary development of regulation of the circulation, their reliability must be beyond question. This was the aim of this investigation.

EXPERIMENTAL METHOD

Under ether anesthesia a cannula was inserted into the abdominal vein of male frogs (*Rana temporaria*) weighing 30-80 g, and viadril (13 mg/100 g body weight) was injected through it. BP was measured in the left sciatic artery with an electromanometer. To record ETI, one of the thickest trunks running from the sympathetic chain to the kidney was isolated, divided, and its central end was placed on bipolar silver electrodes. Impulses from them were led through a UBP2-03 amplifier to an oscilloscope and integrator (integration time 10 sec). BP, integral ETI, and heart rate (HR) were recorded with an automatic writer. The animals' temperature was maintained at 18-20°C. To change FVS a three-way tube connected to the abdominal vein, the right sciatic artery, and a syringe was used. To increase FVS, a 2.5% solution of rheopolyglucin was injected into the abdominal vein in the course of 3 to 5 pulse beats, and to reduce FVS blood was withdrawn from the sciatic artery for the same period of time. The vagus nerves were divided 1-2 mm proximally to the origin of the gastric branch. Changes in ETI were studied in 14 frogs: in nine of them dependence of ETI on the degree of FVS was studied before and after bilateral vagotomy, in four frogs before vagotomy only, and in one frog after vagotomy only. Immediately before ETI began to be recorded the animals were immobilized with flaxedil (2 mg/100 g intravenously). The initial BP was between 20 and 40 cm water. To assess the responses, the average of 10 consecutive integrations of ETI was determined. The statistical significance of changes in ETI was determined by Student's t test.

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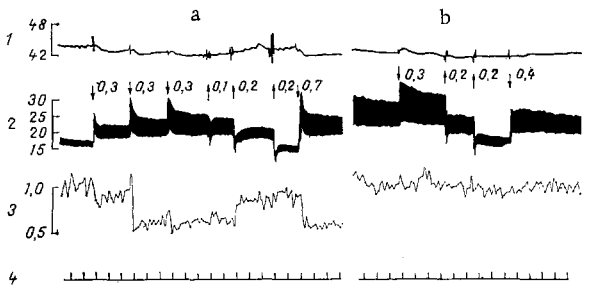

Fig. 1

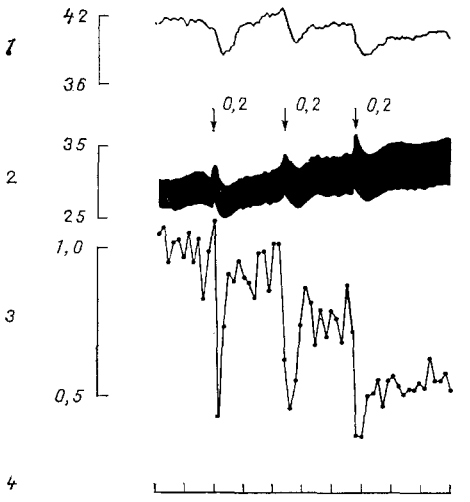

Fig. 2

Fig. 1. Dynamics of tonic impulsation in renal nerve during a change in FVS before (a) and after (b) division of vagus nerves (experiment No. 2). 1) HR, beats/min; 2) BP (in cm water); 3) integral of ETI (result of averaging 10 consecutive integrations at the smallest value of FVS was taken as unity); 4) time marker 1 min. Arrow pointing downward indicates time of increase of FVS, arrow pointing upward indicates time of decrease of FVS. Number on right of arrow indicates volume (in ml) of fluid injected or withdrawn. Initial BP under 20 cm water, for FVS was reduced beforehand.

Fig. 2. Dynamics of sympathetic activity (before vagotomy) in response to change in FVS (experiment No. 6). Legend as to Fig. 1.

EXPERIMENTAL RESULTS

A stepwise increase in FVS led to a stepwise weakening of ETI when the vagus nerves were intact, and a decrease led to stepwise restoration of its intensity (Fig. 1a). In half of the experiments a dynamic component of the reaction was found: strong temporary (10-30 sec) inhibition of ETI after an increase in FVS (Fig. 2), and in the other half of the experiments no dynamic component was found, possibly because it could have been significantly shorter than the integration time (10 sec). The response of BP to an increase in FVS developed in two phases (Fig. 1a). During the first phase, the transition process due to the temporary strong increase in the venous return to the heart, BP rose sharply and then fell slowly. The second phase was marked by a steady BP with systolic pressure higher than initially, and with a slightly changed diastolic pressure. This may have been due to the increased stroke volume of the heart. A temporary and marked fall of BP accompanhing a decrease in FVS appeared both before and after vagotomy and was due to rapid pumping of blood from the arterial system (Fig. 1). Sometimes after the first phase of change in BP, it fell in a series of waves, during which BP could actually fall below its initial value (Fig. 2). This fall of BP occurred when and only when a dynamic component of the response of ETI was discovered. Consequently, this fall of BP was evidently due to deep inhibition of discharges of vasocontrictor neurons. After vagotomy neither an increase nor a decrease in FVS changed ETI even temporarily (Fig. 1b). Meanwhile the first phase of the change in BP during an increase in FVS disappeared, and the static component of the response of a rise of BP was potentiated. The only exception was one experiment in which a statistically significant ($P < 0.001$) increase in ETI appeared systematically after vagotomy in response to a decrease in FVS, but subsequent increases in FVS did not cause any statistically significant changes in ETI ($P > 0.05$).

The heart rate was unchanged during an increase (decrease) in FVS, or it decreases (increased) a little even before vagotomy, probably due to the vagolytic action of flaxedil [2]. In fact, in a control experiment the bradycardia which appeared in response to stimulation of the peripheral end of the divided vagus nerve, was completely abolished by flaxedil in the standard dose for these experiments. Receptors of the atrium play an important role in these reactions [3]. To discover the possible contribution of mechanoreceptor zones of the arterial system, the zone in the pulmo-cutaneous artery, for example [5, 7], changes in ETI were cor-

related with changes in mean BP. It was considered that since the distribution of values of the ETI integral was chosen as normal, any possible correlation could be discovered by calculating the coefficient of correlation [13]. However, changes in ETI correlated only weakly with changes in the mean BP (the coefficient of correlation as a linear approximation $|r| = 0.02$, which is less than the 10% confidence limit) and, consequently, ETI depended weakly on the mean BP. Meanwhile, just as in [3], it was found that ETI correlated strongly before vagotomy with the pulse pressure. Even as a linear approximation $|r| = 0.79$, which is much greater than the 0.1% confidence limit. With small changes in heart rate and no change in the arterial elastic reservoir, the pulse pressure, according to Starling's law, depends on diastolic filling of the heart. Absence of correlation between changes in ETI and the mean BP, and strong correlation between changes in ETI and the pulse pressure may therefore mean that receptors transmitting information about the degree of filling of the heart play a determinant role in the responses of ETI to changes in FVS. For instance, the dynamic component of the response (Fig. 2), which was observed in 50% of experiments, was evidently due to an increase in stretching of the heart during the increase in FVS. The scatter of the experimental points may be due to differences in the initial FVS. In fact, in experiments 3-7, before recording of ETI began, no fluid was injected (except the solutions of viadril and flaxedil). Calculation for these five experiments only gives $|r| = 0.88$. After vagotomy, correlation between ETI and pulse pressure disappeared; the coefficient of linear regression was 0 with a level of significance $P < 0.01$.

Unlike other workers [3, 4], we recorded the integral of ETI, so that we were able to study the dynamics of the response. However, in our experiments also, vagotomy led to disappearance of changes in ETI, even temporarily, in response to changes in FVS. This confirms the view that mechanoreceptor reflexogenic zones in frogs controlling the state of the circulatory system as a whole are innervated by the vagus nerves only.

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