THE NERVOUS REGULATION OF CARDIAC RHYTHM IN FISH

COMMUNICATION II

THE VAGUS NERVE IN FISH – AN EFFERENT PATH OF DOUBLE REFLEX INFLUENCES ON THE CARDIAC RHYTHM

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The reflex regulation of the heart in fish has been studied by many authors [2, 3, 11, 13, 15-19, 21]. It has been established that stimulation of various intero- and exteroceptors, or of afferent nerves, causes cardiac arrest or slowing of the rate of cardiac contractions. At the same time, certain authors [2, 15, 16, 21] observed reflex acceleration of the heart rate. Neither inhibition nor stimulation of the heart rate is observed after section of the cardiac branches of the vagus [2, 15, 17] whereas destruction of the first sympathetic ganglion, the forebrain and the spinal cord does not abolish these reflex effects [16]. The most pronounced tachycardia and bradycardia are noted on stimulation of the swim-bladder and the gills, i.e., organs which send sensory fibers to the medulla within the branches of the vagus.

Reflex tachycardia described in the works mentioned is usually considered to be the result of lowering of the inhibitory tonus of the vagi. Thus, F.D. Vasilenko and K.S. Koshtoiants [2] explain the tachycardia observed on reduction of pressure within the swim-bladder as a passive consequence of exclusion of afferent influences which maintain reflexly the tonus of the vagal centers. This interpretation is contradictory to observations of other authors who have shown that increased heart rate may result on raising the pressure within the swim-bladder [21], i.e., that it may arise as the result of the appearance rather than disappearance of afferent impulses. Lutz [16] found in his experiments that acceleration of the rate of flow of fluid through the gills was accompanied by increase in the heart rate and reduction in the rate of flow by slowing of the heart.

On the basis of the possibility discovered by us [4] of actively accelerating the heart rate in fish by stimulating the peripheral portions of the cardiac branches of the vagi it was decided to trace the way in which this mechanism was utilized in reflex regulation of the heart rate.

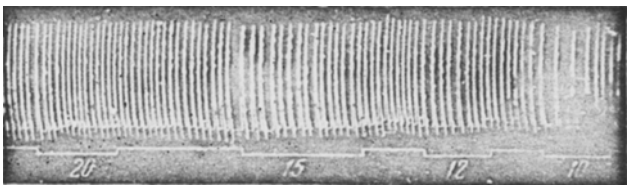
The experimental method used in these experiments has been described in the previous communication [4]. Certain additional procedures will be cited in the course of presentation of experimental material.

EXPERIMENTAL RESULTS

Both inhibition and stimulation of the heart rate were observed on stimulation of the central portions of individual gill branches of the vagus. The two effects were not specific for the different gill nerves. Stimulation of each of the gill nerves could lead to acceleration or slowing of the heart rate and to complete cardiac arrest for varying periods of time. The nature of the effect depended in each individual case on the strength of stimulation of the given nerve. Relatively weak stimulation caused acceleration of the heart rate (Figure 1a, 2c). Increasing the strength of stimulation led to inhibition of cardiac activity (Figure 1, b, c; 2, b); this inhibi-

tion became even more marked when the stimulation of the same afferent nerves was increased in strength (Fig. 1d, 2a). Such consistent results could be obtained on stimulation of the central portion of any of the four gill branches both on the right and on the left sides.

The results cited do not exclude the hypothesis that weak stimulation of the gill nerves brings into play some special accelerator fibers whose influence is overshadowed on stronger stimulation by the activity of specific inhibitory nerve elements. If this were so it should have been expected that simultaneous stimulation of two afferent nerves, separate stimulation of each of which elicited acceleration of the heart rate, would lead in all cases to even greater acceleration of the heart, whereas simultaneous stimulation of gill branches, whose separate stimulation elicited acceleration in the case of one and slowing of the heart rate in the case of the other, would lead to some degree of levelling off of the effects of their separate stimulation.



a b c d

Fig. 1. Reflex changes in the rhythm of cardiac contractions in pike on electric stimulation of the central end of the 3rd gill branch of the vagus. Records from above down: mechanical record of cardiac activity, stimulus marker; figures denote distance between the induction coils.

In order to test these hypotheses simultaneous stimulation was applied to the central ends of two different gill nerves whose separate stimulation could, as shown above, produce the most varied effects.

a b c

Fig. 2. Reflex changes in the rhythm of cardiac contractions in perch on electric stimulation of the central end of the 5th gill branch of the vagus. Records as in Figure 1.

Two afferent nerves were placed on stimulating electrodes, separate for each gill branch. Each pair of electrodes was connected with separate induction apparatus. The two stimulating circuits shared the circuit breaker and the charging source which ensured that, with equal distances between the primary and secondary coils, the current strength and frequency of breaks were the same. When reproducible results of separate stimulation of the gill nerves with a selected strength of current were established two branches were stimulated simultaneously. The results of some of these experiments are summarized in the table on the following page.

The results of the first two examples given in the table can only be comprehended if it is assumed that during separate stimulation of each branch with subthreshold (but approaching threshold) current strength a certain number of afferent fibers entering the branch is excited. However, this number of reflex arc elements involved in activity proves to be inadequate to effect reflex changes in the work of the heart. Increase in the number of active afferent fibers by means of similar "subthreshold" stimulation of another branch leads to definite reflex acceleration of the heart rate, i.e., produces the same effect as that observed in response to weak but suprathreshold stimulation of each of these nerves separately (Figure 1a; 2c) when the number of excited fibers in the one nerve trunk is already sufficient to produce this effect. When stimulation of one of the nerve branches leading to acceleration of the heart rate was joined by "subthreshold" stimulation of another, the resultant effect

of such simultaneous stimulation was expressed not in reinforcement of the positive influence, but in its conversion to the functionally opposite effect – inhibition. The same result was obtained on simultaneous stimulation of two branches which, stimulated separately with a certain current strength, elicited acceleration of the heart rate (examples 4 and 5). The results of the next three examples cannot be understood in terms of the presence of specific accelerator or inhibitor nerve fibers in the gill nerves. It appears that an increase in the number of nervous elements involved in activity leads to reflex inhibition of the heart, although formerly, stimulation of these elements caused tachycardia. This hypothesis is also vividly brought out by results of Examples 6-9 which show that simultaneous stimulation of two afferent nerves, which gave opposite effects when stimulated separately, leads not to the mutual attenuation of these opposite effects but to enhancement of one of them, viz. inhibition; a qualitatively similar result is obtained on combination in time of two stimuli each of which had caused weak inhibition of the heart rate (Example 10). Comparison and analysis of ten examples of combined stimulation of various gill nerve branches thus permits only one interpretation of the results of this series of experiments: the nature of the reflex reaction of the heart depends on the number of qualitatively similar afferent conductors involved in activity by means of one or another stimulus. Excitation of a definite number of such fibers determines the reflex acceleration of the heart rate; when this number is increased, cardiac inhibition develops and becomes progressively more profound as more and more afferent fibers are involved in activity.

No. in order	Heart beats per minute			
	without stimulation	with separate stimulation		simultaneous stimulation of two branches
		one branch	another branch	
1	29	29 (III)	29 (IV)	34 (III+IV)
2	17	17 (III)	17 (IV)	29 (III+IV)
3	17	19 (II)	17 (III)	14 (II+III)
4	19	22 (II)	24 (III)	17 (II+III)
5	19	22 (II)	22 (III)	14 (II+III)
6	24	19 (II)	29 (IV)	15 (II+IV)
7	24	20 (II)	28 (IV)	8 (II+IV)
8	24	17 (III)	27 (IV)	7 (III+IV)
9	24	19 (IV)	31 (II)	2 (IV+II)
10	26	24 (IV)	21 (III)	5 (IV+III)

Note: The numbers in brackets refer to the gill nerves.

The results cited (Figures 1 and 2) do not resolve the question as to the particular part of the reflex arc in which those concrete conditions are laid down and which determine the character of the cardiac reaction to a given influence. This may occur at the vagal center or in the heart itself. In the first case the effects noted could be regarded as the result of change in the central tonus of the vagi, or it may be assumed that drawing in of different numbers of uniform afferent fibers into activity is associated with involvement of different afferent nerve elements, which exert a specific action on the heart leading only to well-defined changes in its activity.

In order to elucidate these questions a special series of experiments was performed in which the central end of one of the gill nerves was stimulated repeatedly by current of the same strength, causing definite cardiac inhibition (Figure 3a). In the intervals between stimuli the previously prepared cardiac branches of the vagi were transected successively; these branches carried the centrifugal pathways of the reflex under consideration. Following transection of one of the four cardiac branches the inhibitory effect of the stimulation of the second gill branch with the former current strength was found to be diminished (Figure 3b). After transection of another cardiac branch the same stimulation caused very feeble slowing of the heart rate (Figure 3c). Transection of yet another (third) efferent branch was associated with acceleration and not slowing of the heart rate when the same stimulus was applied (Figure 3d). Tachycardia could not be elicited after transection of the last cardiac branch. When the order of transection of the cardiac nerves was altered no change occurred in the successive phenomena demonstrated in Figure 3. This refutes the hypothesis that accelerator fibers were concentrated in some one branch in the presence of which reflex stimulation led to tachycardia. At the same time these experiments leave no doubt that vagal tachycardia cannot be the result of interference with the tonus of the vagi but results from reflex excitation of an artificially limited number of vagal efferent fibers.

The material presented permits representation of the reflex regulation of the heart in fish as follows: stimuli differing in intensity involve a definite number of afferent fibers in activity and lead to reflex excitation of a greater or lesser number of efferent elements of the medulla and consequently of a corresponding number of centrifugal nerves whose active impulse action on the heart determines the final effect exerted on it by the reflex. The centrifugal pathway of functionally opposite reflex influences on the heart is constituted by homogeneous elements of the vagi. These elements are not in their constitutional properties either enhancing or inhibitory. When they are drawn into activity in definite, relatively small numbers they elicit tachycardia, while excitation of a larger number of the same efferent fibers elicits various degrees of cardiac inhibition. Therefore, without confirming the hypothesis of qualitative specificity of the inhibitory action of the vagus on the heart, whose logical development is the theory proposed by Skramlik [20] suggesting a unilaterally inhibitory influence of the nervous system on the heart in fish, we interpret the results obtained in accordance with concepts developed by M.G. Udel'nov[6], who showed that the structural-functional basis of cardiac regulation is the characteristically parasympathetic intramural synaptic apparatus concerned with dispersion and multiplication of impulses, leading not only to tachycardia, but also to bradycardia. It is characteristic that the heart in fish is that organ in which the vagal fibers first acquire synaptic discontinuity, i.e., features of a parasympathetic nerve.

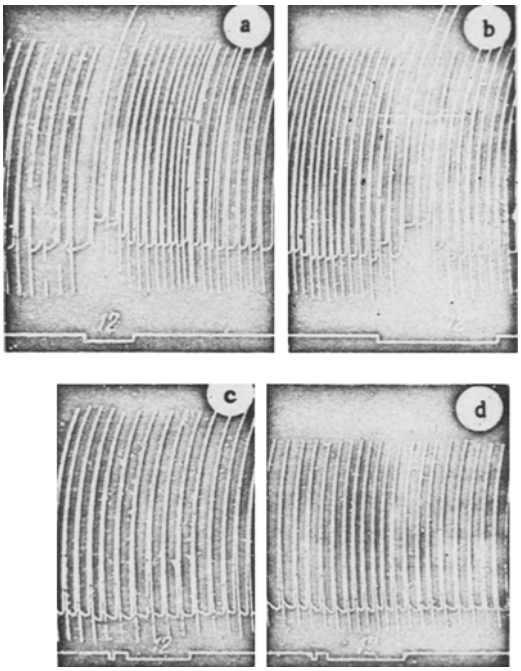


Fig. 3. Reflex changes in the rhythm of cardiac contractions in pike on electric stimulation, identical with respect to strength, of the central end of the 2nd gill branch of the vagus with all cardiac branches of this nerve intact (a), after transection of one of these branches (b), after transection of 2 cardiac branches (c), after transection of 3 cardiac branches of the vagus (d). Records the same as in Figure 1.

The fact that stimulation of the reflexogenic zone of the gills can exert a dual effect on the heart becomes particularly significant in connection with the hypothesis first enunciated by Koch [14] and recently elaborated in detail by Chernigovskii [7, 8] which is concerned with the origin of the carotid sinus, aortal and pulmonary reflexogenic zones of pulmonates in the gill reflexogenic zone of the lower vertebrates. This hypothesis has found sound basis in the investigation of Boyd [9], and Bystrov [1].

V.N. Chernigovskii visualizes the high sensitivity of the carotid sinus, aortal and pulmonary reflexogenic zones as the result of all these afferent areas arising from the blood vessels of the gill region, i.e., from vessels which in fish are constantly in direct contact with the external environment. This suggestion is confirmed by the data of a number of authors which show that the strongest cardiac reflexes arise on stimulation of the gill reflexogenic zone [21]. The functional characteristics of this zone as a source of bilateral regulatory influences on the heart supplement this analogy and permit the inclusion of the buffer properties of the strongest reflexogenic zones of the higher vertebrates – carotid sinus, aortal and pulmonary zones – among the buffer-compensatory reflex influences on cardiac activity; this can be deduced from such properties shown by their homolog, the gill reflexogenic zone, which exerts a bilateral and therefore buffer-compensatory effect on the heart. Taking into account that the heart is the main organ maintaining the necessary circulatory level in fish and that the vagus in these animals is the only centrifugal pathway for all cardiac reflexes, the origin of vagal function in higher vertebrates as the main regulator of general blood pressure can be readily understood [5, 6].

SUMMARY

It was established that electric stimulation of the central endings of different gill nerves in fish cause a reflex change of heart rate. Weak stimulation results in increased frequency of the heart rate. Stronger stimulation causes decreased frequency or complete arrest of the heart. Simultaneous stimulation of two gill nerves by

the current which caused tachycardia when each nerve was stimulated separately, caused cardiac inhibition. Simultaneous stimulation of two gill nerves, the separate stimulation of one of which resulted in decrease of the heart rate and of the other in increase, caused even more pronounced inhibition of the heart. If the strength of stimulation of one of the gill nerves is kept constant the effect of the reflex action changes depending on the number of the intact efferent nerves of the heart. If all the nerves are intact the frequency of the heartbeat is decreased, while if a number of them are cut tachycardia results. This provides evidence against the qualitative specificity of the individual afferent or efferent elements in the reflex arc under investigation. It points to the dependence of the quality of the reflex effect on the number of the nerve elements involved in this reaction.

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