AFFERENT IMPULSES FROM CARDIAC VAGAL FIBERS IN MYOCARDIAL ISCHEMIA

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Many authors [2-5, 11] have shown that afferent impulses in the cardiac nerves are considerably altered in experimental myocardial ischemia. However, many of the results are contradictory.

Because most of the work on this subject has been carried out by making records from whole nerve trunks, the object of the present work has been to study afferent impulses in individual vagal fibers.

EXPERIMENTAL METHOD

We carried out 45 experiments on cats anesthetized with 80 mg/kg chloralose. After the thoracic cavity had been opened respiration was maintained artificially. At the level of the inferior edge of the auricle of the left atrium on the descending branch of the left coronary artery (together with the vein) we applied a loop which enabled us to compress or release the vessel. The left vagus was divided at the ganglium nodosum. Paintal's method was used to divide the nerve trunk in the neck into small bundles and into single fibers. The animals were placed in a screened room. Potentials were picked up on platinum electrodes. Photographic records were made from a cathode ray tube on a 3-channel electromyograph made by the firm "Diza." At the same time we recorded the electrical activity from a single fiber, and the ECG, Lead II. The descending branch of the left pulmonary artery was compressed for 5-15 min.

EXPERIMENTAL RESULTS

Normal afferent impulses in fibers from the receptors of the atria and ventricles occur in volleys containing a variable number of impulses all equal in amplitude and occurring at definite phases of the cardiac cycle.

In 33 of the 45 experiments, on compression of a descending branch of the left coronary artery we observed an increase in the number of impulses in each volley; the volley did not occur immediately after cessation of the flow in the coronary artery, but after 30 sec or more. Figure 1 indicates the nature of the change. Figure 1a shows volleys consisting of six impulses all equal in amplitude (Paintal's type). Thirty seconds after compression of the coronary artery, in the intervals between the volleys new impulses of the same amplitude occurred. During this time there was an elevation of the trace during the ST interval and the period included the beginning of the formation of the coronary T wave (Fig. 1b). After the loop had been released from a coronary artery the impulses and the ECG returned to their original state. These results therefore confirm those of many other investigators [2, 3, 5, 11] who found that an increase of afferent impulsation in the cardiac nerves. We must, however, note that the afferent impulsation does not always change in this way. In some of the experiments the number of impulses in a volley showed practically no increase, and it was merely the arrangement of the impulses within the volley, i.e., the structure of the volley which was altered. It is known that the arrangement of impulses from the atrial receptors varies during the respiratory cycle. The number of impulses per volley from a single atrial receptor increases with increase of the blood flow to the heart. Therefore, the activity of receptors of the right atrium reaches a maximum intensity at a moment of maximum inspiration, and maximum activity of receptors of the left atrium occurs at the onset of expiration.

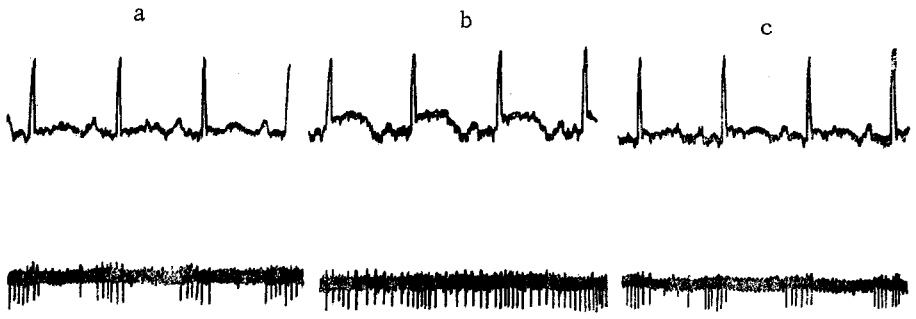

Fig. 1. Change in afferent impulsation in a single cardiac fiber of the left vagus after compression of the descending branch of the left coronary artery. a) Original background impulsation; b) impulses 30 sec after compression of the coronary artery; c) 5 sec after removal of the loop from the coronary artery. Above—ECG in Lead II; below—afferent impulses in a fiber (paper speed 5 cm/sec).

After compression of the coronary vessel there was an irregular increase and then a reduction in the number of impulses during the respiratory cycle, i.e., there was an alteration in the form of the volley.

In two experiments, after compression of the coronary artery there was a reduction of afferent impulsation, and in one case there was no change at all. In this last case we must suppose that in the vagal bundle investigated there were no fibers whose endings were situated in or near the ischemic zone.

In order to test this hypothesis we attempted to find in the nerve trunk a fiber which ran if not from the ischemic zone itself at any rate from some region nearby.

It was found that on compression of the anterior descending branch of the left coronary artery afferent impulsation in a fiber from the receptors in the left ventricle may remain almost unchanged.

Figure 2 shows impulses in such a fiber. In this fiber the background activity (Fig. 2a) was very definitely enhanced after the compression of the aorta at its origin (Fig. 2b); it returned to normal after removal of the aortic clamp (Fig. 2c), and disappeared almost completely after compression of the pulmonary artery at the base of the heart (Fig. 2d). In our opinion all these facts are evidence that the receptors of the fiber in question lie in the left half of the heart.

If, however, we take into account that the volleys of the impulses coincided with the QT section of the ECG, and that the number of impulses in each volley does not vary during the respiratory cycle, then it would appear that these receptors must be ventricular [10]. Despite the marked changes in the ECG there was no increase in background activity (Fig. 2f) either 3 min (Fig. 2g) or 6 min (Fig. 2h) after compression of the descending branch of the left coronary artery. There was a reduction of one impulse one minute after restoration of flow in the coronary vessel (Fig. 2i). We must note that there was a considerable increase in the number of impulses in this fiber during general asphyxia produced by arrest of artificial respiration (Fig. 2j).

Sometimes when the coronary artery was compressed for as long as 10-15 min we were able to observe that immediately after the increase of afferent impulses there was some reduction in rate, and then a further increase; here, the intervals between such "waves" varied according to the experiment from 3 to 10 min.

It is known that even 1 min after obstruction of the coronary artery the ischemic portion becomes considerably extended during the period of isometric contraction [12, 13]. Were this distension the cause of the increased flow of impulses then it would be expected that when the descending branch of the left coronary artery was compressed there would be a particularly large increase of impulses in a fiber originating in a left ventricular mechanoceptor. However, in our experiments this effect was by no means always observed.

On the other hand there is no reason to suppose that the variation in afferent impulsation in the cardiac nerve fibers which follows compression of the coronary artery is due to any "specific" adequate stimuli and not to dilatation of the myocardium.

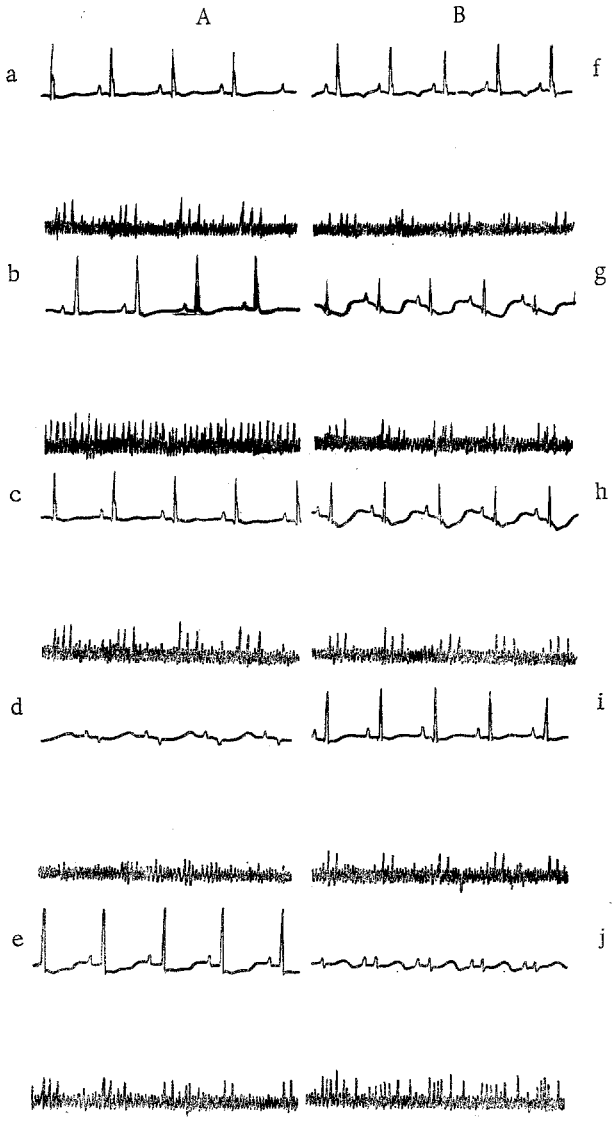

Fig. 2. Impulses in an afferent fiber of the left vagus running from a single mechanoceptor of the left ventricle. A) Identification of fiber: a) initial background impulsation; b) impulses under compression of the aorta at the base of heart; c) after removal of aortic clamp; d) after compression of pulmonary artery; e) after removal of clamp from pulmonary artery; B) change in impulses in the same fiber after compression of the descending branch of the left coronary artery: f) original background impulsation; g) impulses 3 min after compression of the coronary artery; h) after 6 min; i) 1 min after restoration of blood flow in coronary artery; j) impulses in the same fiber during general asphyxia. Above - ECG in Lead II; below - afferent impulses in a fiber.

According to Paintal [8, 9] there are two kinds of receptors in the heart: type A (in the right atrium) and type B (in both atria). He considers that increase of atrial pressure constitutes an adequate stimulus for type A receptors, and that filling of the atria is the adequate stimulus for type B receptors.

However, recently many authors [6, 7] have shown that there are no grounds for belief in the existence for such specificity.

Therefore, to explain a variation in afferent impulsation in our experiments it would appear to be necessary to postulate the existence of various conditions into which the receptors in question may fall after compression of the coronary artery.

We must note that even after a considerable change in the ECG indicating the existence of a myocardial ischemia, in many experiments we found no marked change of afferent impulsation. Consequently, in these experiments we failed to confirm that metabolic products accumulating in cardiac muscle as a result of the ischemia directly influence cardiac mechanoceptors. This might be true of a certain proportion of the receptors for the duration of the coronary compression used in our experiments.

Most probably the change in flow in the cardiac vessels and the consequent metabolic change in the myocardium influence the contractile power of cardiac muscle. Then the extent to which the cardiac wall may be extended varies from one part to another. The total effect is reflected in changes of afferent impulsation from the cardiac mechanoceptors which are then exposed to different conditions and react in different ways to the altered work of the cardiac muscles. It is this variation which in all probability comprises the complete information carried from the heart to the central nervous system when the coronary artery is compressed.

SUMMARY

When the descending branch of the left coronary artery is compressed the rate of flow of impulses in different cardiac fibers may be affected variously: there may be an increase, a decrease or a fluctuating change at different times after compression of the coronary vessels.

In certain fibers there was no change in impulse rate, although these fibers ran from cardiac mechanoceptors located in or near the ischemic zone.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
