

REFLEXES FROM THE MECHANOCEPTORS OF THE UPPER RESPIRATORY TRACT ON THE HEART IN EXPERIMENTAL MYOCARDITIS

(UDC 616.127-002-092.9-06:612.211/.232-009.81)

R. A. Fel'berbaum

Leningrad Research Institute of Diseases of the Ear, Throat, Nose, and Speech

(Director, Professor I. A. Lopotko; Scientific Director, Active Member

of the Academy of Medical Sciences of the USSR Professor V. I. Voyachek)

(Presented by Academician V. N. Chernigovskii)

Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 61, No. 6,
pp. 33-37, June, 1966

Original article submitted November 5, 1964

It was shown in a previous communication that stimulation of the mechanoreceptors of the upper respiratory tract of healthy cats and of cats with tuberculosis of the larynx caused only a slowing of the heart rate. No changes were observed in the conductivity and contractility of the heart muscle. Reflex changes in the heart rhythm in responses to experimental stimulation of the upper respiratory tract have been discovered by other investigators [5, 6, 8, 12, 15, 16-21].

Reflex effects from various internal organs on the diseased heart (myocarditis, etc.) are known to be more severe than on the healthy heart, and they include changes, not only in its rhythm, but also in the conductivity and the contractile power of the heart muscle [1, 2, 3, 4, 7, 11-14].

The object of the present investigation was to study the character and degree of the reflex influences from the mechanoreceptors of certain areas of the upper respiratory tract—the trachea, larynx, and pharynx—on the heart affected by a pathological process (experimental myocarditis).

EXPERIMENTAL METHOD

As described by other authors [3, 4], experimental myocarditis was produced in cats by injecting caffeine benzoate (0.25 ml of a 10% solution per kilogram body weight), followed 2 min later by adrenalin hydrochloride (0.2 ml of a 0.1% solution per kilogram body weight), into the femoral vein. These injections were repeated six times on alternate days.

Acute experiments were conducted on 21 animals 2-8 weeks after the beginning of the injections. Under superficial urethane anesthesia, the trachea was divided in the cats between the 7th and 8th tracheal rings. A plastic cannula was inserted into its caudal end to record respiration, and a rubber balloon connected to a rubber bulb and a mercury manometer was introduced into its cranial end for stimulating the mechanoreceptors of the upper respiratory tract. The blood pressure was recorded in the carotid artery. The electrocardiogram (ECG) was recorded on a type EKN-4 electrocardiograph in standard lead II, by means of needle electrodes. On each ECG all the intervals and waves were counted before and during stimulation and the statistical significance of the changes in all the components of the ECG in each case arising as a result of stimulation was determined separately. Only statistically significant (according to Student's criterion) reactions of the heart were considered. After each experiment the heart was investigated histologically.

EXPERIMENTAL RESULTS

In six cats multiple granulomas or foci of infiltration of proliferating connective-tissue cells and lymphoid elements were observed in the myocardium, intermuscular and perivascular in their localization. These changes were a sign of productive focal (granulomatous) myocarditis (Fig. 1). In eight animals a few small collections of proliferative connective-tissue cells or histiocytes were present between the muscle fibers of the myocardium. In

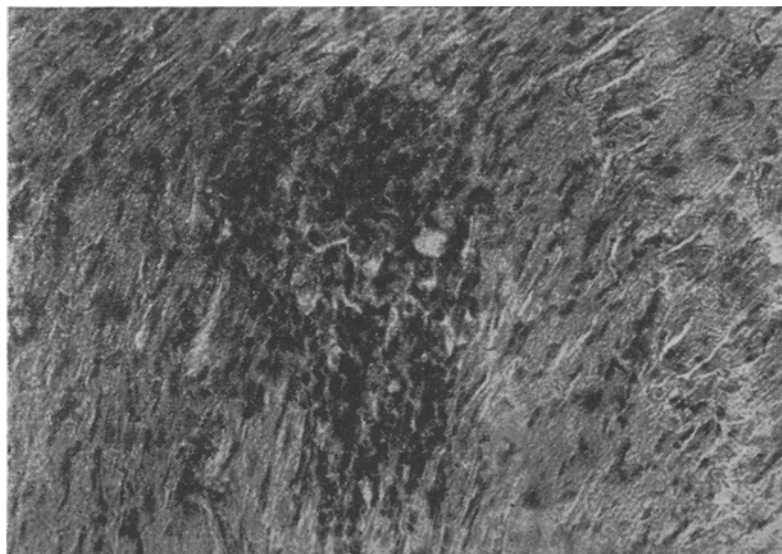


Fig. 1. Productive myocarditis in cats. A granuloma of lymphoid and connective-tissue cells is seen between the muscle fibers (experiment on June 26, 1962). Stained with hematoxylin-eosin. Objective 20, ocular 15.

some of these animals congestion was found, with cloudy swelling of the muscle fibers, an indication of a moderately severe, mainly productive myocarditis. In seven cats no changes were seen in the myocardium apart from a very moderate proliferation of the connective-tissue cells seen in occasional sections.

In healthy cats, according to previous investigations [9, 10], the heart rate was 118-176 beats/min ($M = 153$, $\sigma = \pm 19.77$). Stimulation of the mechanoreceptors of the sub- and epiglottic spaces of the larynx and pharynx slowed the heart rate by 6-12 beats/min, or 3-11% of its original level. The conductivity and contractility of the myocardium were unchanged. The thresholds of stimulation of the mechanoreceptors of the sub- and epiglottic spaces of the larynx and pharynx, in relation to the reaction of the heart rate, were 100, 60, and 40 mm Hg respectively. An increase in the intensity of stimulation sometimes caused a more marked slowing of the rhythm (by 6-12 beats/min).

In some cases the voltage of the waves was slightly reduced on the ECG of the cats with myocarditis; in three cases a negative T wave was present and in one case depression of the ST interval. The heart rate in the cats without morphological changes in the myocardium was essentially indistinguishable from this index in the animals with moderate and severe lesions, and the mean value for the groups was 150-180 beats/min.

Stimulation of the mechanoreceptors of the sub- and epiglottic spaces of the larynx and of the pharynx in most cases caused a slowing of the heart rate by 18-24, and in some cases by as many as 42-78, beats/min (Fig. 2). The most marked slowing of the heart rate took place in all the cats in response to stimulation of the mechanoreceptors of the epiglottic space of the larynx. No connection was noted between the original heart rate and the degree by which it was slowed during stimulation.

In the cats with obvious changes in the myocardium, the reaction of the heart rate to stimulation of the mechanoreceptors of the larynx and pharynx was less marked than in the animals without these changes. In the former, stimulation of the mechanoreceptors of the subglottic space of the larynx with an intensity of 20 mm Hg above threshold caused a mean slowing of the original rate of 6.3% (2.65-14.23%); during stimulation of the mechanoreceptors of the epiglottic space of the larynx the heart rate was slowed by 13.77% (6.06-22.22); and during stimulation of the mechanoreceptors of the pharynx—by 6.95% (3.06-12.12%). In the cats without morphological changes in the myocardium the same stimulation of the mechanoreceptors of the subglottic space of the larynx slowed the heart rate on the average by 17.7% (3.7-46.42%); stimulation of the mechanoreceptors of the epiglottic space of the larynx, by 19.1% (7.69-42.3%); and the pharynx, by 14.5% (4.3-40.9%).

In the experimental cats, in contrast to the healthy animals and the cats with tuberculosis of the larynx, during stimulation of the mechanoreceptors of the larynx and pharynx, in some cases statistically significant changes

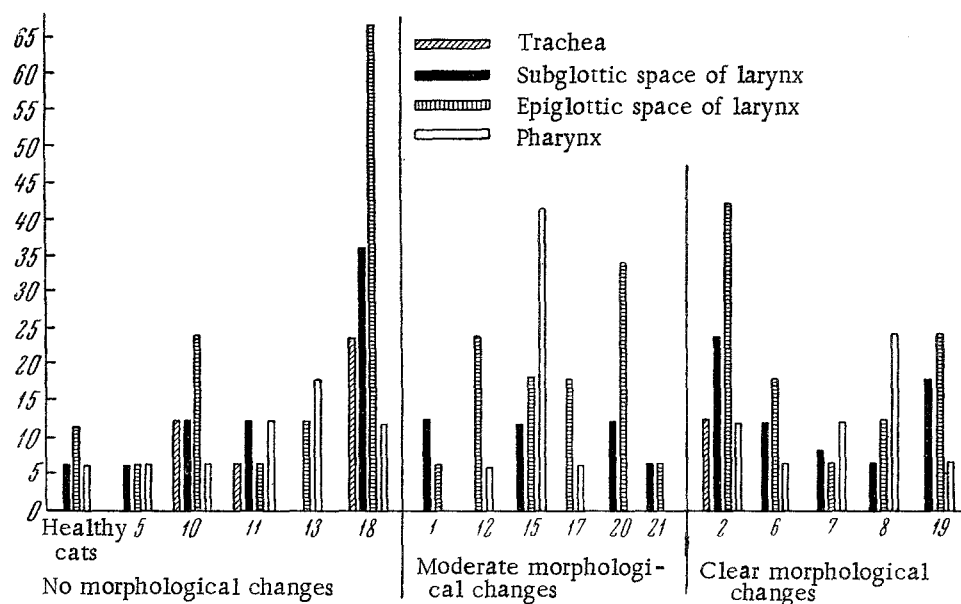


Fig. 2. Changes in magnitude of the reaction of the heart rate (slowing) to superthreshold stimulation of equal strength of the mechanoreceptors of various parts of the upper respiratory tract (20 mm Hg above the corresponding threshold of stimulation) in experimental myocarditis. Along the axis of abscissas—cat No.; along the axis of ordinates—degree of slowing of the heart rate (beats/min).

developed in the conductivity of contractility of the heart muscle. Of the 7 cats without changes in the myocardium, in 4 in response to stimulation of the mechanoreceptors of the larynx and pharynx, on some occasions a decrease or increase in the amplitude of the P wave by 0.2-0.61 mm was observed, in 3 cats—a decrease or increase in the amplitude of the P wave by 0.3-1.22 mm, in 1 cat—a decrease in the T wave by 0.25 mm, in 1 cat—lengthening of the PQ interval by 0.01 sec, and in 1—lengthening of the QT interval by 0.02-0.03 sec. The most marked changes were found in the ECG during stimulation of the mechanoreceptors of the epiglottic space of the larynx in cat No. 10. Besides slowing of the heart rate by 24-42 beats/min, extrasystoles developed, arising from the middle of the atrio-ventricular node. In this case, stimulation caused a displacement of the pacemaker of the heart and brought to light a heterotopic focus of increased excitability of the myocardium.

Of the 8 cats with moderately severe signs of myocarditis, during stimulation of the mechanoreceptors of the larynx and pharynx in some cases in 1 cat the P wave was increased in amplitude by 0.39 mm, in 3—the R wave was decreased or increased by 0.25-0.8 mm, in 1—the T wave was increased by 0.23 mm, and in 1 cat the PQ interval was lengthened by 0.03 sec and the QT interval by 0.02-0.03 sec.

Of the 6 cats with obvious signs of myocarditis, during stimulation there was sometimes a decrease or increase in the P wave in 2 cats by 0.18-0.42 mm, in 3—an increase in the P wave by 0.46-1 mm, in 1—a decrease in the T wave by 0.48 mm, in 3 cats the PQ interval was lengthened by 0.005-0.008 sec, and in 2 cats the QT interval was lengthened by 0.007-0.015 sec. In one case, during stimulation of the mechanoreceptors of the pharynx, a negative T wave developed, which was absent before stimulation. These results show that a change in the conductivity and excitability of the myocardium took place more often in the cats with an obvious lesion of the myocardium.

In 1 cat with marked changes in the myocardium, and in 3 cats without changes, stimulation of the mechanoreceptors of the trachea caused a slowing of the heart rate by 6-12 beats/min, which was never observed in the healthy cats and in the animals with tuberculosis of the larynx [9, 10].

The thresholds of stimulation of the mechanoreceptors of the larynx and pharynx in the cats with marked myocarditis were almost the same as in the healthy cats, while in the animals without morphological changes in the myocardium they were slightly lower.

The reflexes from the mechanoreceptors of the investigated areas of the upper respiratory tract on the respiration and blood pressure, and also the thresholds of stimulation of these receptors, were the same in all the cats as in

healthy animals. This finding evidently indicates that the effector link, when damaged by the pathological process, reacts differently in the course of the reflex act from the intact effector links.

The intensification of the reaction of the heart to stimulation of the mechanoreceptors of the larynx and pharynx of the cats with no morphological changes in the myocardium suggests that the administration of caffeine and adrenalin, while not causing organic changes in the heart, nevertheless modified its reactivity.

The absence of changes in cardiac activity in many of the cats before stimulation of the mechanoreceptors of the larynx and pharynx and the considerable changes taking place during stimulation confirm the results indicating that interoceptive stimulation may detect latent cardiac pathology [13].

The differences in the character of the reflex changes in cardiac activity during stimulation of the mechanoreceptors of the upper respiratory tract of the cats with tuberculosis of the larynx [10] and of cats with myocarditis are evidently dependent on which part of the reflex arc—afferent or efferent—is influenced by the pathological process.

LITERATURE CITED

1. L. Ya. Balonov, Conditioned-Reflex Regulation of Human Cardiac Activity [in Russian], Moscow-Leningrad (1959).
2. K. M. Bykov and V. N. Chernigovskii, Byull. éksper. biol. 29, No. 1, 12 (1950).
3. I. E. Ganelina, Reflex Influences from the Chemoceptors of the Gastrointestinal Tract on the Heart, Especially in Atherosclerosis. Doctorate dissertation, Leningrad (1957).
4. I. E. Ganelina, Byull. éksper. biol., No. 1, 32 (1957).
5. R. A. Zasosov, I. I. Isakov, and I. B. Soldatov, Trudy Voen.-morsk. med. Akad. (Leningrad), 39, 66 (1952).
6. K. N. Karpenko, Trudy Voen.-morsk. med. Akad. (Leningrad), 42, 157 (1948).
7. V. S. Livshits, Byull. éksper. biol., 31, No. 5, 14 (1951).
8. G. Ya. Priima, Fiziol. zh. SSSR, No. 10, 946 (1958).
9. R. A. Fel'berbaum, Fiziol. zh. SSSR, No. 6, 736 (1963).
10. R. A. Fel'berbaum, Byull. éksper. biol., No. 7, 46 (1963).
11. S. I. Frankshtein, Disturbance and Restoration of the Function of the Injured Organ [in Russian], Moscow (1948).
12. V. V. Frol'kis, Reflex Regulation of the Activity of the Cardiovascular System [in Russian], Kiev (1959).
13. V. N. Chernigovskii, Trudy Voen.-morsk. med. Akad. (Leningrad), 24, 35 (1950).
14. V. N. Chernigovskii, In book: I. P. Pavlov's Theory in Theoretical and Practical Medicine [in Russian], No. 2, Moscow (1953), p. 357.
15. N. Chistovich, Ezhenedel'n. klin. gazeta, No. 28, 555 (1887).
16. M. Eskat, L. Lareng, and J. Cistas, Rev. Laryng. (Bordeaux), 83 (1962), p. 1012.
17. J. Fine, J. Laryng., 74 (1960), p. 333.
18. P. Forester and J. Nyboer, Am. J. Physiol., 183 (1955), p. 149.
19. I. Jimenez-Vargas, J. Flörer, and C. Gomez-Lavin, Rev. esp. Fisiol., 18 (1962), p. 139.
20. C. Oppenheimer and O. Weiss, Gröndriss der Physiologie. Leipzig, T. 2, S. 95 (1922).
21. J. Troquet and J. Lecomte, C. R. Soc. Biol., 154 (1960), p. 854.

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 the first issue of this year.
