# THE EFFECT OF A PHYSICAL LOAD ON THE FREQUENCY OF CONTRACTION OF A HEART DEPRIVED OF SPINAL SENSORY INNERVATION

(UDC 612.176.4-06:612.178.1/.2)

G. M. Karavaev

Department of Normal Physiology (Head, Professor M. G. Zaikina),
Yaroslavskii Medical Institute

Presented by Active Member AMN SSSR V. V. Parin
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 59, No. 1
pp. 10-14, January, 1965
Original article submitted September 24, 1963

From morphological studies it is known that the sensory innervation of the heart arises from the stellate ganglion as well as the dorsal root ganglia of the upper thoracic segments [6, 10, 12]; extirpation of the latter influences the condition of the heart [1, 4, 8]. It has also been established that under these conditions there is a reduction of both sympathetic and vagal tone [2]. These results suggest that the sensory fibers entering the cord via the dorsal roots must be functionally related to the vagus and sympathetic nerves and must be involved in the regulation of cardiac activity.

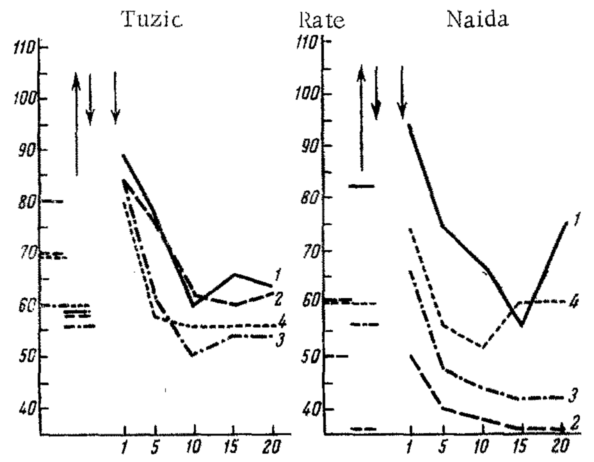


Fig. 1. Change in heart rate after a measured amount of physical work under conditions of blockade of the sympathetic nerves by dihydroergotoxin. 1) Change in heart rate after a calculated amount of physical work, and before de-afferentation; 2) in the first month after de-afferentation; 3) at the 5th month; 4) at the 8th month. An arrow pointing upwards indicates the injection of dihydroergotoxin, an arrow pointing downwards indicates physical exercise.

We have set out to determine the influence of a measured amount of physical work on the rate of contraction of de-afferented heart. According to many authors the increase in heart rate which follows muscular activity is due to reduction of vagal tone as well as to an increased sympathetic influence [5, 13]. In this connection it seemed worth while to study the extent to which the vagal and sympathetic nerves were involved in bringing about the increased rate by making observations of the effect of a measured amount of muscular work on an animal whose heart was deprived of spinal sensory innervation.

## EXPERIMENTAL METHOD

The experiments were carried out on 5 male or female adult dogs from which the dorsal root ganglia were removed bilaterally from segments $T_1$-$T_5$. The animals were made to run for 20 min on a moving track whose speed was set at 6 km per h. Records were made of the ECG (3 standard leads) before and immediately after the animals had run, and the measurements were continued till the original heart rate had been restored.

To study the part played by the vagal and sympathetic nerves in causing the increase in heart rate in response to physical work we blocked the autonomic innervation by use of atropine and dihydroergotoxin.

TABLE 1. Effect of a Physical Load on Heart Rate Before and After Extirpation of the Dorsal Root Ganglia

| Heart rate (no. of beats per min) | Period (in months) elapsing after operation | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | before running | after running | before running | after running | before running | after running | before running | after running | before running | after running |
| | Naida | | Belki | | Busina | | Mulya | | Tuzik | |
| Before operation | | | | | | | | | | |
| | 110 | 155 | 103 | 148 | 80 | 107 | 74 | 102 | 86 | 122 |
| After operation | | | | | | | | | | |
| 1-first month | 60 | 78 | 58 | 80 | 59 | 88 | 62 | 90 | 70 | 100 |
| 2- " " | 60 | 77 | 70 | 87 | 60 | 92 | 57 | 82 | 73 | 102 |
| 3- " " | 60 | 80 | 64 | 82 | 60 | 90 | 68 | 87 | 80 | 104 |
| 4- " " | 70 | 112 | 60 | 90 | 60 | 86 | 62 | 80 | 66 | 98 |
| 5- " " | 60 | 80 | 60 | 87 | 60 | 76 | 60 | 79 | 65 | 102 |
| 6- " " | 58 | 88 | 60 | 87 | 60 | 74 | 60 | 80 | — | — |
| 7- " " | 59 | 82 | — | — | — | — | 60 | 84 | — | — |
| 8- " " | 60 | 90 | 68 | 100 | 60 | 80 | 60 | 84 | 68 | 88 |
| 9- " " | 58 | 80 | 62 | 82 | 60 | 80 | 58 | 76 | 68 | 92 |
| 10- " " | 60 | 78 | 62 | 82 | 64 | 76 | 58 | 80 | — | — |

TABLE 2. Effect of Physical Exercise on Heart Rate Before and After Extirpation of the Dorsal Root Ganglia and after Blockade of the Sympathetic Nerves with Dihydroergotoxin

| Heart rate (beats per min) | Time between operation and investigation | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | before running | after running | before running | after running | before running | after running | before running | after running | before running | after running |
| | Naida | | Belki | | Busina | | Mulya | | Tuzik | |
| Before operation | | | | | | | | | | |
| | 82 | 95 | 63 | 69 | 60 | 80 | 63 | 84 | 58 | 90 |
| After operation | | | | | | | | | | |
| 1-first month | 36 | 50 | 46 | 50 | 52 | 62 | 58 | 70 | 58 | 84 |
| 2- " " | 44 | 60 | — | — | — | — | 56 | 68 | 60 | 84 |
| 3- " " | — | — | 42 | 58 | 60 | 68 | 56 | 60 | 60 | 84 |
| 4- " " | 60 | 70 | 50 | 60 | 54 | 60 | 58 | 64 | 60 | 80 |
| 5- " " | 56 | 66 | 56 | 68 | 50 | 64 | 52 | 84 | 56 | 84 |
| 6- " " | — | — | 48 | 64 | 52 | 80 | — | — | 62 | 80 |
| 7- " " | 54 | 68 | — | — | — | — | 48 | 60 | 56 | 72 |
| 8- " " | 60 | 74 | 58 | 90 | 50 | 60 | 52 | 68 | 60 | 80 |
| 9- " " | 50 | 60 | 46 | 60 | 52 | 60 | 64 | 72 | 52 | 76 |
| 10- " " | 50 | 60 | 54 | 76 | 54 | 60 | 52 | 72 | — | — |

These drugs were given intravenously; 0.2 mg/kg atropine over a period of 10 min before running on the track, and 0.05 mg/kg dihydroergotoxin 20 min before the exercise.

Experiments on the effect of exercise either with or without elimination of the autonomic nerves were carried out before and after extirpation of the dorsal root ganglia. Observations were made on the dogs for 9-10 months after the operation.

## EXPERIMENTAL RESULTS

Before extirpation of the dorsal root ganglia in all the animals exercise caused an increase in heart rate. In

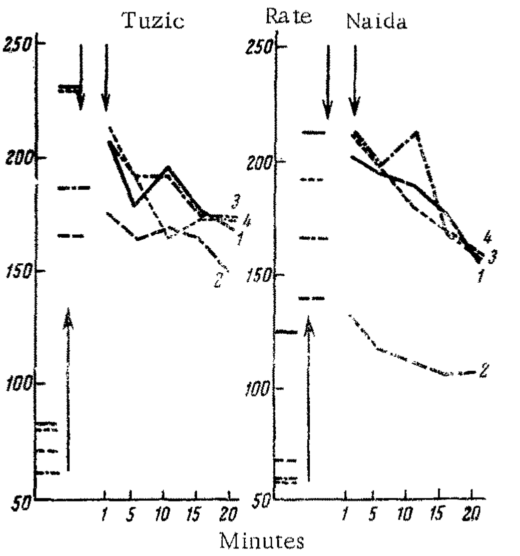


Fig. 2. Changes in the heart rate after a calculated amount of physical exercise and after blockade of the vagi by atropine. An arrow indicates injection of atropine; an arrow downwards indicates physical exercise. Other indications as in Fig. 1.

Tuzik 1 min after running on the moving track the heart rate increased from 86 to 122, and in Naida it rose from 110 to 115 per min. During the next 20 min the rate returned to normal, but in Naida, Belka, and Mulya it fell below the original rate. These results may be attributed to an enhanced vagal tone operating as part of a mechanism of recovery of heart rate after exercise [3, 5, 7, 11].

After extirpation of the dorsal root ganglia in all the dogs the heart rate fell by 20-25%, and physical exercise caused a smaller increase in heart rate than it did in normal animals (Table 1).

Before the operation 20 min after intravenous injection of dihydroergotoxin in all dogs the heart rate fell by 15-20 beats per min (Fig. 1), a results which indicates the existence of sympathetic tone before the injection of the drug.

After blockade of the sympathetic nerves by dihydroergotoxin a given amount of muscular work caused a smaller increase in cardiac contraction than previously. Before the operation in Tuzik the intravenous injection of dihydroergotoxin caused the heart rate to fall from 80 to 58 per min (see Fig. 1). In this condition 20 min of physical exercise caused the heart rate to increase to 90 per min. In Naida the initial heart rate before the injection of dihydroergotoxin was higher. After blockade of the sympathetic nerves the rate fell from 110 to 82 per min. Under the influence of physical exercise the heart rate rose by no more than 13 beats per min. In Belka, Busina, and Mylya, as can be seen from Table 2 the intravenous injection of dihydroergotoxin caused the heart rate to fall to 60-63 beats per min. However, the dogs responded differently to the influence of physical exercise. In Belka running on the track caused an increase in heart rate of 6 beats per min, whereas in Mylya and Busina the increase was 20 beats per min. These results indicate that activation of the sympathetic system plays an important part in the mechanism of increase of heart rate. At the present time many authors emphasize the special part played by the sympathetic innervation in the cardiac response to physical exercise [9, 14-18].

In the first months after the operation in all dogs the heart rate was reduced by dihydroergotoxin, but the decrease was less than before the extirpation (see Fig. 1). Under these circumstances in most cases physical exercise led to an increase in heart rate, though the increase was smaller than before the operation (Naida and Belka). In the subsequent months in these animals the cardiac response to physical exercise after elimination of sympathetic action increased considerably. In Tuzik, after de-afferentation and blockade of the sympathetic nerves, physical exercise would be expected to increase the heart rate almost to the level obtaining before the operation. Under these conditions Mulya and Busina reacted to physical exercise by a greater increase in heart rate than did Naida and Belka, but the increase was less than in Tuzik (Table 2).

The increase in heart rate due to physical exercise when the sympathetic innervation had been eliminated was due to reduction in vagal tone both in the operated and unoperated animals.

Intravenous atropine caused a marked increase in heart rate up to 220-300 per min.

When the vagi were blocked with atropine physical exercise caused no further increase in heart rate; on the contrary after the exercise there was even some reduction in rate (Fig. 2).

In the first month after operation injection of atropine caused a smaller increase in heart rate than it had done before the operation; subsequently the reaction to atropine recovered. After de-afferentation and after blockade of the vagi with atropine in most cases physical exercise caused no further increase in heart rate, or if it did the increase was very small. Probably the different response to physical exercise among the different dogs depended upon the relationships between parasympathetic and sympathetic influences on the heart.

The greatest increase in heart rate in response to exercise occurred in dogs in which sympathetic influence was very marked. In such animals (Naida and Belka) de-afferentation caused a decrease of sympathetic tone. Consequently, physical exercise had less influence on heart rate.

In Tuzik, Mulya, and Busina in which the sympathetic tone was weaker the increase in heart rate due to physical exercise was brought about chiefly through reduction in vagal tone. In these animals reduction of sympathetic tone had less influence on the effect of physical exercise on heart rate.

At the same time injection of atropine showed that after extirpation of the dorsal root ganglia, especially in the first few months, there was a reduction in vagal tone. This was shown by the fact that intravenous injection of atropine caused a smaller increase in heart rate than it had done before the operation.

The results obtained show that after extirpation of the dorsal root ganglia from the upper thoracic segments there is a change in the functional condition of the cardiac sympathetic and parasympathetic centers. This difference shows up more clearly under conditions of physical exercise.

## SUMMARY

Long-term experiments were performed on dogs. A study was made of the effect produced by physical exercise on heart rate before and after extirpation of the spinal ganglia from the 5 upper thoracic segments. The vagi were blocked with atropine, and the sympathetic nerves with dihydroergotoxin. A decrease in heart rate occurred after the extirpation. The de-afferented heart showed a smaller increase in rate in response to physical exercise.

Removal of the dorsal root ganglia was followed by a disturbance of vagal tone and by a more marked disturbance of sympathetic tone.

## LITERATURE CITED

1. M. G. Zaikina, In book: Collected scientific works of the Yaroslavskii Medical Institute (1957), No. 15, p. 57.
2. M. G. Zaikina, G. M. Karavaev, and E. I. Kuznetsov, In book: Transactions of the 3rd Povolzhskii Conference of physiology, biochemistry, and pharmacology. Gor'kii (1963), p. 65.
3. I. V. Murav'ev, In book: Problems of the physiology and pathology of vascular tone [in Russian], Kiev (1961), p. 244.
4. A. A. Nikitin, Transactions of the Military medical academy, Leningrad (1948), Vol. 42, p. 245.
5. P. D. Olefirenko, Med. biol. zh. (1930), Vol. 6, Nos. 1-2, p. 84.
6. E. K. Plechkova, In book: The morphology of the sensory innervation of the internal organs [in Russian], Moscow (1947), p. 46.
7. I. A. Peimer, Transactions of the All-Union society of physiology, biochemistry, and pharmacology (1956), Vol. 3, p. 84.
8. Z. I. Sobieva, G. V. Chernysheva, N. B. Lavrent'eva, E. B. Khishman, L. S. Ul'yaninskii, A. A. Gurvich, and V. F. Eremeev, In book: The physiology and pathology of the circulation [in Russian], Moscow (1962), p. 51.
9. V. S. Farfel', A course in the physiology of man [in Russian], Moscow (1948).
10. A. Ya. Khabarova, The afferent innervation of the heart [in Russian], Moscow, Leningrad (1961).
11. A. M. Cheshkov, A year and 7 months in the life of a dog after simultaneous division of both vagi in the neck. Dissertation, St. Petersburg (1902).
12. I. A. Chervova, Dokl. AN SSSR (1955), Vol. 103, No. 2, p. 321.
13. F. A. Bainbridge, The physiology of muscular activity [in Russian] Moscow, Leningrad (1927).
14. S. Bevegard, Acta physiol. scand. (1963), Vol. 57, p. 61.
15. A. Rosen (1961), Vol. 52, p. 291.
16. R. F. Rushmer, O. Smith, and D. Franklin, Circulat. Res. (1959), Vol. 7, p. 602.
17. R. F. Rushmer, O. A. Smith, Jr., and E. P. Lasher, Physiol. Rev. (1960), Vol. 40, p. Supple. N 4, p. 27.
18. S. C. Wang and C. V. Chai, Am. J. Physiol. (1962), Vol. 202, p. 31.