The Cardio-Accelerator Fibers in the Vagus Nerve.

By

Shigeo Okinaka, Kiku Nakao, Masao Ikeda and Kazuo Shizume.

(From III Medical Clinic of Prof. S. Okinaka, School of Medicine, University of Tokyo.)

(Received for publication, May 29, 1951)

Although some investigators claim the existence of the cardioaccelerator fibers which emerge from the medulla oblongata through the vagus nerve, this problem does not seem to be clearly decided.

Rutherford (1869)¹, Schiff (1879)² and Tulgan (1923)³ observed an increase in heart rate on electrical stimulation of the peripheral end of the cut cervical vagus after paralysis of the cardio-inhibitory endings with atropine.

On the other hand, Boehm (1875)⁴, Rossbach (1875)⁵ and Hering (1924)⁶ denied that stimulation of the vagus nerve after atropine could accelerate the heart rate. Then Jourdan and Nowak (1936)⁷ demonstrated the acceleration of the heart on electrical stimulation of the peripheral end of the cut vagus rootlets intracranially after application of atropine in the dogs, and Kabat (1939)⁸ demonstrated the vagal cardioaccelerator fibers on electrical stimulation of the cervical vago-sympathetic trunk and of the vagus rootlets intracranially in the dogs after atropine. More recently Pannier (1946)⁹ found vagal cardioaccelerator fibers in 80 per cent of the dogs.

These experiments besides Pannier’s, of which the details were not clear, are undertaken by using atropine in order to eliminate the cardioinhibitory action.

Okinaka and his collaborators (1942)¹⁰ confirmed that the cardioinhibitory fibers of the vagus nerve of the dog were localized in the caudal portion of the myelencephalic rootlets of the accessory nerve (in Fig. 1, AH and the caudal part of AM). If the probable vagal accelerator fibers run through the other portion of the rootlets, they will be demonstrated by eliminating the cardioinhibitory action by cutting the portion of the rootlets which contain the cardioinhibitory fibers without the use of atropine. This assumption was considered and following experiments were undertaken.
Nineteen dogs were used. Most of them were anesthetized with morphine hydrochloride which was injected subcutaneously in dosis of 6–8 mg per kg. of body weight 30–60 minutes before the stimulation. Blood pressure and heart rate were recorded from the femoral artery by means of a mercury manometer on a smoked kymograph drum. The Du Bois-Reymond induction coil was used to provide electrical stimulation. The duration of stimulation were 10–20 seconds. In 7 cases electrocardiographs were recorded from the right upper limb and the left lower limb at the same time. The experiments were carried out in the following manner.

A) Electrical stimulation of the vagus rootlets V (see Fig. 1) intracranially.

Right vagus rootlets were cut at the junction of the medulla oblongata and the peripheral end of the cut rootlets V was stimulated. Four dogs were used. In one case, because of bleeding, stimulation was impossible. And three cases were successfully stimulated, but the cardiac acceleration was not demonstrated in any case. On the other hand bradycardia was often observed presumably by the spread of stimulation to rootlets AM and AH.

B) Electrical stimulation of the vagus rootlets V, two weeks after the section of rootlets AM and AH, which contain the cardioinhibitory fibers.

In order to eliminate the cardio-inhibitory action, rootlets AM and AH were cut intracranially and two weeks later, when the cardioinhibitory fibers degenerated, the vagus rootlets V was cut and the peripheral end was stimulated intracranially. Two dogs were used. One died on the
first post-operative day and in the other slight cardiac acceleration was demonstrated by stimulation.

(The following figures indicate numbers of heart beats in periods of 5 seconds).

11.0 9.5 12.0 12.0 14.0 14.0 13.0 11.0

stimulation (10 sec.)

In this experiment cardiac inhibition was not observed because of the degeneration of the cardio-inhibitory fibers. But the stimulation of the rootlets V two weeks later intracranially is difficult because of bleeding from the granulation tissue which developed from the previous operation. Consequently other experiments were undertaken.

C) Electrical stimulation of the cervical vago-sympathetic trunk two weeks after the section of rootlets AM and AH which contain the cardio-inhibitory fibers.

As in the previous experiments, rootlets AM and AH were cut intracranially and two weeks later, when the cardio-inhibitory fibers degenerated, the cervical vago-sympathetic trunk was cut at the level about 3 cm peripheral from the nodose ganglion. And the peripheral part was stimulated near the cut end. Five dogs were used. In three of them moderate cardiac acceleration was observed as follows.

(The following figures indicate numbers of heart beats in periods of 5 seconds).

16.0 16.0 16.0 17.0 19.0 18.0 16.0 16.0

stimulation (10 sec.)

4.0 4.5 5.0 4.5 4.5 6.5 6.5 5.0 4.5

stimulation (10 sec.)

8.0 8.0 8.0 8.5 9.5 9.5 8.5 8.5 9.5 8.0

stimulation (15 sec.)

Fig. 2 and Fig. 3 also show graphically results of these experiments. In one of the remaining two, cardiac acceleration was observed by stimulation after the extirpation of the right stellate ganglion (Fig. 4).

D) Electrical stimulation of the cervical vago-sympathetic trunk two weeks after the section of rootlets AM and AH on the right side and the extirpation of the right stellate ganglion.

In order to heighten the tonus of the vagal cardio-accelerators, and also to avoid possible stimulation of cardio-accelerator fibers from the
spinal cord, the right stellate ganglion was extirpated together with section of the cardio-inhibitory rootlets. Two weeks later the cervical vagosympathetic trunk was cut and stimulated at the same level as in the previous experiments. Eight dogs were used. One died on the first postoperative day and stimulation was performed in seven dogs. Cardiac acceleration was demonstrated in three. In one of these, the acceleration was especially marked, before stimulation 4.5 beats per 5 seconds, after stimulation 10 per 5 seconds (Fig. 5).

Fig. 6 and Fig. 7 also show the results in this experiment. In two of the remaining four, cardiac acceleration was observed only after the in-
Injection of atropine. In these cases the existence of rootlets AM was revealed in the autopsy which was made after the experiment. Therefore cardiac acceleration occurring only after atropine seems to be due to the remaining cardio-inhibitory fibers in the rootlets AM.

In these 10 experiments in which the vagal cardio-accelerator fibers were demonstrated, the stronger the stimulation, the clearer was the acceleration, and some latent period was observed between the onset of stimulation and the appearance of its effect. The effect was most marked 5 to 10 seconds after the onset of the stimulation, and it continued from 5
to 30 seconds after the end of the stimulation.

With the acceleration of the heart, slight elevation of the blood pressure and the decrease of the pulse-amplitude were noted. In four cases of these experiments electrocardiographs were recorded and in some cases lowering of the T wave with the decrease of RR interval was observed by stimulation (Fig. 8).

![Change in electrocardiograph by stimulation of the vagal cardio-accelerator fibers](image)

**CONCLUSIONS.**

1. Efferent vagal cardio-accelerator fibers were demonstrated by section of the cardio-inhibitory fibers in the myelencephalic rootlets of the accessory nerve followed by stimulation of the cut myelencephalic vagus rootlets or peripheral end of the cut cervical vagus nerve.

2. These vagal cardio-accelerator fibers were better demonstrated by the extirpation of the stellate ganglion on the same side.

**References.**

2) Schiff, M.; Pflüger's Arch., 1878, 18, 172.
4) Boehm, R., Arch. f. exper. Pathol. u. Pharmakol., 1875, 4, 255.
5) Rossbach, M. J., Pflüger's Arch., 1875, 18, 383.