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On the Effect of Vagus Stimulation upon the Conductivity of Tortoise Heart.

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Abstract

It was found, from the curves of mechanical registration that the precise determination of atrio-ventricular interval and the time interval from the beginning to the summit of ventricular contractions cannot be accurately measured. But it was clearly shown that the height of auricular contractions is obviously reduced by vagus stimulation as well as by the application of acethylcholin, which is generally known as "Vagusstoff" while on the other hand the ventricular contractions are not affected. It is considered, therefore, from the results obtained, that the acethylcholin like substance is produced from the endings of vagus nerve as a conseqence of its activity through stimulation, and that this substance causes a decrease in the height of auricular contractions. Examining the electrocardiogramm, it clearly showed a marked prolongation of the atrio-ventricular interval, and in the last three curves it failed to record the ventricular contraction after each P-spike. Hence it is considered that vagus stimulation affects the conductivity of atrio-ventricular bundle and causes diminution of its conductivity. The results above cited, agree with those of the former investigators. But with regard to the duration of the electrical response of the ventricle, it was found contrary for the vagus stimulation has no effect upon the function of ventricular muscle. From the results obtained, the following conclusions may be made. 1. The vagus stimulation affects the negative inotropic only on auricular contraction. 2. The vagus stimulation affects the negative dromotropic on the conductivity of atrio-ventricular bundle. 3. The vagus stimulation has no effect upon the function of ventricular muscle.

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On the Effect of Vagus Stimulation upon the Conductivity of Tortoise Heart.

By

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Introduction.

There are numerous reports found concerning the inhibitory effect of vagus stimulation on the conductivity of heart muscle but their results do not agree. Consequently the author performed the following experiments from the critical point of view as to whether the results of former investigators and his own would show good agreement or not, employing *Siemen*'s electrocardiograph.

Reviewing the results of experiments by *Einthoven*[1], *D. Dale* and *G.R. Mines*[3], *A. Samojloff*[2], *E. Schliephake*[4], *H.E. Hering*[5], *Rothberger* and *Winterberg*[6], *Robinson* and *Draper*[7], and *Herzog*[8], on human beings and animals, the P-R interval of electrocardiogramm was generally prolonged by vagus stimulation, and the first three above authors observed the shortning of the duration of the electrical response of the ventricle, while *Herzog* established the lengthening on the contrary.

Experimental.

A. Mechanical registration.

Throughout the experiment, tortoises of medium size were used. After the brain and spinal cord had been pitched, the carapace dissected, the pericardium opened and the heart was exposed. The auricle and ventricle were attached to two levers respectively which register the movement on a rotating smoked drum.

On the other hand, the vagus nerve was prepared in the neck

and provided for faradic stimulation. The inductorium of *Du Bois-Reymond*'s Type was used for stimulation, of which a primary coil was connected to an accumlator (2 volts) and a secondary coil leading off to the platinum electrodes.

Previously the liminal strength of the stimulation just to evoke the cessation of heart beats was determined, then rotating the smoked drum by (a) at high speed, by (b) at low speed, the vagus stimulation was applied during the suitable length of time until heart beats stopped after a certain numbers of beats.

The following measurements were made on the registered curves:

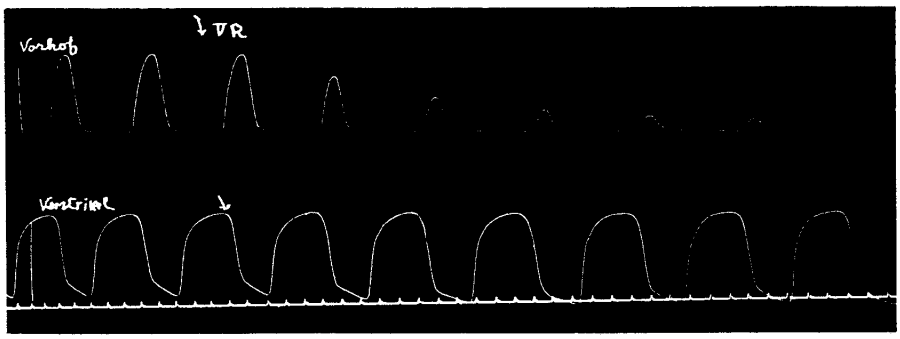
(a) The atrio-ventricular interval and interval from the beginning of ventricular contractions to its summit.

(b) The height of contractions of auricle and ventricle respectively.

The results are represented in the following figures 1 and 2.

(a).

Fig. 1.



Time signal, 1/5 sec.
Right vagus nerve was stimulated.
Strength of stimulation at the coil distance of 10.5 cm.

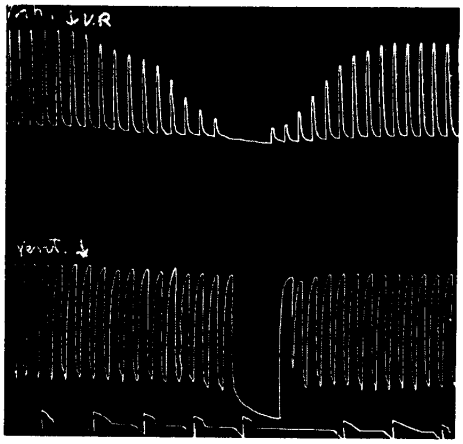
The upper curve seems to indicate that the atrio-ventricular interval and the duration of ventricular contractions do not alter by the stimulation of vagus nerve.

However it is considerably difficult to determine precisely by this method the beginnings and the summits of each contractions of each contractions of auricle as well as ventricle even when under slower rotation of smoked drum. Consequently the results obtained are so uncertain that no definite statement can be made.

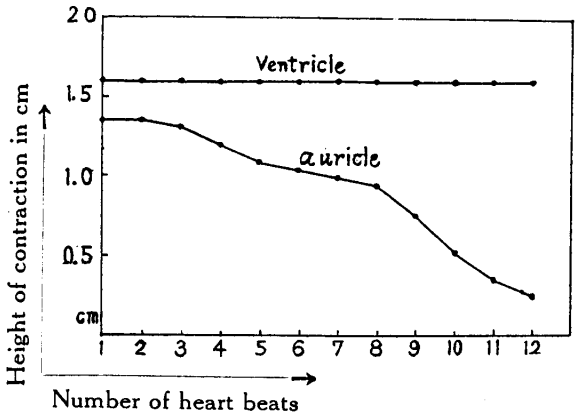
(b).

1. Vagus stimulation.

Fig. 2.



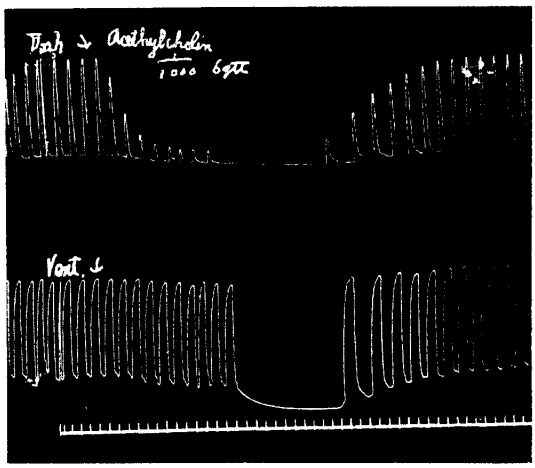
Time signal, 5 sec.
Right vagus was stimulated.
Strength of stimulation at the coil distance 10 cm.



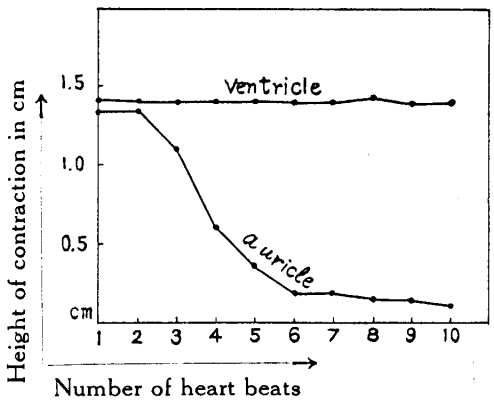
2. Influence of acethylcholin.

Some drops of acetnylcholin (1/1000) were applied instead of vagus stimulation.

Fig. 3.



Time signal 1 sec.



B. Electrocardiograph.

With the employment of *Siemen*'s electrocardiograph, the action current of tortoise heart was recorded as follows: one of the electrodes was placed on the basis of the right auricle and the other on the apex of the ventricle; and only the auricle was suspended through the cord to the recording lever. The electrocardiograph recorded after some normal beats, stimulation of vagus nerve began and continued to the cessation of heart beats.

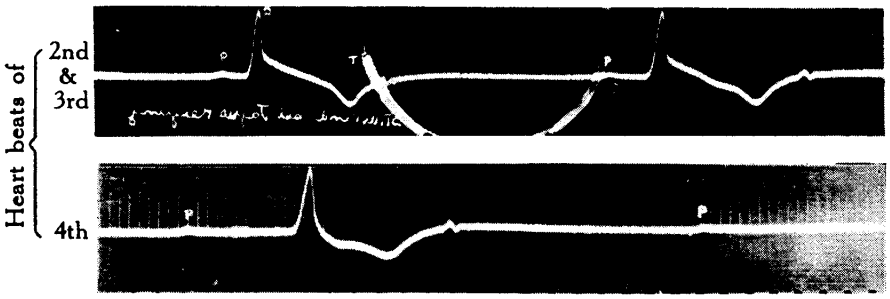
The following measurements were made on the curves obtained:

1. The duration of the atrio-ventricular interval; from the beginning of the auricular variation to the beginning of the ventricular variation on the electrocardiogramm.

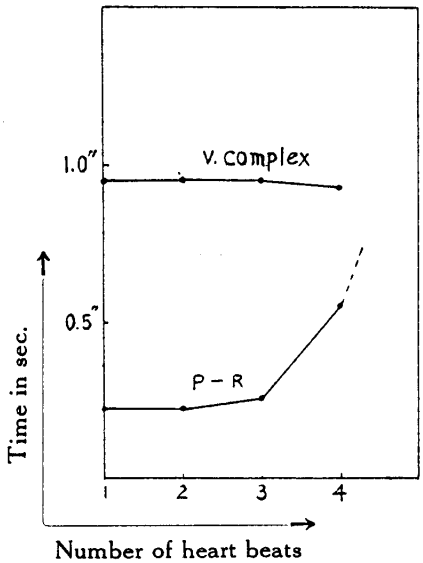
2. The duration of the electrical response of the ventricle. The results are represented in Figures 4, 5 and 6.

(1).

Fig. 4.



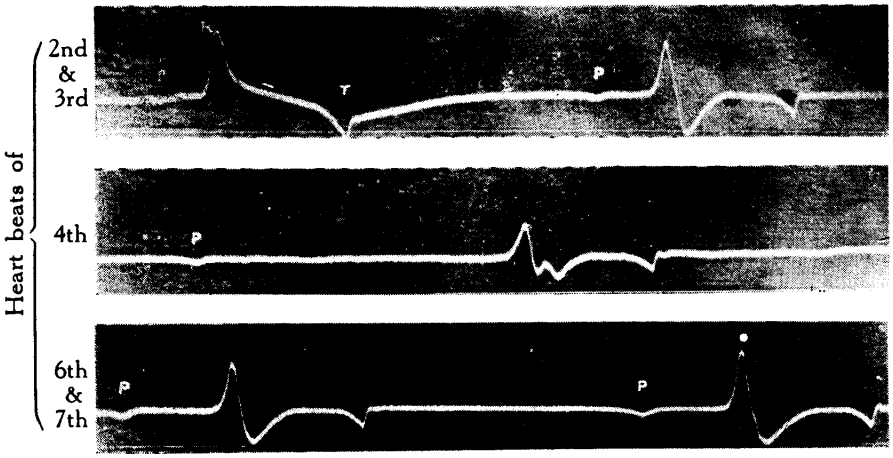
Time signal, 1/20 sec.



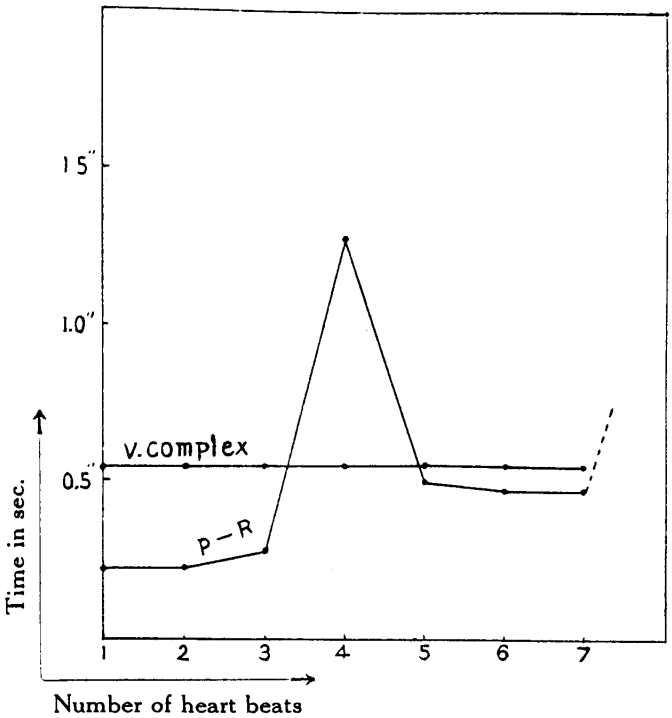
Left vagus nerve was stimulated. Strength of stimulation at the coil distance, 8.0 cm.

(2).

Fig. 5.



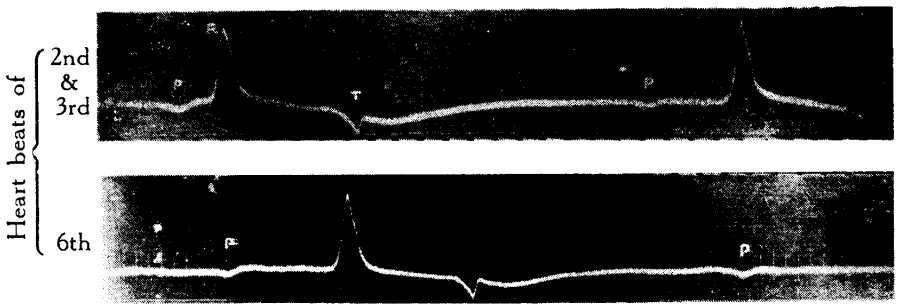
Time signal, 1/20 sec.



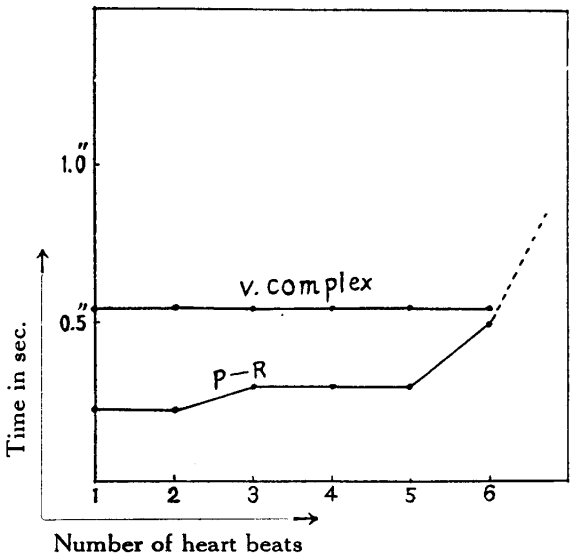
Right vagus nerve was stimulated.
Strength of stimulation at the coil distance, 12.5 cm.

(3).

Fig. 6.



Time signal, 1/20 sec.



Right vagus nerve was stimulated.
Strength of stimulation at the coil distance, 10 cm.

Summary and Conclusions.

It was found, from the curves of mechanical registration that the precise determination of atrio-ventricular interval and the time interval from the beginning to the summit of ventricular contractions cannot be accurately measured. But it was clearly shown that the height of auricular contractions is obviously reduced by vagus stimu-

lation as well as by the application of acethylcholin, which is generally known as "Vagusstoff" while on the other hand the ventricular contractions are not affected.

It is considered, therefore, from the results obtained, that the acethylcholin like substance is produced from the endings of vagus nerve as a conseqence of its activity through stimulation, and that this substance causes a decrease in the height of auricular contractions.

Examining the electrocardiogramm, it clearly showed a marked prolongation of the atrio-ventricular interval, and in the last three curves it failed to record the ventricular contraction after each P-spike. Hence it is considered that vagus stimulation affects the conductivity of atrio-ventricular bundle and causes diminution of its conductivity.

The results above cited, agree with those of the former investigators. But with regard to the duration of the electrical response of the ventricle, it was found contrary for the vagus stimulation has no effect upon the function of ventricular muscle.

From the results obtained, the following conclusions may be made.

1. The vagus stimulation affects the negative inotropic only on auricular contraction.

2. The vagus stimulation affects the negative dromotropic on the conductivity of atrio-ventricular bundle.

3. The vagus stimulation has no effect upon the function of ventricular muscle.

My sincere thanks are due to Prof. *S. Oinuma* for suggesting this investigation and also for his constant advices.

Literature.

[1] *Einthoven*, Pflüger's Archiv Bd. 122, S. 532, 1908. — [2] *A. Samojloff*, Ebenda Bd. 135, S. 460, 1910. — [3] *D. Dale* and *G. R. Mines*, J. of Physiol. Vol. 46, P. 319, 1913. — [4] *E. Schliephake*, Z. f. Biol. Bd. 82, S. 107, 1913. — [5] *H. E. Hering*, Pflüger's Archiv Bd. 127, S. 155, 1909. — [6] *Rothberger* and *Winterberg*, Ebenda Bd. 135, S. 506, 1910. — [7] *Robinson* and *Draper*, J. of exp. Med. Vol. 14, P. 224, 1911. — [8] *Herzog*, Deutsch. Archiv f. klin. Med. Bd. 105, S. 235, 1912.