

NOTE ON VAGUS STIMULATION OF THE ADRENALISED HEART. BY M. KURODA AND YAS KUNO.

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A RESEARCH on the nervous regulation of the heart was carried out by one of us (Y. K.) in conjunction with Dr Dittler in Leipzig in 1914, which will be published at the end of the war. In these experiments the statement made by Gourfein and Langley⁽¹⁾ etc., that the vagus is rendered inexcitable by the injection of adrenalin was confirmed. It is doubtful however whether this phenomenon is due to a direct effect of the adrenalin or to the increased intracardiac pressure caused by the adrenalin, for it is a well-known fact that stimulation of the vagus has no longer any effect when the intracardiac pressure rises considerably.

This question was investigated by Bessmertny⁽²⁾, who found that a preparation called "Emostasin" caused a very remarkable rise in the arterial pressure (even to 240 mm. Hg) unaccompanied by any diminution in the excitability of the vagus, while adrenalin (Parke-Davis) had the above-mentioned effect. He concluded from these results that the abolition of the excitability of the vagus caused by the injection of adrenalin was not due to the increased pressure in the heart.

Experiments were carried out by us on the heart-lung preparation, in order if possible to throw more light on this question. In the first place the intracardiac pressure was raised by increasing the venous supply and raising the arterial resistance, and the threshold of vagus stimulation determined under different heights of the aortic pressure. In the next place we added adrenalin to the blood in the venous reservoir, whereby practically no alteration took place as a rule in the arterial pressure, and compared the effects of vagus stimulation before and after the addition of adrenalin. In every case the vagus was stimulated in the neck with an induction current by means of Ludwig's platinum electrodes. The results of the experiments were as follows.

In the first series of experiments it was only possible to vary the aortic pressure between 30 and 200 mm. Hg. Within these limits the effect of vagus stimulation was always the same. Examples of this

are shown in Fig. 1 A and B, taken from a heart of 51 gms. In Fig. 1 A the aortic pressure was 80 mm. Hg and in Fig. 1 B 190 mm. Hg. The strength of stimulus coil used in both cases was 70 mm., a figure which was very little above the threshold of stimulation. As will be seen from the figures, the results of stimulation do not differ very greatly. In Fig. 1 A the venous pressure rose on stimulation from 22 to about 65, and in Fig. 1 B from 394 to 416.

As we could not determine the limits of the aortic pressure within which vagus stimulation was still effective, we are not justified in excluding the possibility that the abolition of vagus excitability is caused by the increased blood-pressure which occurs with adrenalin.

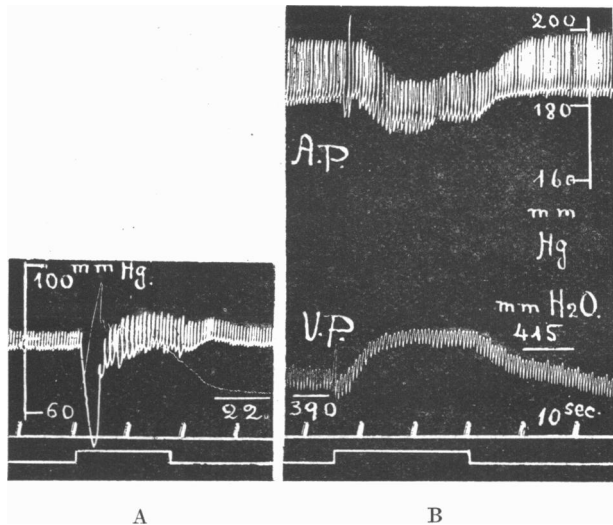


Fig. 1.

We observed however repeatedly in intact animals that vagus stimulation had no effect when the arterial pressure was raised to less than twice its normal height by the action of adrenalin.

In the second series of experiments we added .03 to .1 mg. adrenalin (Parke-Davis) to the blood in the venous reservoir. The blood-pressure remained as a rule unaltered, but the heart rate was increased. After the addition of .03 to .05 mg. adrenalin, stimulation of the vagus was as effective as or even more effective than under normal conditions. If however the amount of adrenalin was increased (.07 to .1 mg.) the heart did not react at all to the maximal vagus stimulation.

In intact animals the abolition of vagus action after intravenous injection was only temporary. Several workers and also one of us have observed this. In the present experiments however the excitability of the vagus was not recovered. This might be caused by the very long duration of the action of the adrenalin in the heart-lung preparation (cp. Fühner and Starling⁽³⁾) or by the gradual dying off of the vagus. It is quite certain that the abolition of vagus action observed in our experiments was not the result of the dying off of the vagus but was caused by the adrenalin, since it was proved that this abolition occurred suddenly after the addition of adrenalin while in the heart-lung preparation the vagus usually responds for at least an hour to electrical stimulation.

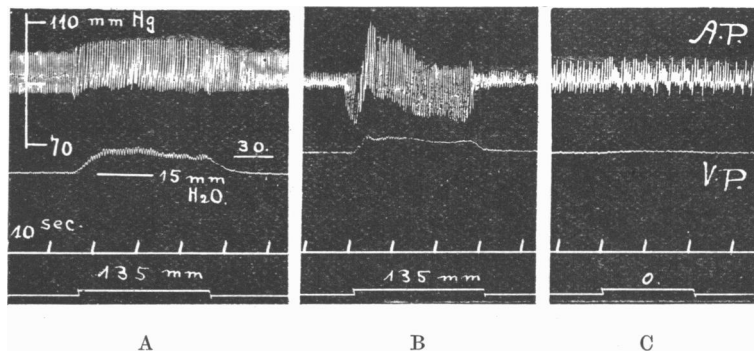


Fig. 2.

Fig. 2 *A-C* taken from a heart of 57 gms. is an example of this series of experiments. The blood-pressure in the three cases was about 91 to 94 mm. Hg. The left vagus was stimulated in *A* and *B* with a strength of coil of 135 mm. and in *C* with the coil distance 0. *A* was taken under normal conditions, *B* after the addition of .03 mg. adrenalin, and *C* after a second addition of .05 adrenalin. In *A* and *B* the effect of vagus stimulation was very marked, while in *C* it was hardly noticeable. An interval of seven minutes elapsed between *B* and *C*.

We desire to thank Prof. Starling for his advice throughout this research.

REFERENCES.

- (1) Langley. *This Journal*, xxvii. p. 237. 1901.
- (2) Bessmertny. *Ztsch. f. Physiol.* xlvii. S. 400. 1905-6.
- (3) Fühner and Starling. *This Journal*, xlvii. p. 286. 1913.