ventricle is often followed by a premature beat of the auricle. When the evidence of such retrograde contractions is best, the indicated time of backward conduction is 7/30 to 8/30 of a second.

8. There is no evidence of lengthened *As–Vs* intervals except at the extra-systoles.

9. There is a fairly fixed time relation between the normal and abnormal ventricular systoles.

10. There is often a considerable delay in the appearance of the carotid pulse resulting from the extra-systole.

11. There is a marked lack of synchronism between the carotid pulse and the "c" wave. The latter may precede the former by 1/20 of a second.

12. The site of the abnormal stimulus is either in the ventricle or some part of the conduction system, *e. g.*, the node, or some lower point.

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On the differences in the effects of stimulation of the two vagus nerves on rate and conduction of the dog's heart.

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The effects obtained in the action of dogs' hearts on stimulating the peripheral stumps of both vagus nerves were studied in fifty-four experiments. The dogs were anesthetized with ether without adjuvant, artificial respiration was maintained by the Meltzer-Auer method, and registration was accomplished by the galvanometric method. When the chest was opened, curves of auricular and ventricular contractions were also inscribed. Faradic stimulation was employed. The secondary coil of an inductorium, fed by a 2-volt dry cell, placed arbitrarily at 50 mm., was the source of the current.

On stimulating the right vagus nerve, the usual effect was obtained; both auricles and ventricles ceased to beat, generally throughout the period of stimulation. Occasionally the ventricles escaped from inhibition, but then the impulse to contraction arose

and spread in an abnormal manner; the structures normally concerned with these functions remained inhibited.

When the left vagus nerve was stimulated in the same dog, with a current of the same strength, a difference from the effect of stimulating the right nerve was observed in 88 per cent. of the fifty-four experiments. The auricles did not cease to beat, they were merely slowed,—sometimes 100 or more beats. In one group, normal ventricular contractions ceased entirely in twenty-four cases. In a second of twenty-four other cases, a ventricular contraction followed every second, occasionally every third, fourth or more auricular beats, the mechanism being one of incomplete dissociation. In a third group, the only effect of stimulation was an increase in the time occupied in conduction from auricles to ventricles.

In the first group when the left vagus was stimulated, as has been stated, normal ventricular activity ceased, but abnormal activity occasionally continued. The rate of the abnormal ventricular contractions differed from that of the slowed auricles and complete *As–Vs* dissociation resulted. Similar ventricular activity occurred also when the right vagus was stimulated, but the auricles, as is usual, ceased to contract.

The explanation offered for the phenomena occurring on left vagus stimulation is that the main effect consists in depressing the conduction system. In the first group, the ventricles cease to beat, because, on account of the great depression of conduction, they receive no impulses from the auricles. If the ventricular muscle is irritable, abnormal stimuli may be formed and an idioventricular rhythm result. When a slighter degree of depression of conduction has taken place, every second or third beat passes along the *A–V* bundle and initiates a ventricular contraction. A still slighter degree of depression results in a mere lengthening of the time of conduction.

The mechanism which results from stimulating the right vagus nerve and the negative chronotropic effect of the left nerve are not discussed at present.