THE INFLUENCE OF THE VAGUS NERVES ON THE FARADIZED AURICLES IN THE DOG'S HEART.*

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Plates 71 to 78.

The experiments on which this paper is based were undertaken in order to study the influence of the vagus nerves on auricular fibrillation. The problem was suggested by the study of a patient suffering from transient attacks of auricular fibrillation, whose heart was affected to an unusual degree by pressure over either of the vagi. The patient was of neurotic temperament, and the circumstances under which the attacks of auricular fibrillation occurred indicated that the controlling nervous mechanism of the heart might have played an important part in determining the onset and determination of the attacks. The case itself will be described in detail in another communication. The experiments were undertaken with the idea of determining whether the activity of the inhibitory mechanism played any part in rendering the heart susceptible to auricular fibrillation or in preventing its occurrence.

Certain experimental investigations have been carried on regarding the influence of the vagi on the fibrillating auricles. There has not been, however, entire agreement of interpretation of results by the various workers. Some have said that vagus stimulation may cause auricular fibrillation or at least increase the susceptibility of the heart to such a condition, while others consider that it prevents auricular fibrillation or causes established fibrillation to cease.

Knoll (1), Hewlett (2), Winterberg (3, 4, 5), and Cushny (6) point out that vagus stimulation increases the susceptibility of the heart to auricular fibrillation, and that faradization of the nerves alone may sometimes produce the condition in the mammalian heart. Winterberg (4, 5), who has investigated this question more thoroughly than anyone else, states that a weaker faradizing current is required to throw the auricles into fibrillation when combined with vagus stimu-

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lation or when drugs which raise the vagus tone have been administered, than when the auricles alone are stimulated. He also says that vagus stimulation may fix an auricular fibrillation set up by a short period of faradization, so that it continues after auricular faradization is removed.

McWilliams (7), Fischel (8), Philips (9), Hirschfelder (10), Garrey (11), Lewis (12), and Cushny (6) state that vagus stimulation, on the other hand, arrests auricular fibrillation. Winterberg (4) has noted that stimulation of the vags may both make the heart more susceptible to auricular fibrillation and also arrest it when established. McWilliams, Lewis, Winterberg, Rothberger and Winterberg (13), and Hewlett describe the effects which vagus stimulation has on established auricular fibrillation, and all agree that it causes the fibrillary movements to become more rapid, finer, and less easily seen. Rothberger and Winterberg consider this effect to be the same as that which causes a great diminution or disappearance of the P wave in the electrocardiogram of the normal heart-beat. Finally, Kronecker and Spillata (14) conclude that vagus stimulation does not affect auricular fibrillation.

METHODS.

The material upon which the present communication is based consists of twenty-three experiments on dogs. Medium sized or rather large animals, weighing from 6,800 to 17,000 grams, were used. As the experiments were varied, the number bearing on the various points considered is not always the same, and so will be mentioned in each instance. Records were obtained by means of the string galvanometer (Edelmann), and in one experiment by suspension curves from auricles and ventricles as well. Direct observation of the heart also proved useful.

The usual method of experimentation was as follows: The animal was etherized by intratracheal insufflation (Meltzer-Auer method), and German silver electrodes over cotton soaked with salt solution were applied to the right fore leg and the left hind leg. The string of the galvanometer was always adjusted so that one millivolt caused a movement of ten millimeters on the record. After an electrocardiogram had been taken, the chest was opened by means of a lateral incision through the third or fourth intercostal space. The right auricle was exposed by a small incision of the pericardium, and to it were attached, about five millimeters apart, two small clips wrapped separately in rubber tissue. From these clips light wires ran to the secondary coil of a Harvard inductorium. Both vagi were then dissected out in the neck, but not, as a rule, cut. The
right auricle was usually stimulated for fifteen seconds by a faradic current. If the auricles did not continue to beat tumultuously after the faradization ceased, it was repeated three times at intervals of one minute. If still unsuccessful, the vagi were cut and stimulation of the auricle was repeated in the same manner. Several minutes were allowed to elapse between the two sets of stimulations. If rapid, tumultuous auricular activity became established during the first set of stimulations, electrocardiograms were made before and after cutting the vagi, so that the effect of cutting the nerves could be observed. After cutting the vagi, the peripheral end of each nerve was laid in a shield electrode, which was protected by rubber tissue and dry gauze. The effect of stimulation of both the right and left vagi was investigated when the auricles were affected by faradization, as well as when the heart was beating normally. When the altered activity following auricular faradization could not be established so that it continued independently, vagus stimulation was begun immediately before ending auricular faradization. As the altered auricular activity almost always persisted during vagus stimulation, the effects of stimulating the nerves upon it could be studied in this way.

The strength of the faradizing current did not bear a constant relation to the length of time the auricles remained affected after faradization was ended. A current just strong enough to keep the auricles constantly in a state of tumultuous activity seemed as effectual as stronger currents in causing this activity to continue after faradization had ceased. With one two-volt cell, it was found in several experiments that when the secondary coil of the inductorium was out eighty or ninety millimeters, the current was just strong enough to maintain auricular fibrillation. To be well within this limit but to avoid very strong currents, the auricle was usually stimulated with the secondary coil sixty millimeters out. The vagi were stimulated by a faradic current from an inductorium, the secondary coil of which was extended fifty millimeters. The strength of this current is the same as that used by Cohn (15) in his experiments on the vagus control of the normally beating heart, so that the results of his experiments and those of this series are comparable.
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The importance of considering the effect of stimulating each vagus separately has become obvious since Cohn (15) has shown that there is a definite difference in function between the two nerves almost constantly demonstrable in dogs. This difference is as follows: when the right vagus in the dog is stimulated, there is, in a large proportion of cases, stoppage of the entire heart, while when the left is stimulated, the auricles beat at a rate not greatly reduced while partial heart-block or ventricular asystole usually occurs. The effect of stimulation of the two vagi must therefore be considered separately.

RESULTS.

1. The Effect of Faradization on Auricular Activity.—The auricles always beat in a rapid tumultuous manner during faradization. In some dogs this altered rhythm ceased synchronously with the end of faradization if no other measures were employed, while in others it continued and became established. This altered rhythm became established by faradization alone in eleven experiments, while in four others its establishment was accomplished by combining auricular faradization with vagus stimulation. It continued in these fifteen experiments from five minutes to over an hour after faradization, thus giving opportunities for studying its character and the effect of vagus stimulation upon it.

Direct observation of the exposed heart showed that the auricular activity following faradization consisted in very rapid movements, apparently contractions of the whole auricles, which were sufficient to produce definite movements of the recording tambour attached to the auricular myocardiograph (figure 11). Besides this rapid auricular tachycardia, fine fibrillatory movements in the various fibers could be seen. During the right vagus stimulation, the effect of which will be discussed later, the coarser movements ceased and the typical fine fibrillations persisted, and when the stimulation was removed the coarser movements could be seen definitely gradually returning and being coexistent with the fine fibrillations. This direct observation was confirmed by my colleague, Dr. Cohn, who first observed this phenomenon in one of my experiments.

The electrocardiogram (figure 7) shows no waves which resemble those caused by the normal auricular contractions, but between the
rapidly and irregularly recurring ventricular complexes, irregular waves of various sizes occur usually at a rate averaging 500 to 600 per minute. The waves may be four to five millimeters in height, but the larger waves are often interrupted by much smaller, finer, and more rapid movements of the string. The irregularities in the size and shape of the ventricular complexes are probably caused by various degrees of coincidence with the waves of auricular activity.

It is evident that the altered activity of the auricles usually resulting from faradization is not true fibrillation alone, but consists of two different forms of cardiac activity occurring simultaneously in the auricles; namely, tachycardia and fibrillation.

The auricular activity following faradization has been described and recorded by electrocardiograms by Rothberger and Winterberg and by Lewis. The records obtained by these observers agree closely with ours. From their direct observations they describe different grades of fibrillation, varying in extent of auricular movements. Lewis states that actual correspondence between the auricular activity and the electrocardiograms may be found only rarely, while Rothberger and Winterberg consider that no correlation can be made out. Beside these various grades of fibrillation Rothberger and Winterberg describe finally what they call unreines Schlagen. This form of auricular activity consisted of definite contractions of the auricles which affected at least the main mass of the musculature, together with either fibrillatory movements seen more or less distinctly, especially on the margins of the auricles, or weak peristaltic waves running across the auricles, occurring at the same time. This peculiar combination of the normal beat and delirium caused no corresponding impression on the electrocardiogram. The auricular wave was usually somewhat split, and here and there the rhythm was disturbed. They do not state how frequently this type of auricular activity was seen or under what conditions it occurred, and no further comment is made upon it. It is evident from their description that Rothberger and Winterberg have observed the same form of auricular activity which resulted almost constantly from auricular faradization in our experiments. The various grades of fibrillation which these observers and Lewis describe did not appear in our experiments except under conditions to be discussed,
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although a pure auricular tachycardia, as observed by Hirschfelder and others, followed faradization sometimes in some of our experiments. The rapid auricular activity may be conveniently designated tachycardia in this paper, as this was practically always the most obvious effect of auricular faradization.

2. The Influence of Cutting Both Vagi. (a) On the Susceptibility of the Normally Beating Heart to Auricular Faradization (Fourteen Experiments).—Prolonged auricular tachycardia was more readily produced by faradization after cutting both vagi in three experiments, while in ten no change in the susceptibility of the auricles to faradization could be detected. In one tachycardia seemed more readily produced before than after cutting the nerves. The protocol of experiment 12 shows the positive influence that cutting the vagi may have.

11:23 A.M. Right vagus tied and cut. Tachycardia for 10 minutes after end of faradization. Normal beat returned only after four vagus stimulations.
11:24 A.M. Left vagus tied and cut.
11:27 A.M. Auricular faradization, 15 seconds.

(b) On the Heart with Established Auricular Tachycardia (Eight Experiments).—In these experiments prolonged tachycardia was established with the vagi intact, so that records could be obtained before and after they were cut. This procedure had apparently very little effect on the auricular activity. In six experiments no effect could be detected in the electrocardiograms, while in one the waves of auricular activity were considerably slowed and in another rendered larger and more distinct. The ventricular rate was increased in four experiments, while in three there was practically no change. In the one other experiment, no conclusions can be drawn, as other procedures may have influenced the ventricular rate. In several experiments there was seen a gradual increase in ventricular rate.
as auricular tachycardia continued, so caution is necessary in interpreting these results, but in three, at least, of the four positive experiments, the results seem quite definite. This is seen especially well in experiment 13 where the ventricular rates during auricular tachycardia were as follows:

- 86 per minute 1 minute after onset.
- 160 per minute 5 minutes after onset.
- 168 per minute 7 minutes after onset (figure 1).
- Both vagi cut 9 minutes after onset.
- 300 per minute 12 minutes after onset (figure 2).
- 240 per minute 20 minutes after onset.

When the heart was beating normally, cutting the vagi caused a definite increase in the rate of the heart-beat in thirteen of seventeen experiments.

3. The Influence of Stimulation of Each Vagus Nerve. (a) On the Auricular Activity.—Stimulation of the right vagus nerve (nineteen experiments) had a definite and almost constant effect on the auricular tachycardia, which could be observed by watching the heart directly and by changes in the electrocardiograms. Suspension curves were made in one experiment in which the heart behaved in a manner entirely typical of the other experiments. The auricular activity could be seen to undergo the marked change mentioned above. Instead of the auricles moving about rapidly as a whole, they became practically stationary, allowing the true fibrillatory contractions of the various muscles fibers to be plainly seen. They were similar to those observed in the ventricles when in fibrillation. In the electrocardiograms the undulations representing auricular activity became more rapid, blurred, and often almost disappeared. The large waves, sometimes four to five millimeters in height, rarely appeared (figures 5, 8, 12). In the experiment in which suspension curves were also made of the right auricle and right ventricle, it was found that the auricular tachycardia produced movements of the recording tambour nearly as distinct as those produced by the normally beating heart (figures 10 and 11). When the right vagus was stimulated (figure 12), the waves of the auricular suspension curve became smaller and smaller and finally disappeared, synchronously with the replacement in the electrocardiogram of very fine
small waves of true fibrillation for the well defined coarse waves of auricular tachycardia. Finally, it may be mentioned again that soon after right vagus stimulation, distinct contractions of the entire auricles could be seen accompanying but not replacing the fine fibrillatory movements in the auricular walls. It is evident, therefore, that when the right vagus was stimulated, it exerted its inhibitory influence on the auricular tachycardia, causing it to cease, while the true fibrillatory movements were apparently unaffected. This effect of right vagus stimulation occurred in seventeen of the nineteen experiments (89.5 per cent.), and may therefore be considered characteristic. It is apparently similar to that which has been described by other experimenters already mentioned. In one of the two instances where the typical effect was not observed, the character of the record changed in an unusual manner. At first the waves were blurred and very irregular. Then separate, distinct little waves, resembling the normal P waves, appeared at a rate of 560 per minute, and continued to occur as long as the vagus was stimulated. Here the auricular tachycardia apparently succeeded in reasserting itself. In the second instance no change could be observed in the auricular activity. In this experiment even the normally beating heart did not react to right vagus stimulation in the usual manner, as a distinct delay occurred between the time of onset of stimulation and stoppage of the heart.

The effect of left vagus stimulation on auricular fibrillation was studied in seventeen of the nineteen experiments in which right vagus effects were observed. A constant difference was observed between the influence of right and of left vagus stimulation in twelve of these seventeen experiments (70.6 per cent.). During left vagus stimulation direct inspection showed that the coarser movements of the auricles were not disturbed, but appeared sometimes perhaps even more distinctly. It was difficult to determine with certainty whether the fibrillatory movements ceased or were influenced. In some of the electrocardiograms there was seen slight blurring and quickening in rate of the waves of auricular activity similar to, but always less than, that seen with right vagus stimulation in the same animal. In other records the waves of auricular activity occurred more regularly, uniformly, and distinctly during
left vagus stimulation than before, and only the larger waves appeared, the small, fine rapid movements of the string which interrupted the larger waves practically disappearing (figures 6, 9, and 13). While some records, those first mentioned, indicate that the difference between the effects of right and left vagus stimulation seems to be one of degree rather than of kind, the others show that the type of response to the two nerves seems different. The distinctness and regularity with which the waves of auricular activity appeared may have been due to the removal of the confusing ventricular complexes, but the records suggest strongly that left vagus stimulation affected the auricular activity in an opposite manner from right vagus stimulation, and a change was produced which, instead of blurring and quickening the auricular waves, rendered them clearer, more uniform, and more regular.

The suspension curve from the auricles (figure 13) shows that the coarser auricular activity was not disturbed during left vagus stimulation, while during right vagus stimulation the suspension curve gave no evidence of motion. It seems evident, therefore, that stimulation of the left vagus often had practically no influence on the auricular tachycardia, while there is some evidence that it exerted an inhibitory influence on the true fibrillation.

In four experiments no definite difference could be made out between the effect of the two vagi on the auricular activity resulting from faradization. In three of these fairly marked changes occurred when each nerve was stimulated, while in the fourth the effect of each nerve was very slight.

The effect of stimulation of each nerve on the normally beating heart was studied in seventeen experiments, and in twelve of these (70.6 per cent.) the characteristic differences between the effects of stimulation of the two nerves were seen, just as described by Cohn (figures 3 and 4). Although the difference in effect of stimulation of the two nerves on the normally beating heart is more striking, it does not appear to occur more constantly than the difference in effect on auricular activity after faradization.

The difference in the effects of stimulation of the two vagi are seen in the figures. The curves in figures 3 to 6 are from one experiment (No. 18) and show the action of the two vagi on the heart,
both when it was beating normally and when auricular tachycardia was present. In this experiment prolonged auricular tachycardia could not be established, so vagus stimulation was begun before the end of auricular faradization. The curve in figure 7 (experiment 11) is an electrocardiogram obtained after the auricles had been faradized, and figures 8 and 9 from the same experiment show the changes in the electrocardiograms which resulted from vagus stimulations. Figures 10 to 13 show suspension curves from the auricles and ventricles, and illustrate the changes in auricular activity which have been described.

The results of the various experiments indicate that the length of time that tachycardia had been established did not apparently influence the vagus action upon it, and that the difference in effect of stimulation of the two nerves was observed regardless of which nerve was stimulated first.

(b) On Reëstablishment of the Normal Sequential Beat (Thirteen Experiments).—It was frequently seen that after auricular tachycardia had become established and had continued for several minutes without showing any signs of ceasing spontaneously, it would cease several seconds after the end of stimulation of one of the vagi (figure 14). It was concluded from the constant behavior of the dog's heart in this respect that vagus stimulation was the cause of the reëstablishment of the normal sequential beat. Of thirteen experiments, stimulation of each nerve was followed by the reëstablishment of the normal beat in seven. Left vagus stimulation alone produced this result in three, right vagus stimulation produced it in one, while in two experiments the normal beat never returned as a result of vagus stimulation. In other words, right vagus stimulation caused cessation of the abnormal auricular activity in 61.5 per cent. of the experiments, while left vagus stimulation had this effect in 76.9 per cent. Although numerically considered, this difference in the two nerves is not great, it is clear that stimulation of the left vagus was more effectual in reëstablishing the normal sequential beat than that of the right. Vagus stimulation did not usually seem to produce any permanent change in the susceptibility of the auricles to faradization, although in two or three experiments the auricles apparently became more resistant to faradization after the vagi had been stimulated a number of times.
(c) On the Susceptibility of the Auricles to Faradization.—In the experiments in which prolonged auricular tachycardia could not be established by auricular faradization alone, the influence of vagus stimulation in aiding the auricles to maintain prolonged tachycardia could be observed. The method of determining this influence was to throw the auricles into tachycardia by faradization for fifteen seconds, and to begin vagus stimulation just before ending auricular faradization. Vagus stimulation was continued for five to eight seconds. Twice in ten experiments right vagus stimulation caused the tachycardia to become established and to continue for some minutes after the vagus stimulation ceased. The same result was obtained in two other experiments with left vagus stimulation. In seven of the eight experiments in which faradization combined with right vagus stimulation did not succeed in establishing the tachycardia, the auricles always remained in a state of tachycardia as long as the nerve was stimulated. It seemed quite evident that the auricular tachycardia was maintained by vagus action. In the remaining experiment tachycardia continued during three out of four vagus stimulations, so it can be said that when the auricles were thrown into a state of tachycardia by auricular faradization, they continued almost invariably in such a state as long as the right vagus was stimulated. With left vagus stimulation this result was obtained in but five of eight experiments, and in three experiments in which right vagus stimulation maintained auricular tachycardia, left vagus stimulation, although active in other respects, failed to do so. It appears that stimulation of the right vagus nerve was more effectual than stimulation of the left in aiding the auricles to maintain the activity set up by faradization, a fact that was especially noted in the protocols of several experiments.

(d) On Initiating Auricular Tachycardia.—In two experiments on hearts in which auricular tachycardia was readily established by faradization, it was also initiated by right vagus stimulation alone. In one (No. 25) it began on two occasions during vagus stimulation (figure 15), and in the other (No. 13) this occurred two seconds after the stimulation ceased (figure 16). This first experiment (No. 25) is especially interesting because auricular tachycardia was stopped as well as started by right vagus stimulation. Twenty-
four minutes after the tachycardia had been established by right vagus stimulation alone (figure 15), it ceased four seconds after stimulation of the same nerve (figure 14). This result was obtained twice in this experiment with right vagus stimulation, but stimulation of the left nerve was also followed by a return of the normal beat two minutes after auricular tachycardia had been set up by right vagus stimulation alone.

Stimulation of the left nerve alone never caused auricular tachycardia in these experiments, although it did follow left vagus stimulation in one case in a long series of vagus stimulations by Dr. Cohn.

The influence of vagus stimulation on the ventricles during the type of cardiac activity which results from auricular faradization will not be considered in this paper.

DISCUSSION.

The auricular activity which follows faradization of the auricles is apparently the result of a change in the physiological properties of the auricular musculature. This change seems to alter the excitability and the power of stimulus formation in the auricles and finds expression in the establishment synchronously of auricular tachycardia and true fibrillation. This altered auricular activity persists after faradization of the auricles has been withdrawn, indicating that the altered cardiac properties do not immediately return to their normal state as soon as the exciting cause is removed.

The normal tonic vagus activity has in some animals a definite control over these cardiac properties under the conditions of our experiments, as cutting the vagi sometimes increases the susceptibility of the auricles to faradization. After the auricles take on the activity resulting from faradization, the tonic vagus action on the auricles seems very slight, as but little if any effect can be detected when the vagi are cut during auricular tachycardia. The influence of this procedure on the ventricular activity is, however, in some experiments quite definite, and results in a marked increase in ventricular rate. The result depends obviously upon a change in conductivity in the auriculoventricular conducting system. The depression of auriculoventricular conduction is one of the most obvious
activities of the vagi, and when this depression is removed, the ventricles are stimulated to contraction by a larger number of impulses from the rapidly contracting auricles than when the vagi are intact. That this increase in ventricular rate is not constant when the vagi are cut indicates that conductivity is not always depressed by tonic vagus action.

The effects of vagus stimulation on the auricular activity resulting from faradization are in accord with the effects which stimulation of the two nerves has on the normally beating heart. Stimulation of the right vagus stops the auricular tachycardia just as it stops the auricles in the normally beating heart, and stimulation of the left nerve fails to do so in both instances. True auricular fibrillation obviously is not controlled by right vagus stimulation, while there is insufficient evidence to allow any definite statement as to the effect of left vagus stimulation upon it. The electrocardiograms seem to show, however, that auricular fibrillation may be inhibited by left vagus stimulation while the tachycardia proceeds undisturbed.

Auricular tachycardia in man may sometimes be controlled by pressure over the right vagus nerve, as will be shown by electrocardiograms to be published shortly by my colleagues, Drs. Cohn and Fraser, while Draper and I (16) have published electrocardiograms which indicate that right vagus pressure does not influence auricular fibrillation in man.

The mechanism by which the normal sequential beat is restored a few seconds after vagus stimulation is difficult to understand. Left vagus stimulation seems somewhat more effectual in producing this result than right vagus stimulation, so that it cannot come from inhibitory influences of the auricular tachycardia. More facts are needed before an explanation of this phenomenon can be attempted.

Vagus stimulation increases the susceptibility of the auricles to faradization. This is indicated by the fact that in animals whose auricles are resistant to faradization, the abnormal auricular activity continues after faradization as long as one of the vagi is stimulated. This effect probably results from the inhibitory influence of vagus stimulation on the normal auricular activity. As long as the normal pace-maker is depressed, there is probably a diminished tendency for the abnormal auricular activity to be superseded by the normal
sequential beat. The more marked influence which the right vagus has on the normal auricular activity explains why it is more effectual in holding the auricles in the abnormal activity than the left nerve. It may be that it is through this same mechanism that the abnormal auricular activity can be originated by vagus stimulation alone. If the auricles are in a state very favorable to the establishment of tachycardia and fibrillation, inhibition of the normal pace-maker alone may suffice for the establishment of the abnormal auricular activity. The fact, however, that the tachycardia may come on during right vagus stimulation without showing the usual effect of the stimulation (figure 15), or that it may come on at a time after the stimulation when the normal pace-maker usually reasserts itself (figure 16), does not lend support to this possibility. It is not clear also why a short vagus stimulation following the faradization should assist in the establishment of the abnormal activity of the faradized auricle as an independent auricular activity.

SUMMARY AND CONCLUSIONS.

An abnormal auricular activity is produced by faradization of the right auricle of the dog, which frequently becomes established and continues for varying periods of time after faradization is discontinued. This auricular activity consists of a rapid auricular tachycardia coexisting with true auricular fibrillation. In some dogs the auricles are thrown into this abnormal activity more readily by faradization after the vagi have been cut than before. Cutting the nerves has little or no effect on the abnormal auricular activity, but the ventricular rate may be much increased if the vagi are cut after the abnormal auricular activity has been established, apparently because of an improvement in the auriculoventricular conductivity.

Stimulation of the right vagus nerve changes the character of the activity of the faradized auricles by inhibiting the auricular tachycardia while the fibrillation is uninfluenced. Stimulation of the left vagus nerve has little or no apparent inhibitory effect on the auricular tachycardia, but has possibly an inhibitory effect on the auricular fibrillation.

Vagus stimulation increases the susceptibility of the auricles to faradization. The abnormal activity set up by faradization may be
EXPLANATION OF PLATES.¹

PLATE 71.

Fig. 1. Experiment 13. Auricular tachycardia, vagi intact. Ventricular rate, 168 per minute.
Fig. 2. Experiment 13. Auricular tachycardia, vagi cut. Ventricular rate, 300 per minute.
Fig. 3. Experiment 18. Normal beat. Effect of right vagus stimulation of 3.2 seconds' duration.

¹Time marking in all curves is in 1/5 and in 1/50 of a second.
PLATE 72.


FIG. 5. Experiment 18. Auricular tachycardia. Effect of right vagus stimulation of 7.6 seconds' duration, begun during auricular faradization.
PLATE 73.

Fig. 6. Experiment 18. Auricular tachycardia. Effect of left vagus stimulation of 5.4 seconds' duration, begun during auricular faradization.

Fig. 7. Experiment 11. Auricular tachycardia without vagus stimulation.
Plate 74.

Fig. 8. Experiment I. Auricular tachycardia. Effect of right vagus stimulation of 5.6 seconds' duration, begun ½ minute after the onset of auricular tachycardia.

Fig. 9. Experiment II. Auricular tachycardia. Effect of left vagus stimulation of 6.2 seconds' duration, begun 2 minutes after the onset of auricular tachycardia.
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PLATE 75.

Fig. 10. Experiment 27. Normal beat. Suspension curves from auricles and ventricles and electrocardiogram.

Fig. 11. Experiment 27. Auricular tachycardia without vagus stimulation. Auricular rate, 550, as shown by the suspension curve and by the electrocardiogram.
FIG. 10.

FIG. 11.

(Robinson: Influence of Vagus Nerves.)
PLATE 76.

FIG. 12. Experiment 27. Auricular tachycardia. Effect of right vagus stimulation as shown by the suspension curve and electrocardiogram. True fibrillation during the period of ventricular asystole.

FIG. 13. Experiment 27. Auricular tachycardia. Effect of left vagus stimulation. Auricular rate before stimulation, 525. During stimulation, 523 per minute, as seen in suspension curve. The electrocardiogram is confused by an escape of the stimulating current.
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PLATE 77.

Fig. 14. Experiment 25. Normal beat resumed 4 seconds after the end of right vagus stimulation of 5.4 seconds' duration. Auricular tachycardia had been present 24 minutes previous to the onset of right vagus stimulation.

Fig. 15. Experiment 25. Auricular tachycardia set up by right vagus stimulation of 3 seconds' duration. Tachycardia began during vagus stimulation, 1.2 seconds after the stimulation was begun.
FIG. 16, a and b. Experiment 13. Auricular tachycardia set up by right vagus stimulation of 5 seconds' duration. Tachycardia began 3 seconds after the end of vagus stimulation. 16 b is a direct continuation of 16 a.
established in hearts otherwise refractory by vagus stimulation of short duration following the faradization. Vagus stimulation usually holds the auricles in the abnormal activity set up by faradization as long as it is continued in hearts in which, without vagus stimulation, the sequential beat always returns as soon as faradization is stopped. The right vagus is more effectual in this respect than the left. In some hearts vagus stimulation alone is capable of initiating the same abnormal auricular activity which is caused by auricular faradization. The normal sequential beat is often restored by vagus stimulation. It replaces the abnormal auricular activity not during, but a few seconds after, the termination of vagus stimulation. Left vagus stimulation is somewhat more effectual in producing this result than right vagus stimulation.

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