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THE MECHANISM OF AURICULAR PAROXYSMAL TACHYCARDIA

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AURICULAR paroxysmal tachycardia was long ago described and recognized as a clinical entity, but the fundamental mechanism or mechanisms responsible for this disorder have not yet been finally ascertained.[1, 2] Unlike auricular flutter and auricular fibrillation, it cannot be readily induced in experimental animals, and cannot, therefore, be easily studied by this method. Speculations as to its nature must, therefore, be based on pertinent observations on man. We propose to discuss from this standpoint the following features of this disturbance: (1) the form of the auricular deflections; (2) the effects of exertion, vagal stimulation, digitalis, quinidine, and other drugs upon the auricular rate and the duration of the paroxysms; (3) similarities, differences, and relations between it and auricular flutter and fibrillation; (4) the spontaneous occurrence of auriculoventricular block in a small number of cases and the difficulty or impossibility of producing it in most of the others; and (5) the occurrence of alternation in the auricular cycle length. We wish particularly to examine the suggestion[3] that auricular paroxysmal tachycardia is caused by circus rhythm involving one of the specialized auricular nodes.

When Mines[4] described circus rhythm he suggested that it might be responsible for some cases of paroxysmal tachycardia in man. Iliescu and Sebastiani[5] were among the first to suggest that auricular paroxysmal tachycardia is due to circus contraction. Their reasoning was based chiefly on the action of quinidine in this disorder. Lewis[2] pointed out that the total amount of auricular muscle is not sufficiently large to accommodate a circus mechanism at known rates of conduction in auricular

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muscle and with cycles as long as those which occur in auricular paroxysmal tachycardia. For this reason and because of the isoelectric intervals separating the auricular deflections, he could not accept circus rhythm as the cause of this disorder. He did not mention the possibility that the path of the circus impulse might pass through one of the specialized auricular nodes. Ashman and Hull[3] have suggested that in auricular paroxysmal tachycardia there is a circus rhythm in which the re-entrant impulse passes through one of the nodes. In a necessarily brief discussion they presented only a part of the evidence which supports this view. They mentioned especially the effects of vagal stimulation and the slowness of the rate, as compared with auricular flutter, in relation to the slowness of conduction through nodal tissue.

The Form of the Auricular Deflections.—The form of the auricular deflections was examined in the electrocardiograms of one hundred unselected cases of auricular paroxysmal tachycardia. In thirty-four they were upright in Leads I and II, and in many of these the P waves closely resembled, or were identical with, those recorded when normal sinus rhythm was present. In fourteen cases the auricular deflections were inverted in Leads II and III. In fifty cases these deflections were intermediate in form, very small, flat or diphasic, or they were not clearly visible because they were small and superimposed upon some part of the ventricular complex. In many of the cases in which there were upright P waves the impulse must have entered the main mass of auricular muscle near the normal pacemaker in the upper part of the sinoauricular node. The inverted P waves suggest beats arising in or near the auriculoventricular node, or in some outlying part of the auricular muscle. The point of origin of beats represented by auricular deflections of intermediate form cannot be stated with certainty. Lewis[2, 6] has shown that deflections of this form may arise in a region between the upper part of the sinoauricular node and the auriculoventricular node, and that they may be produced by impulses arising in the lower part of the sinoauricular node. With two exceptions the form of the auricular deflections in the cases of auricular paroxysmal tachycardia which we reviewed is not inconsistent with the view that the paroxysmal focus lay in or near the sinoauricular node or in or near the auriculoventricular node. The exceptions were two cases in which the auricular deflections were inverted in Lead I and upright in Leads II and III. Such cases are very uncommon; in them the form of the auricular deflections suggests a focus located in the upper part of the left atrium.

The Stability of the Rate.—In auricular paroxysmal tachycardia the auricular rate is ordinarily remarkably constant. It is not, as a rule, influenced by rest, posture, emotion, or exertion. It is not under the control of the extrinsic cardiac nerves, as are slower rhythms of the normal type arising in the sinoauricular node or the auriculoventricular node. Even at the onset of attacks, and at the end of paroxysms terminating

spontaneously, there is usually little or no alteration of the rate. The patients with upright auricular deflections in Leads I and II, and especially those with atrioventricular block, show somewhat more variation in auricular rate than do those whose auricular waves are inverted or intermediate in form, with no block.

Exercise.—The auricular rate in auricular paroxysmal tachycardia is sometimes increased by exercise,[7*, 8] but in auricular fibrillation the opposite is usually observed.[9†] Slowing of the fibrillating auricles on exertion is attributed to a reduction of vagal tone; atropine also slows the rate of the fibrillating auricles.[11] The acidosis accompanying exercise should, by depressing conduction in the auricles, tend further to reduce their rate.[4, 12, 13] The occasional increase in the rate of auricular paroxysmal tachycardia on effort may be attributed to a reduction of vagal tone; acidosis should cause slowing, whatever the mechanism.

Stimulation of the Vagus.—In auricular paroxysmal tachycardia, pressure upon the carotid sinus and other measures which stimulate the vagus nerves not uncommonly slow the rate of the paroxysm and frequently restore normal rhythm.[14, 15] As a rule the slowing occurs immediately preceding the termination of the attack, but occasionally the rate is slowed when the attack is not stopped.

Vagal stimulation diminishes the rate of impulse production in the sinoauricular and the auriculoventricular node, and depresses conduction in the latter. Its effect upon the rate of conduction in the sinoauricular node and upon the rate of impulse production in auricular muscle outside the nodes is not known. Vagal stimulation shortens the effective refractory period of ordinary auricular muscle and improves its conductivity. In disorders caused by circus rhythm in auricular muscle these effects tend to increase the number of cycles per minute.[16] The slowing of the rate of paroxysmal tachycardia and the restoration of normal rhythm by vagal stimulation cannot be explained by the effect of increased vagal tone upon the characteristics of the ordinary auricular muscle, and are most logically attributed to the action of these nerves upon the nodes.

Digitalis.—Like vagal stimulation, digitalis often slows the rate of auricular paroxysmal tachycardia and restores normal rhythm.[17] Occasionally the drug slows the rate without stopping the tachycardia, but usually the slowing occurs only a short time before the termination of the attack. Digitalis acts directly upon the heart muscle, and indirectly by stimulating the vagus nerves. In auricular fibrillation and flutter the increase in the circus rate produced by the drug is apparently caused primarily by increased vagal tone, which shortens the refractory period of the ordinary auricular muscle. The slowing of the ventricular rate

*Case 9.

†Doumer[10] has observed in auricular flutter an increase in auricular rate after exercise. This is exceptional.

is due to both a direct and an indirect depression of the conductivity of the atrioventricular node. The effects of digitalis in paroxysmal auricular tachycardia cannot be attributed to shortening of the refractory period of the auricular muscle, and would seem to depend upon its nodal action or upon some effect as yet unknown. The effect of digitalis upon conduction within the sinoauricular node is not known. The direct action of the drug upon auricular muscle is to increase its effective refractory period and to depress its conductivity.[18, 19] This effect can scarcely account for its ability to slow or abolish auricular paroxysmal tachycardia; it is opposed by the indirect vagal action of the drug upon the auricular muscle, and the effects of digitalization and vagal stimulation in the condition are similar, not opposite. The effect of the drug upon the rate of impulse production in auricular muscle outside the nodes is not known. If the slowing of the rate and the restoration of normal rhythm in auricular paroxysmal tachycardia are caused by any of the known effects of this drug, it would seem that they must be attributed to its effects upon nodal tissue.

Quinidine.—Quinidine and quinine likewise often slow the rate of auricular paroxysmal tachycardia and sometimes restore normal rhythm.[5, 8, 20] These drugs, too, act upon the heart both directly and indirectly. The indirect effect is a reduction of vagal tone. In the auriculoventricular node the direct and vagal actions are opposed; the direct effect is to depress conduction and the indirect to improve it. Consequently, the changes in auriculoventricular conductivity are somewhat variable.[21] In auricular muscle both the direct and the indirect actions increase the effective refractory period and depress conductivity. It is through these effects that these drugs invariably slow the circus rate and often restore normal rhythm in auricular fibrillation and flutter.[19, 21, 22] In man they do not slow the rate of simple sinus tachycardia;[20, 23] indeed, they often increase the rate of slower sinus rhythm.[20] In dogs both acceleration and slowing of the sinus rhythm have been observed under different conditions.[21, 24, 25] It is difficult to understand how the known effects of these drugs upon conductivity and upon the effective refractory period can bring about slowing of the rate and restoration of normal rhythm in abnormal tachycardias not caused by circus rhythm.

Relation to Auricular Flutter and Fibrillation.—Auricular paroxysmal tachycardia sometimes changes spontaneously to auricular flutter or fibrillation, or flutter or fibrillation may change to paroxysmal tachycardia.[26, 27, 28] In some cases digitalis may have converted paroxysmal tachycardia into fibrillation.[7, 29] Such changes in rhythm, although not common, suggest a relationship between these disorders.

In the preceding paragraphs mention has been made of some of the similarities and differences between auricular paroxysmal tachycardia, on the one hand, and auricular flutter and fibrillation, on the other, with respect to the effects of exertion, vagal stimulation, digitalis, and

quinidine. Like paroxysmal flutter and fibrillation, auricular paroxysmal tachycardia begins and ends abruptly. The auricular rate in paroxysmal tachycardia, as in flutter and fibrillation, is quite stable; it is not often influenced appreciably by rest, posture, emotion, or exercise, as is the normal sinus rhythm. Auricular paroxysmal tachycardia with partial auriculoventricular block resembles auricular flutter in many respects.[7, 30] Quinidine usually slows the heart rate in paroxysmal tachycardia, and always slows the circus rate in fibrillation and flutter, but does not slow sinus tachycardia. These features of auricular paroxysmal tachycardia suggest that its mechanism, like that of flutter and fibrillation, may depend on circus rhythm.

The mechanism of auricular paroxysmal tachycardia cannot, however, be a circus contraction involving ordinary auricular muscle only, like that of auricular flutter or fibrillation. In some instances exercise increases the rate of auricular paroxysmal tachycardia, but it usually slows the rate of the fibrillating auricles. Vagal stimulation and digitalis often slow the rate of paroxysmal tachycardia, whereas, in flutter and fibrillation, they increase the circus rate. Furthermore, as pointed out by Lewis,[2] the total amount of auricular muscle is not sufficiently large to accommodate a circus mechanism at known rates of conduction and with cycles as long as those of auricular paroxysmal tachycardia. In addition, the auricular deflections of paroxysmal tachycardia, unlike those of flutter, are separated by intervals of electrical quiescence, in which the galvanometer string is at rest in the isoelectric position.[2, 7, 31]

The separation of the auricular deflections of paroxysmal tachycardia by isoelectric intervals is shown commonly in standard leads, usually in chest leads, and always in esophageal leads; it is most clearly seen in cases of tachycardia with auriculoventricular block in those auricular cycles which are entirely free of ventricular deflections.[7, 31] This is quite different from flutter, in which the electrocardiographic oscillations are continuous; the tracing never comes completely to rest at the isoelectric line. Complete separation of the auricular deflections is inconsistent with circus rhythm unless some way is found to account adequately for isoelectric intervals during the continuous activity of the circus movement. They could be accounted for if the circus path traversed either the sinoauricular or the auriculoventricular node. While the advancing wave of activity was passing through the node, the amount of tissue entering the active state would be too small to produce electrical forces detectable by present methods. The observed auricular deflection would be inscribed by the activation of auricular muscle after the impulse emerged from the node. Its form and direction would be governed by the point at which the impulse entered the main body of the auricular muscle. Impulses emerging from the upper part of the sinoauricular node should give rise to upright deflections not unlike those of normal rhythm, impulses emerging from the atrioventricular

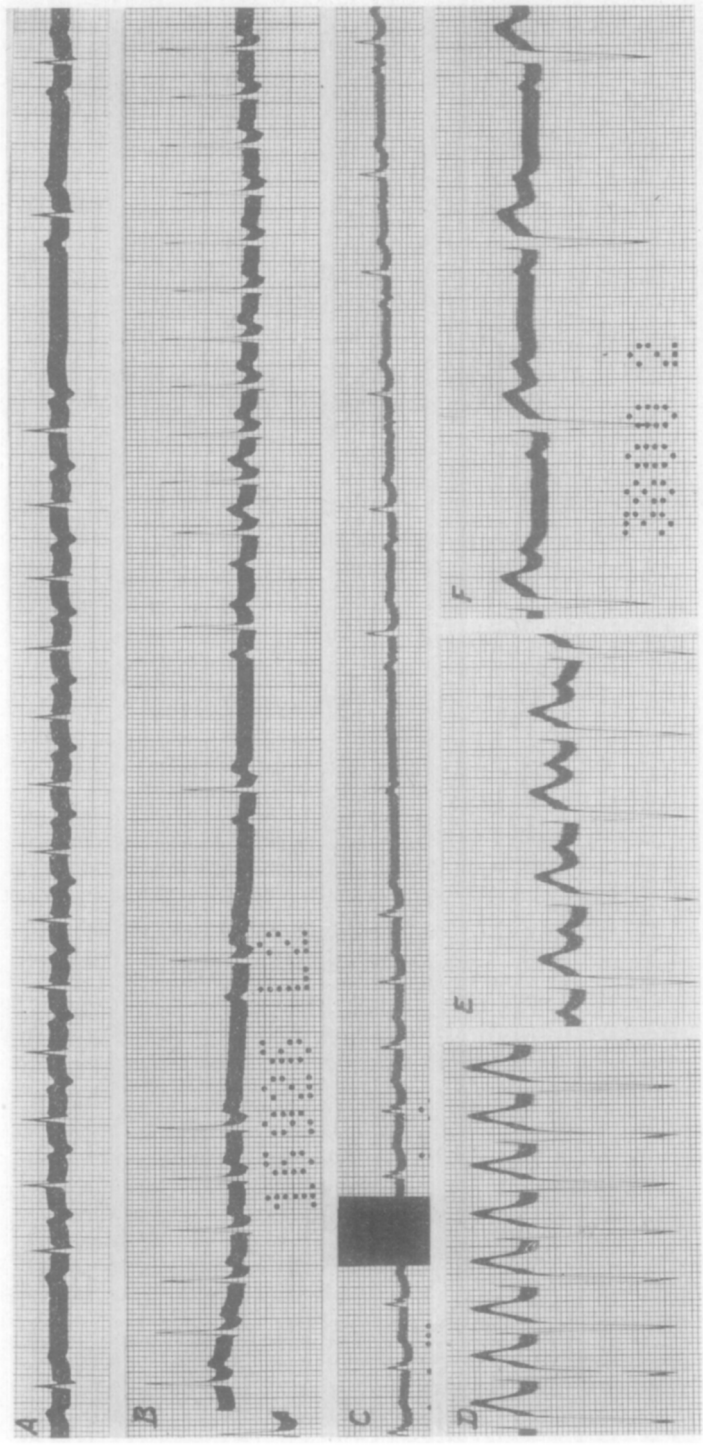

Fig. 1.—Electrocardiograms illustrating the relationship between the termination of attacks of auricular paroxysmal tachycardia and depression of atrioventricular conductivity. *A*, Lead II: a paroxysm ends spontaneously with an abnormal auricular beat which is blocked. *B*, Lead II: deep breathing; the P-R interval is prolonged (0.28 second) after the first normal auricular beat, and somewhat shorter (0.24 to 0.22 second) in subsequent beats. *C*, Lead II: pressure upon the carotid sinus; the first normal auricular beat is blocked. *D*, *E*, and *F*, chest leads: the paroxysm (*D*) was stopped by digitalis; normal rhythm (*E*) was followed almost immediately by 2 to 1 atrioventricular block (*F*).

node should give rise to inverted deflections like those of atrioventricular rhythm, and deflections of intermediate form might be produced by impulses emerging from the lower part of the sinoauricular node.[2, 6] In a mechanism such as this a circus rhythm could be accommodated in auricular muscle and in one of the specialized nodes at known rates of conduction and with cycle lengths such as occur in auricular paroxysmal tachycardia. The rate of the tachycardia would depend chiefly upon the speed of conduction and the length of the path within the nodal tissue.

Auriculoventricular Block.—Auriculoventricular block is extremely rare in those cases in which inversion of the auricular deflections suggests that the paroxysmal focus lies in or near the auriculoventricular node. It seems strange, in view of the high auricular rate, that block should occur so rarely. The most reasonable explanation would seem to be that the agents which tend to produce block, namely, vagal stimulation and digitalis, terminate the paroxysm before block occurs. It is easy to understand that this might happen in tachycardia which is dependent upon circus rhythm involving the atrioventricular node. At any rate, it appears that, in many cases of this kind, unimpaired conduction in this node is essential to maintenance of the paroxysmal tachycardia, and that depression of conduction in this node is intimately related to the termination of the attacks.

Not infrequently this relationship is apparent when the end of the paroxysm is recorded; sometimes the last paroxysmal auricular impulse is blocked, or block appears immediately after the cessation of the attack. Several electrocardiograms illustrating this phenomenon are shown in Fig. 1. Curve *A* (Lead II) shows a short attack of auricular paroxysmal tachycardia in its entirety. It begins with a premature inverted auricular deflection which resembles an auricular extrasystole, and ends spontaneously with a similar beat which is blocked. The P-R interval is slightly prolonged (0.24 second), both during the normal and during the abnormal rhythm. The last four cycles of the paroxysm are longer than those that precede them. A somewhat more complicated disturbance is shown in Curve *B;* in this case, deep breathing temporarily stopped an attack of auricular paroxysmal tachycardia. During the paroxysm the P-R interval was prolonged and the inverted auricular deflections (Lead II) followed closely the R waves of the preceding beats. The attack ended with a blocked auricular beat. The first and second beats after the end of the paroxysm are complicated by blocked auricular extrasystoles, as shown by the inverted auricular deflections which closely follow the R waves. The third beat is not so disturbed, but the fourth beat is followed immediately by a similar premature auricular beat which initiates another paroxysm of tachycardia. The P-R interval is abnormally long (0.28 second) after the first normal auricular beat, and somewhat shorter (0.24 to 0.22 second) in subsequent beats—the patient had received digitalis. In a third patient (Tracing

C) pressure upon the carotid sinus stopped the paroxysm. The auricular deflections (Lead II) are not clearly visible during the tachycardia. The first normal auricular beat is blocked, and subsequently the P-R interval is prolonged (0.25 to 0.27 second). In a fourth case the paroxysm (Curve *D*) was stopped by a large dose of digitalis given intravenously; the onset of normal rhythm (Curve *E*) was followed almost immediately by 2 to 1 atrioventricular block (Curve *F*). These last curves were obtained by means of a chest lead.

Partial auriculoventricular block occurs spontaneously in a few patients with auricular paroxysmal tachycardia, and it can be induced by vagal stimulation, or by digitalis, in a few others. In the great majority of all the cases in which block is present or can be induced, the P waves are similar in form to those recorded when normal sinus rhythm is present.[7] It is conceivable that in such cases the tachycardia is caused by circus rhythm involving the sinoauricular node, and that agents which tend to induce atrioventricular block do not depress conductivity within the sinoauricular node sufficiently to block the circus path. The auricular rate appears to be notably more variable in this group of cases than in those in which block cannot be induced.

It should be pointed out, however, that block cannot be induced in many cases in which the form of the P wave suggests that the paroxysmal focus lies in or near the sinoauricular node, and also that it occasionally occurs or can be induced in cases in which the form of the P waves suggests that the paroxysmal focus lies in or near the atrioventricular node.

Alternation of Cycle Length.—In a small proportion of cases of auricular paroxysmal tachycardia there occurs a slight irregularity characterized by alternation of relatively long and relatively short auricular cycles.[32] This has been observed in auricular flutter, also.[33] This phenomenon can be explained most satisfactorily by assuming that the mechanism of auricular paroxysmal tachycardia is circus rhythm.[32]

DISCUSSION

The various features of auricular paroxysmal tachycardia which have been discussed suggest that (1) the specialized nodes are involved in its mechanism, (2) the abnormal mechanism is circus rhythm, and (3) the circus rhythm is of a special kind. That the specialized nodes participate in the abnormal mechanism is suggested by (1) the form of the auricular deflections, (2) the acceleration of the auricular rate by exercise, (3) the slowing of the auricular rate and the termination of attacks by vagal stimulation and by digitalis, and (4) the rarity of atrioventricular block, especially in paroxysms arising in the region of the auriculoventricular node. That the mechanism of the disorder is circus rhythm is suggested by (1) the abrupt onset and termination of attacks, (2) the remarkable stability of the rate, (3) the slowing of the auricular rate and the termination of paroxysms by quinidine, and (4) the occasional alternation of

cycle length. That the circus rhythm is of a special kind is indicated by (1) the slowing of the auricular rate and the termination of attacks by vagal stimulation and by digitalis, (2) the acceleration of the auricular rate by exercise, (3) the relatively slow rate and long cycle length, and (4) the separation of the auricular deflections by isoelectric intervals.

A circus rhythm involving in its path one of the specialized auricular nodes most adequately accounts for these features of auricular paroxysmal tachycardia. No one of them is of itself decisive, but all together point strongly toward this mechanism. It accounts most satisfactorily for the abrupt onset and termination of the paroxysms, the stability of the rate, the form of the auricular deflections and their separation by isoelectric intervals, the relatively slow rate and long cycle length, the rarity of atrioventricular block and the impossibility of producing it in most cases, the acceleration of the rate by exercise, and the slowing of the rate and termination of attacks by vagal stimulation, digitalis, and quinidine.

It is necessary, however, to consider several exceptions which cannot readily be explained in this manner. One exception is represented by the rare cases which resemble paroxysmal tachycardia but in which the rate slows gradually to normal.[34, 35] In these cases the auricular deflections are identical with those which occur during normal sinus rhythm. They are probably not examples of true auricular paroxysmal tachycardia of the type under discussion, but rather of an unusual and persistent type of sinus tachycardia. Another apparent inconsistency is represented by those rare cases of auricular paroxysmal tachycardia in which there are auricular deflections of varying form.[7,* 27, 36, 37] They suggest that the impulses originate in two different regions of the auricles. The explanation for this is not apparent. If the mechanism in these cases is circus rhythm, the circus path in the auricles must change markedly, or there must be two different regions of circus activity. A third exception is represented by the cases in which the auricular deflections are inverted in Lead I and upward in Leads II and III. The form of the deflections suggests that they arise in the upper part of the left atrium, far from the nodes of specialized tissue. Such cases are decidedly uncommon; there were only two among our one hundred unselected cases. All of these exceptions are rare, and need not influence our main conclusions.

In the light of the available evidence, which, if not conclusive, is at least very strong, we suggest that the view that auricular paroxysmal tachycardia is caused by circus rhythm involving one of the specialized nodes—the sinoauricular or the auriculoventricular node—deserves serious consideration.

*Case 15.

SUMMARY AND CONCLUSIONS

1. Many of the important features of auricular paroxysmal tachycardia have been considered from the standpoint of their significance in elucidating the mechanism underlying this disorder.

2. The slowing of the auricular rate and the termination of the paroxysms by vagal stimulation and by digitalis and the acceleration of the rate by exercise suggest that the abnormal mechanism is influenced by the action of these agents upon one of the specialized auricular nodes.

3. The form of the auricular deflections indicates that they originate in or near these nodes.

4. The extreme rarity of atrioventricular block in cases in which there are inverted auricular deflections suggests that in these cases the auriculoventricular node is involved in the abnormal mechanism.

5. The abrupt onset and termination of the attacks, the remarkable stability of the rate, the slowing of the auricular rate and the termination of the attacks by quinidine, and the occasional alternation in cycle length suggest that the abnormal mechanism is circus rhythm.

6. The slowing of the auricular rate and the termination of the attacks by vagal stimulation and by digitalis, the acceleration of the rate by exercise, the relatively slow rate and long cycle length, and the separation of the auricular deflections by isoelectric intervals suggest that the circus rhythm is of a special kind.

7. No one of the above features is of itself decisive, but all together point strongly toward circus rhythm in the auricles, the path of which passes through one of the specialized auricular nodes, as the underlying mechanism of auricular paroxysmal tachycardia. This mechanism accounts most satisfactorily for all of the above features.

REFERENCES

1. Lewis, T.: Paroxysmal Tachycardia, Heart 1: 43, 1909.
2. Lewis, T.: The Mechanism and Graphic Registration of the Heart Beat, ed. 3, London, 1925, Shaw & Sons, Ltd.
3. Ashman, R., and Hull, E.: Essentials of Electrocardiography, ed. 2, New York, 1941, The Macmillan Co.
4. Mines, G. R.: On Dynamic Equilibrium in the Heart, J. Physiol. 46: 349, 1913.
5. Iliescu, C. C., and Sebastiani, A.: Notes on the Effects of Quinidine Upon Paroxysms of Tachycardia, Heart 10: 223, 1923.
6. Lewis, T.: Galvanometric Curves Yielded by Cardiac Beats Generated in Various Areas of the Auricular Musculature. The Pace-Maker of the Heart, Heart 2: 23, 1910.
7. Barker, P. S., Wilson, F. N., Johnston, F. D., and Wishart, S. W.: Auricular Paroxysmal Tachycardia With Auriculoventricular Block, AM. HEART J. 25: 765, 1943.
8. Lenhartz, H., and Samet, B.: Beiträge zur Kenntnis der Tachykardie, Wien. Arch. f. inn. Med. 9: 71, 1924.
9. Blumgart, H.: The Reaction to Exercise of the Heart Affected by Auricular Fibrillation, Heart 11: 49, 1924.
10. Doumer, E.: Tachycardie Paroxystique d'Origine Nodale avec Block Partiel Sous-nodal de Rythme 2/1, Arch. d. mal. du coeur 26: 579, 1933.
11. Lewis, T.: The Actions of Atropine and Quinidine in Fibrillation of the Auricles; Clinical and Experimental Studies, Am. J. M. Sc. 164: 1, 1922.

12. Andrus, E. C., and Carter, E. P.: The Development and Propagation of the Excitatory Process in the Perfused Heart, Heart 11: 97, 1924.
13. Drury, A. N., and Andrus, E. C.: The Influence of Hydrogen-Ion Concentration Upon Conduction in the Auricle of the Perfused Mammalian Heart, Heart 11: 389, 1924.
14. Cohn, A. E., and Fraser, F. R.: Paroxysmal Tachycardia and the Effect of the Stimulation of the Vagus Nerves by Pressure, Heart 5: 93, 1913.
15. Wilson, F. N., and Herrmann, G. R.: Some Unusual Disturbances of the Mechanism of the Heart Beat, Arch. Int. Med. 31: 923, 1923.
16. Lewis, T., Drury, A. N., and Bulger, H. A.: Observations Upon Flutter and Fibrillation. Part VI. The Refractory Period and Rate of Propagation in the Auricle, Heart 8: 83, 1921.
17. Wilson, F. N., and Wishart, S. W.: The Effect of the Intravenous Administration of Digitalis in Paroxysmal Tachycardia of Supraventricular Origin, AM. HEART J. 5: 549, 1930.
18. Lewis, T., Drury, A. N., and Iliescu, C. C.: Some Observations Upon Atropine and Strophanthin, Heart 9: 21, 1921.
19. Lewis, T., and Drury, A. N.: Revised Views of the Refractory Period, in Relation to Drugs Reputed to Prolong It, and in Relation to Circus Movement, Heart 13: 95, 1926.
20. Singer, R., and Winterberg, H.: Chinin als Herz- und Gefäszmittel, Wien. Arch. f. inn. Med. 3: 329, 1922.
21. Lewis, T., Drury, A. N., Iliescu, C. C., and Wedd, A. M.: Observations Relating to the Action of Quinidine Upon the Dog's Heart; With Special Reference to Its Action in Clinical Fibrillation of the Auricles, Heart 9: 55, 1921.
22. Wedd, A. M.: Notes on the Action of Certain Drugs in Clinical Flutter, Heart 11: 87, 1924.
23. Barker, P. S., Johnston, F. D., and Wilson, F. N.: The Effect of Quinidine Upon Sinus Tachycardia, Including the Production of Transient Bundle Branch Block, AM. HEART J. 25: 760, 1943.
24. Gold, H., and Modell, W.: The Action of Quinidine on the Heart in the Normal Unanesthetized Dog, J. Pharmacol. & Exper. Therap. 46: 357, 1932.
25. Kwit, N. T., and Gold, H.: Further Experimental Observations on the Combined Effects of Digitalis and Quinidine on the Heart, J. Pharmacol. & Exper. Therap. 50: 180, 1934.
26. Lewis, T.: Auricular Fibrillation and Its Relationship to Clinical Irregularity of the Heart, Heart 1: 306, 1910.
27. Parkinson, J., and Mathias, H. H.: Tachycardia of Auricular Origin and Flutter With Phasic Variation in Auricular Rate and in Conduction, Heart 6: 27, 1915.
28. Carr, F. B.: Auriculoventricular Nodal Paroxysmal Tachycardia and Auricular Flutter. Case Report, AM. HEART J. 7: 668, 1932.
29. Lewis, T.: Observations Upon Disorders of the Heart's Action, Heart 3: 279, 1912.
30. Sprague, H. B., and White, P. D.: Heart-Block During Auricular Paroxysmal Tachycardia (Clinical Observations on Three Cases), M. Clin. North America 8: 1855, 1925.
31. Brown, W. H.: A Study of the Esophageal Lead in Clinical Electrocardiography, AM. HEART J. 12: 307, 1936.
32. Barker, P. S., Johnston, F. D., and Wilson, F. N.: Auricular Paroxysmal Tachycardia With Alternation of Cycle Length, AM. HEART J. 25: 799, 1943.
33. Wilson, F. N.: Report of a Case of Auricular Flutter in Which Vagus Stimulation Was Followed by an Increase in the Rate of the Circus Rhythm, Heart 11: 64, 1924.
34. Field, H., Jr., Barker, P. S., and Alexander, J.: Unusual Sinus Tachycardia With Observations on Vagal Activity, AM. HEART J. 9: 298, 1934.
35. Maddox, K.: Auricular Paroxysmal Tachycardia (Possibly Nomotopic) With Variable Auriculoventricular Conduction Time, AM. HEART J. 14: 183, 1937.
36. Lewis, T., and Schleiter, H. G.: The Relation of Regular Tachycardias of Auricular Origin to Auricular Fibrillation, Heart 3: 173, 1912.
37. Mackinnon, A. V.: The Rhythm of Paroxysmal Tachycardia. An Electrocardiographic Study, Quart. J. Med. 27: 1, 1934.