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Reprinted from the Archives of Internal Medicine
April, 1920, Vol. XXV, pp. 420-428

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CLINICAL OBSERVATIONS ON UNUSUAL MECHANISMS OF THE AURICULAR PACEMAKER

PAUL D. WHITE, M.D.
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In the clinical records from the electrocardiographic laboratory of the Massachusetts General Hospital are some examples of unusual mechanisms of the auricular pacemaker worthy of discussion.

1. AN UNUSUAL TYPE OF AURICULAR PAROXYSMAL TACHYCARDIA

Simple paroxysmal tachycardia is defined as a tachycardia of sudden onset and sudden offset, with a striking regularity of rate, and arising from an abnormal point or ectopic focus in the auricles or ventricles. Such, indeed, is the usual occurrence. Some irritable spot in the heart takes away the initiative or impulse formation from the pacemaker or sino-auricular node in the right auricle. Whipping up the heart on its own account, it carries on this dominant rhythm till exhausted when once more the normal pacemaker resumes its work after a short pause.

Theoretically, an abnormal pacemaker like the ectopic focus is beyond nervous control and ceases its activity spontaneously. Sometimes, one finds clinically, however, that nervous influences may readily induce a paroxysmal tachycardia, and other nerve stimuli, such as pressure on the vagus nerve or eyeball, may stop it. The mechanism is not easy to explain in a number of such cases, but the important exciting factor is certainly extracardiac.

In paroxysmal tachycardia the electrocardiogram usually confirms the idea that the new pacemaker is "ectopic" by presenting a very abnormally shaped P or auricular wave. Normally, the P wave is upright in the electrocardiogram, but in paroxysmal tachycardia of the usual auricular type, the P wave is apt to be inverted.

Rarely one finds a case of auricular paroxysmal tachycardia in which the ordinary definition fails to hold. Such an instance is shown in Figures 1 and 2. Short paroxysms of tachycardia occur in which there is a markedly rapid, but not immediate, increase in the heart rate to the maximum, and a tendency to offset of the same type. Moreover, the shape of the P wave is normal or close to normal. The explanation of such a paroxysm of tachycardia is that it results from unusual nervous stimulation of the normal pacemaker or of a point in close proximity to the normal pacemaker in the sino-auricular node.

The measurements of the intervals between the heart beats in the case described here normally were about 0.77 second. The measurements between the heart beats at the beginning of the paroxysm in Figure 2 are as follows: 0.651 second (the beat preceding the par-

oxysm), 0.407, 0.425, 0.384, 0.323, 0.316, 0.325, 0.308, 0.327, 0.317, 0.336, 0.316. The measurements of the intervals at the end of the paroxysm in Figure 2 were: 0.323, 0.318, 0.310, 0.370, 0.830 (the beat after the paroxysm). At the height of the paroxysm, also shown in Figure 2, the intervals were: 0.316 second, 0.323, 0.310, 0.326. The normal rate of the heart was 78 per minute. The rate of the tachycardia at its maximum was about 188 per minute.

Case 1.—R. M., male, aged 21 years, was born in Italy. He was a worker in a jewelry shop.

Family History.—One sister was demented.

Past History.—The patient has had no serious illness in the past. He never has had rheumatic fever, chorea or tonsillitis. He always has been nervous.

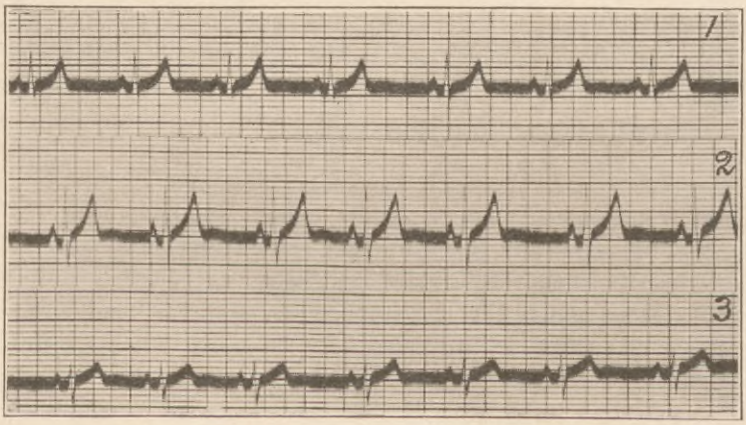

Fig. 1.—Electrocardiogram of R. M. (Case 1), showing normal rhythm in all three leads. T wave in Lead 2 is very high. Some respiratory arrhythmia is present. Lead 1 above, Lead 2 in middle and Lead 3 below. Abscissa = 0.2 sec.; ordinate = 10^{-4} volt.

Present Illness.—His present trouble, "palpitation," began seven years ago with a rapid heart beat in short paroxysms with choking sensation. He feels better now and then, and sometimes has no spells for months.

Examination.—This was entirely negative, except for frequent short paroxysms of tachycardia. The Wassermann reaction was negative. Roentgenogram of the heart was normal. Basal metabolism estimation was normal.

This patient has been under observation for five years, being first seen in October, 1914. He has constantly shown the same phenomenon, but has remained in good health otherwise; he has not had to stop work. Unfortunately, the paroxysms were too brief for experimentation, rarely lasting more than ten seconds each. While being examined under particularly exciting conditions, paroxysms occurred frequently, his heart at times beating more paroxysmally than normally.

Somewhat comparable is the voluntary acceleration of the rate of

the pacemaker as seen in published electrocardiograms.[1] In the voluntary acceleration cases, however, the increase in rate is less rapid. Carter and Wedd[2] have reported a case of paroxysmal tachycardia characterized by unusual control of the fast rhythm. The focus of the ectopic rhythm was apparently near the pacemaker and could be abolished at will, but the rate of the paroxysm was quite regular at about 200 (196 to 204) and was not under accelerator control. In the abrupt transition from an abnormal rate to normal, this case differed from the one reported here.

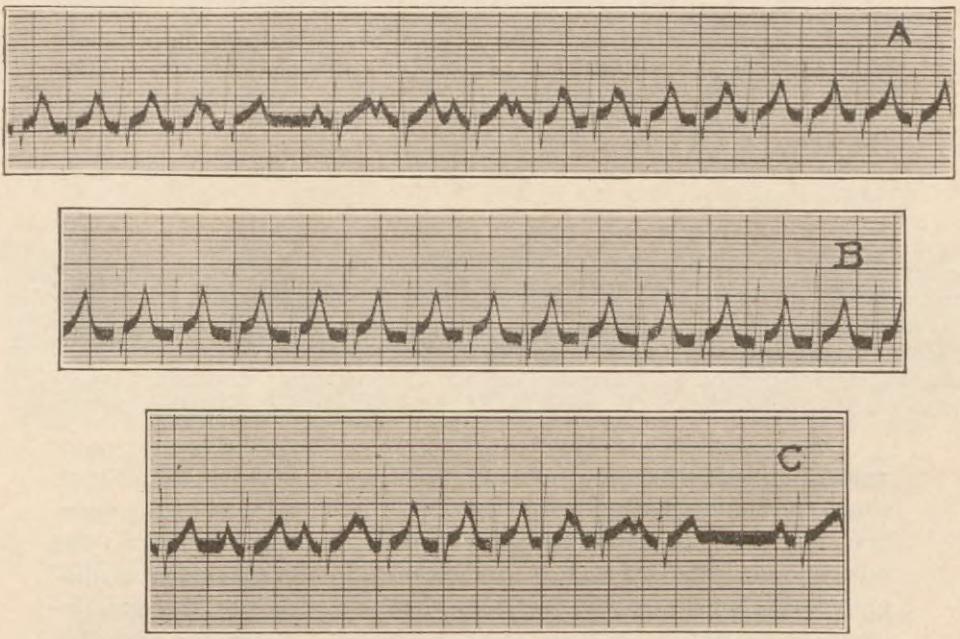

Fig. 2.—Electrocardiograms showing paroxysms of tachycardia of R. M. (Case 1). All are of Lead 2. In the upper record (A) the offset of one paroxysm and the onset of another are shown. In the middle record (B) a paroxysm appears at its height. In the lower record (C) a short complete paroxysm is photographed. Observe in this lower record particularly the gradual onset and gradual offset.

Recently, Prof. Giovanni Galli,[3] of Rome, called attention to the nomotopic (nomos, custom; topos, place) type of paroxysmal tachycardia. He writes on the manner of termination and onset of the attacks in nomotopic paroxysmal tachycardia, and gives the following points in favor of the possible diagnosis of sino-auricular paroxysmal tachycardia:

1. Favill, J., and White, P. D.: Heart **6**:175, 1917.
2. Carter, E. P., and Wedd, A. M.: Arch. Int. Med. **22**:571 (Nov.) 1918.
3. Galli, G.: Arch. d. mal. du coeur, **12**:289, 1919.

1. The sino-auricular node is composed of irritable muscle tissue. Why should it not be the source, then, occasionally of paroxysmal tachycardia?

2. Vagal excitation may influence paroxysmal tachycardia.

3. Atropin may induce paroxysmal tachycardia (Galli mentions a case in which the heart rate jumped from 90 to 200 after administering atropin).

4. Emotion may start a paroxysm.

5. The Valsalva experiment may sometimes stop a paroxysm.

6. There was a progressive increase ("crescendo") and decrease ("diminuendo") in the paroxysm in some of the tracings he obtained. Thus, there was a "parabolic development" with termination by "lysis."

7. Sometimes, at the onset of a "normal sino-auricular tachycardia" there may be a brusque change in heart rate.

8. The P wave in the electrocardiogram may appear of normal or nearly normal shape.

9. Experimental stimulation of the extracardiac nerves, for example, the right stellate ganglia, may induce a marked tachycardia.

10. Nomotopic paroxysmal tachycardia seems to have a better prognosis than the usual paroxysmal tachycardia, as one might expect.

2. VARIATION IN POSITION OF THE PACEMAKER AS SHOWN BY THE SHAPE OF THE P WAVE, THE LENGTH OF THE P-R INTERVAL AND THE HEART RATE

Lewis, Meakins and White [4] showed experimentally that the pacemaker in the sino-auricular node of a dog's heart varies its position with vagal stimulation, moving to or near the distal or tail end of the node. Its movement downward toward the ventricle tends to shorten the auriculoventricular interval or P-R interval as expressed electrocardiographically, but since ordinarily vagal stimulation depresses the auriculoventricular junctional tissues as well as the head of the sino-auricular node, the P-R interval may be actually lengthened rather than shortened.

Einthoven [5] observed the change in the shape of the P wave with prolongation of the P-R interval in vagal stimulation. Goddard [6] noted that the size and shape of the P wave may vary at different times in the same curve, and an inverted P in one record may appear upright in a later tracing, and vice versa.

Wilson [7] has discussed the mechanism of the pacemaker within the sinus node or within its immediate neighborhood, as well as its migration from the sinus to the *a-v* node, and has published electrocardiograms illustrating the migration resulting from respiratory influences.

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4. Phil. Tr. Roy. Soc. London **205**:375 (Series B) 1914.
5. Einthoven, W.: Arch. f. d. ges. Physiol. **122**:517 1908.
6. Goddard, C. H.: Arch. Int. Med. **16**:633 (Oct.) 1915.
7. Wilson, F. N.: Arch. Int. Med. **16**:86 (July) 1915; Am. J. Dis. Child. **10**:376 (Nov.) 1915.

According to theory, with the migration of the pacemaker from the head toward the tail of the sino-auricular node and to the *a-v* node, the P wave becomes flattened and finally inverted, the P-R interval becomes shorter, providing there is little or no simultaneous vagal effect on the *a-v* junctional tissue, and the heart rate drops.

Two electrocardiograms obtained at the Massachusetts General Hospital illustrate such a variation in position of the pacemaker by tendency to negativity of the P or auricular deflection and by a shortening of the P-R interval, and in one case by the decrease in the heart rate. The first instance, the type usually seen, was the result of vagal pressure in a man aged 33 years, who was seriously ill with chronic nephritis, and cardiac enlargement and weakness. The electrocardiogram, Lead 2, is shown in Figure 3. Here, right vagal pressure was applied at first in the record and then released. The P wave at first flattened, returns to a normal upright shape. The P-R interval at first measuring 0.099 and 0.098 second, becomes at the end of the record 0.146 and 0.144

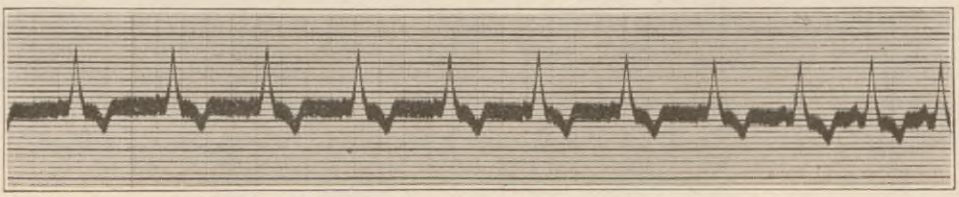

Fig. 3.—Electrocardiogram (Lead 2) of E. M. showing the effect of right vagal pressure on the auricular pacemaker. The dislocation of the pacemaker toward the ventricle is indicated by the change from normal in the shape of the P wave, the shorter P-R interval and the slower heart rate. The T wave is markedly inverted.

second. The heart rate at first 83, becomes 108. Of interest in this case is the fact that the sino-auricular node has been definitely depressed by the vagal stimulation, but the *a-v* conduction has not been slowed.

The second record is that obtained spontaneously from an elderly lady (Mrs. M.) with arteriosclerosis and an arrhythmia consisting of ventricular premature contractions. In the course of one minute, P waves of different shapes were found. The electrocardiogram, Lead 2, is shown in Figure 4. Apparently the pacemaker is moving about rapidly in the node, in the first record alternating between a high and low position, the P-R intervals alternating as well as the change in shape. The first low P carries with it an interval of 0.124 second; the next P, a much higher one, a P-R interval of 0.152 second. The following P-R intervals continue to alternate: 0.124, 0.155, 0.125, 0.155 second. The interauricular intervals are 0.989, 0.977, 0.988, 0.995 and 1.018 seconds. Finally, the inverted P waves in the second record (Fig. 4) have very short P-R intervals of 0.099, 0.103, 0.101 second. It will be noticed

that a premature ventricular beat interrupts the rhythm and is followed by a deeply inverted P wave (retrograde). It is probable that the inverted P wave arises from the region at the lower end of the *s-a* node, at the junction of the auricle with the *a-v* node, or between the two, as indicated by shape and by P-R interval. The interval between the two rhythmic inverted auricular waves in this strip is 0.866 second, which is less than that between the more normal auricular complexes in the upper record. The explanation of this is not clear, unless, perhaps, the premature contraction is disturbing the rhythm.

These records were made from a private patient during the war by Miss Mabel Hopkins, and there was no opportunity to investigate the condition further. The patient had not had digitalis. Of particular interest in this case is the alternation in the site of the pacemaker.

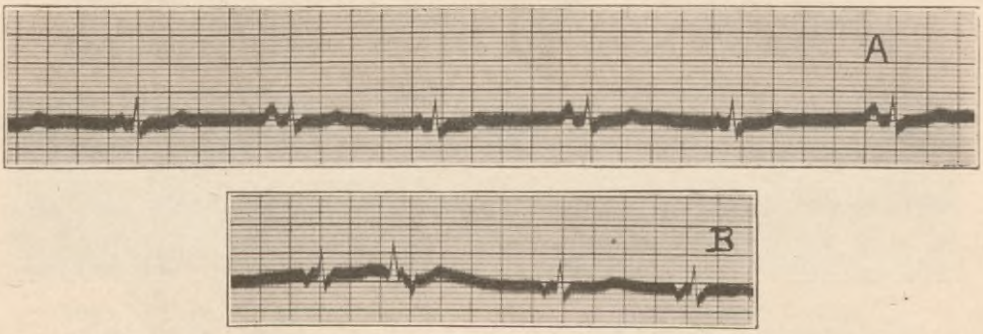

Fig. 4.—Electrocardiograms (Lead 2) of Mrs. M. (Case 3), indicating in the upper record (A) alternation in the position of the pacemaker as shown by the alternating change in shape of the P wave and by the alternating length of the P-R interval. In the lower record (B) the auricular impulse is arising from a point still nearer the ventricle as shown by the inversion of the P wave and by the short P-R interval. A ventricular premature beat with retrograde impulse to the auricles interrupts the rhythm in the lower record (B).

3. SUDDEN HALVING OF THE AURICULAR RATE ("SINO-AURICULAR HEART-BLOCK") AFTER EXERCISE

CASE 2.—W., a young man, aged 24 years, a renowned athlete in college football two years before, showed a sudden halving of the pulse rate coming on one minute after a fast run of about one-quarter of a mile. Electrocardiograms of this occurrence showed the onset of the so-called sino-auricular heart block, the sino-auricular pacemaker changing abruptly from a rate of 88.2 per minute to one of 44.8 per minute, rising shortly to 53.6 (Fig. 5). The rate remains slow. The man was healthy.

The sino auricular block in this athlete was undoubtedly due to marked vagal action following exercise. It is possible that this phenomenon occurs occasionally in athletes.

The more usual form of sino-auricular block, generally a digitalis effect due to the depression of the sino-auricular pacemaker, is shown

in Figure 6. This is an electrocardiogram of a woman, aged 33 years, with heart apparently normal, but with a cough of obscure origin. She was seen in the outpatient department of the Massachusetts General Hospital during the war. She had not had digitalis.

Levine,[8] in 1916, reported four cases of sino-auricular block seen at the Peter Bent Brigham Hospital. Three of these patients had had digitalis. He collected fourteen cases from the literature, four of which are very doubtful, because in two no graphic records were made, and in the two others only radial tracings were taken. The arrhythmia due

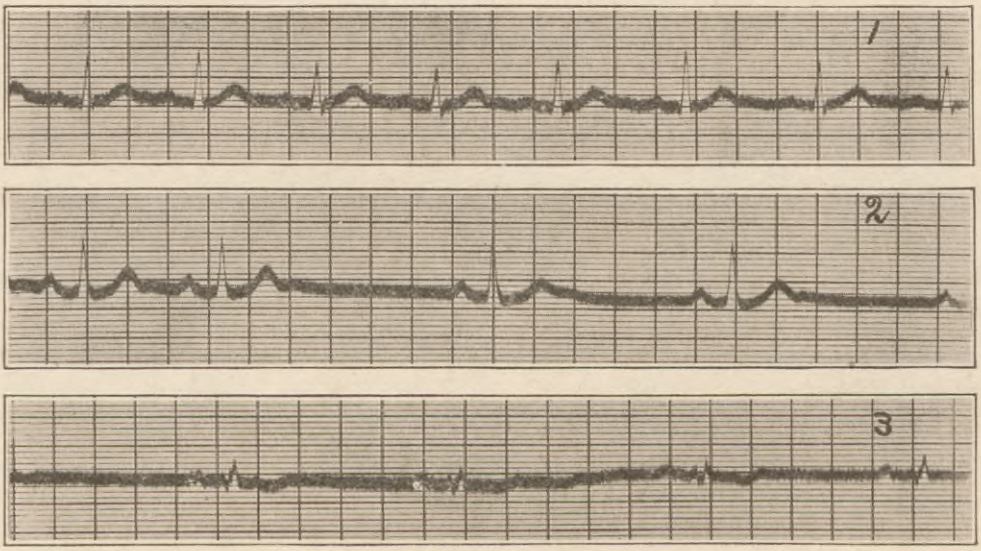

Fig. 5.—Electrocardiograms showing Leads 1, 2 and 3 of P. D. W. taken in order immediately after exercise. In the middle record occurs the sudden halving of the auricular rate (sino-auricular heart block) from 88.2 to 44.8 per minute.

to sino-auricular block was apparently the result of digitalis in seven cases, but in five others there was no statement as to whether or not digitalis had been given.

White,[9] in 1916, reported three cases seen at the Massachusetts General Hospital, in which all evidence of auricular action was temporarily abolished by digitalis, that is, auricular standstill or complete sino-auricular block was present. In another case, reported by White,[10] there was evidence of sinus bradycardia due to digitalis, in which instance the auricular rate was 41 and the ventricular rate 82 at times.

8. Levine, S. A.: Arch. Int. Med. **17**:153 (Jan.) 1916.
9. White, P. D.: Boston M. & S. J. **175**:233 (Aug. 17) 1916.
10. White, P. D.: Arch. Int. Med. **18**:244 (Aug.) 1916.

Thomas Lewis, in his "Clinical Electrocardiography," published two electrocardiograms of sino-auricular block, one from a case of mitral stenosis and one from a case of exophthalmic goiter. In 1913 [11] he reported a case of unusual bradycardia in which there was no trace of auricular activity in the electrocardiogram or jugular tracing. No statement as to digitalis administration was made in this case.

Brown [12] recently described a case of sino-atrial heart block in a child, 11 years of age, with acute arthritis. Here there had been no digitalis administration. Atropin abolished the block.

In spite of the rarity of its publication, it is probable that sino-auricular block occurs more frequently than we have been led to suspect.[13]

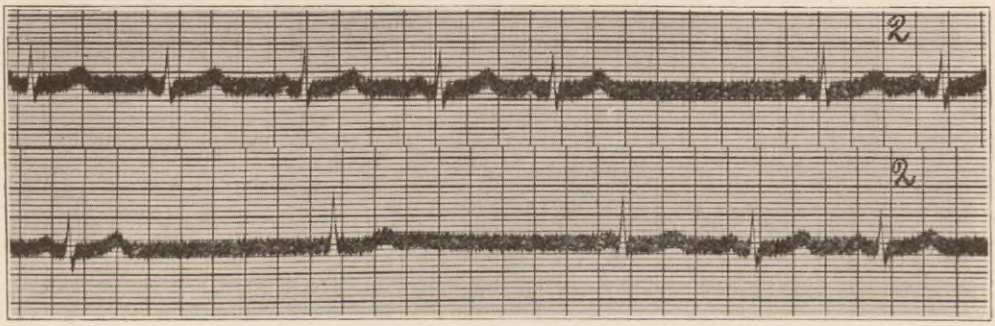

Fig. 6.—Electrocardiogram (Lead 2) of E. L. showing sino-auricular heart block, not resulting from the action of digitalis.

SUMMARY

Three clinical examples of unusual mechanism of the auricular pacemaker are reported:

1. Paroxysmal tachycardia arising in or very near the sino-auricular node and not showing an absolutely abrupt onset or offset.

2. Migration of the pacemaker in the sino-auricular node, two foci alternating action in one case.

3. Sudden halving of the auricular rate (sino-auricular block) after exercise.

11. Lewis, T.: Quart. J. Med. **6**:221, 1913.

12. Brown, N. W.: Arch. Int. Med. **24**:458 (Oct.) 1919.

13. Another case of sino-auricular block has been seen at the Massachusetts General Hospital since this article was written. This makes the seventh seen at this hospital during five years, three with complete and four with partial sino-auricular block. The patient was a woman, aged 56 years, with cardiosclerosis with slight cardiac failure; she had not had digitalis.