A CASE EXHIBITING SLOW AURICULOVENTRICULAR RHYTHM AND PAROXYSMAL TACHYCARDIA WITH UNUSUAL ABILITY TO INTERRUPT THE FAST RATE *

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INTRODUCTION

Tracings from clinical cases exhibiting auriculoventricular rhythm are comparatively rare, although the reports of Belski,¹ Cowan, Fleming and Kennedy,² and Hume³ suggest that the condition is not uncommon, at least as a transient manifestation, in certain more or less acute diseases that especially attack the heart, such as diphtheria, rheumatic fever and subacute endocarditis. It is undoubtedly rare in chronic cardiac disease, and is usually, but apparently not always, associated with extensive myocardial involvement. It may also be produced experimentally in normal persons by various disturbances of the inhibitory apparatus.

Englemann⁴ was the first to recognize auriculoventricular rhythm, following his application of the first Stannius ligature in the frog. Lohmann⁵ obtained the rhythm in the mammalian heart. Since that time, a large amount of experimental work has been done, culminating in the studies of Meek and Eyster,⁶ who proved the actual auriculoventricular nodal origin of the rhythm by the method of initial negativity. These investigators, working on dogs, concluded that the inherent automaticity of the auriculoventricular node, although subject to wide individual variations, averaged about two-thirds that of the sinus node; when impulses did not arrive from above at a more rapid rate than they were being formed at the auriculoventricular node, the latter tended to assume the cardiac autonomy.

Experimentally produced auriculoventricular rhythm may be classified into two types, according to the method by production. Type I, which has been most studied, is produced by depression or abolition

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1. Belski: *Ztschr. f. klin. Med.* **67**:67, 1909.

2. Cowan, Fleming and Kennedy: *Lancet* **1**:227, 1912.

3. Hume: *Heart* **5**:25, 1913.

4. Engelmann: *Arch. f. Anat. u. Physiol., Phys. Abth.*, p. 505, 1903.

5. Lohmann: *Ibid.*, p. 431, 1904.

6. Meek and Eyster: *Heart* **5**:227, 1914; *Arch. Int. Med.* **18**:775 (Dec.) 1916.

of automaticity at the sinus node, or interference with conduction of impulses to the auriculoventricular node. Consequently, it is a relatively slow rhythm. Type II, brought on by exaltation of automaticity of the auriculoventricular node, was first produced by Rothberger and Winterberg⁷ by stimulation of the left accelerator nerve. It is, therefore, relatively more rapid than sinus rhythm.

The above mentioned types of experimentally produced auriculoventricular rhythm, according to Lewis'⁸ classification, are of the ectopic homogenetic type. Most of the clinical cases are also of this type. The majority are due to depressive influences exerted above the auriculoventricular node, although some are doubtless aided in their production by the effect of accelerator influences on the node. As far as we are aware, no clinical case of the homogenetic type has been demonstrated to be due solely to acceleration of auriculoventricular stimulus production, although the work of Rothberger and Winterberg would seem to show that such a case would be theoretically possible.

The paroxysmal junctional tachycardias, although ectopic heterogenetic rhythms and consequently of fundamentally different character, are also classified as auriculoventricular rhythms. This group is rare, eight probable cases being reported. The characteristics of paroxysmal tachycardia distinguish them from homogenetic rhythms, but it has been impossible to differentiate absolutely some of the cases of functional origin from paroxysmal auricular tachycardia with delayed conduction, as in Lewis'⁹ second case. Lewis'¹⁰ first case and Cohn's¹¹ case, however, appear to be clear-cut and establish the condition as an entity.

The case we wish to report shows the following features of interest:

1. The length of time shifting of pacemaker back and forth between the sinus and auriculoventricular nodes was observed.
2. The variations in rate during auriculoventricular rhythm.
3. Alterations in As-Vs relations.
4. Attacks of paroxysmal tachycardia with unusual ability on the part of the patient to interrupt the fast rate.
5. The unusual behavior of the heart following the interruption of the tachycardia.
6. The resemblance of the tracings of the tachycardia, in a case which had previously shown auriculoventricular rhythm, to those of paroxysmal junctional tachycardia.

7. Rothberger and Winterberg: *Arch. f. d. ges. Physiol.* **135**:559, 1910.

8. Lewis: *Quart. J. Med.* **6**:221, 1913.

9. Lewis: *Heart* **2**:127, 1910.

10. Lewis: *Heart* **1**:306, 1910.

11. Cohn: *Heart* **2**:170, 1910.

REPORT OF CASE

Mrs. F. R., aged 62, was seen Dec. 21, 1919, at the Hospital of the University of Pennsylvania.

Present Complaint.—Palpitation of heart. The patient states that she has had attacks of palpitation of the heart since childhood. At that time she would get attacks of rapid heart beating after severe exertion. These attacks lasted a few minutes and disappeared quickly. She nevertheless took part in the ordinary plays of childhood. Gradually these attacks of palpitation increased. For the past two years she has been practically invalidated.

The attacks begin often by clutching in the throat which she says last two days. Occasionally this would arouse her out of sleep, and at that time she would have sudden palpitation. This sudden palpitation makes her faint if she attempts to rise. Occasionally she has blackness before her eyes. From the very first of these very severe attacks of cardiac palpitation, the action could be controlled if she would hold her breath for a considerable length of time. When they could not be so controlled, when they ended they would end suddenly just as suddenly as they began. Occasionally, preceding an attack, she would have dimness of vision. Sometimes the attacks would come on after walking, sometimes as has been said, in her sleep. She has had slight swelling of her legs. Vertigo rather constantly, and vertigo always accompanies the severe cardiac distress. She has shortness of breath on the least exertion lately. Within the past six months she has been considerably worse and came to the hospital for rest. She has some aching in the region of her heart but no distinct pain. If an attack of palpitation of the heart came on while she was walking, she could usually reach home from a moderate distance, but with difficulty.

Physical Examination.—The eyes are entirely normal. There is no arcus senilis. There is slight edema of the skin over the body. Nothing abnormal is noticed about the ears. There was no glandular enlargement. She had some capped teeth, but apparently the teeth in general are in good condition. Her neck and throat are normal. The chest is normal in shape. She is spare for a woman of her general size. There is marked pulsation of the vessels of the arms and neck. Expansion of chest is normal. Abdomen is normal in shape.

The cardiac impulse is feeble. The apex beat is best felt just inside of the nipple line. Dulness of the heart is at the right edge of the sternum, at the upper border of the third rib and midclavicular line. There is a loud systolic murmur over the aorta and sternum, and also over the region of the apex. This murmur is conducted into the axilla. During this examination there is no arrhythmia. The first sound is almost entirely replaced by the murmur. Second is not unduly accentuated. The lungs are entirely normal and abdominal examination is entirely normal. At this first examination her heart was beating at about 50 a minute and was perfectly regular. Jan. 6, 1920, she was seen in an attack of tachycardia. The cardiac impulse was diffuse, but the dulness was apparently not increased. On auscultation, the heart's action was regular, the murmur which was loud during the slow action of the heart was difficult to hear. She was asked to hold her breath, which she did, and immediately after one or two attempts of interruption of the rhythm, the heart slowed down and continued at the rate of about 50 a minute while the examiner was in the room.

All of the laboratory tests were normal.

Apparently, the case was one of ordinary paroxysmal tachycardia in a heart which acted slowly between the paroxysms. Again, there was the unusual ability for her to control the attack of paroxysmal tachycardia by holding her breath, and the time between the first slow beats seemed unusually long, as though some block were occurring.

Fig. 1 A.—Lead III. Taken Jan. 24, 1920. First eight cycles. Auriculoventricular rhythm, rate about 60. No P wave visible. Spontaneous short period of sinus rhythm. Only slight conductivity defect; the P-R interval does not exceed 0.20 second. Slow auriculoventricular rhythm following sinus rhythm. Patient had received atropin sulphate, $\frac{1}{100}$ grain, eight times in the preceding forty-eight hours and showed physiologic effects.

Fig. 1 B.—Lead III. Taken immediately after Fig. 1 A. Deep breath at point indicated by first arrow, held until point indicated by second arrow. Alteration in ventricular complex while breath was held. This tracing shows the typical variations in rate of the auriculoventricular rhythm and their relations to a period of sinus rhythm.

After about two weeks' rest in bed, the heart slowed down and the patient left the hospital January 31, very much more comfortable. She had not had an attack of tachycardia for two weeks.

She went from Philadelphia to Montrose in a comfortable car, developed a severe distressing cough with fever, and in about a week began to manifest signs of cardiac decompensation. She was seen in Montrose with a typical attack of decompensation, the heart beating about 160, dyspnea, large liver, edema of the lungs, and finally death occurred in two weeks after the beginning of this condition. No necropsy was held.

OBSERVATIONS ON THE SLOW AURICULOVENTRICULAR RHYTHM

Tendency for the Rhythm to Alternate Between the Sinus Node and the Auriculoventricular Node.—During the entire period of four weeks, while electrocardiographic studies were being made, a constant shifting of the pacemaker back and forth between the sinus and auriculoventricular nodes was observed, except during the periods of tachycardia. Tracings show from two to twenty cycles of normal rhythm, followed by transition to the slow rhythm, then abrupt change to the normal rhythm again. The periods of auriculoventricular rhythm observed varied from about thirty seconds to at least several minutes. Absolute rest and quiet favored longer periods of slow rhythm. Mild excitement, such as caused by applying electrodes, increased the frequency of periods of normal rhythm. Pressure on the vagi or ocular pressure sometimes cause shifting of the pacemaker back to the sinus node, but the results were inconstant. Once, a slight cough was followed instantly by return of normal rhythm, sometimes deep breathing produced the same result. Under varying amounts of atropin, the tendency for the pacemaker to shift spontaneously was not abolished.¹²

Variations in Rate During Auriculoventricular Rhythm.—The transition to auriculoventricular rhythm was sometimes preceded by a slowing of the sinus rhythm (Figs. 1 A and 1 B), sometimes it came on following an auricular extrasystole (Fig. 2 A), but frequently it occurred abruptly (Fig. 2 B) without preceding slowing of the sinus rhythm or extrasystoles. As a rule, after two or three cycles with increasing R-R intervals, the rate reached from 30 to 45, and with minor variations it tended to increase gradually, until eventually it became fairly steady around from 50 to 60, until the onset of the next period of sinus rhythm.

Owing to the spontaneous variations in rhythm, the case was not favorable for study of changes due to vagal influences. Pressure on the neck, ocular pressure and deep breathing produced no appreciable

12. Atropin sulphate was administered by mouth, $\frac{1}{400}$ grain q. i. d., for one day, and t. i. d. the next four days. All statements in respect of atropin effects refer to observations made during this period.

Fig. 2 A.—Lead II. Taken Jan. 2, 1920. First two cycles end a short period of sinus rhythm, followed by extra systole with delayed conduction. The beginning of auriculoventricular rhythm interrupted by another extrasystole. Gradual development of R-P sequence. Gradual acceleration of rate. (The patient had received tincture of digitalis, 10 minims by mouth the evening before and again on the morning this tracing was made.)

Fig. 2 B.—Lead III. Taken immediately after Figure 2 A. First seven cycles end a period of normal rhythm showing depressed conductivity. Abrupt onset of auriculoventricular rhythm. Gradually decreasing P-R intervals. Tendency toward acceleration of rate during auriculoventricular rhythm.

change except in so far as they sometimes initiated sinus rhythm. The atropin administration also was without effect apparently on the rate of the auriculoventricular rhythm.

The Relations of Auricular Systole to Ventricular Systole.—A slight depression of conductivity, which was not entirely abolished by atropin, was noted in all tracings, and at no time during sinus rhythm was the P-R interval less than 0.20 second. In Figure 2 it varies but reaches a maximum of 0.27 second. In connection with the onset of auriculoventricular rhythm, the P-R interval shows an immediate slight decrease. In general, after maintaining this relation for a few cycles, P gradually approaches R until it becomes lost in the QRS group, sometimes emerging again to the left for a few cycles (Fig. 1 B, last cycle). The changes in P-R intervals occur usually in relation to changes in rate, the intervals decreasing toward zero as the rate increases. However, this relation is not constantly observed. Figure 2 A shows R-P intervals, and is the only tracing in which they were obtained.

Neither pressure on the vagi, ocular pressure, deep breathing nor the administration of atropin produced any appreciable changes in the relation of P to R.

OBSERVATIONS ON THE TACHYCARDIA AND THE INTERRUPTIONS PRODUCED BY THE PATIENT

The tracing shown in Figure 3 was taken during one of the paroxysms of tachycardia. The rate was about 135, the rhythm regular and the ventricular complexes, with minor differences in Lead II are similar to those occurring during sinus rhythm, except for the presence of the P wave between the QRS group and the T wave.

For years the patient had been able to inhibit the tachycardia temporarily, and occasionally terminate an attack, by taking a deep breath and holding it. This maneuver was not always successful, but frequently a cycle of events, of which Figure 4 shows an example, occurred. After the breath was held for from ten to fifteen seconds, the tachycardia would terminate suddenly, sometimes with a moderately long pause followed by an isolated contraction, then an extremely long pause of from four to five seconds (Fig. 5 A), while sometimes the longest pause would occur immediately after the interruption of the tachycardia (Fig. 4). Following the long pause, contractions of supraventricular origin, but showing no evidence of P wave, occurred irregularly with single contractions or short runs of sinus origin interspersed. When the period of interruption lasted longer, a fairly regular slow auricular ventricular rhythm became established, inter-

rupted by short runs of sinus rhythm, similar in every way to the behavior of the heart observed apart from the paroxysms of tachycardia.

The position of the P' wave with reference to the ventricular complex during offset and onset and during the period of interruption is

Fig. 3.—Taken Jan. 3, 1920. Shortly after the onset of the tachycardia. P wave between QRS complex and T wave in all three leads.

of some interest. Figures 5 A and B show no evidence of auricular activity within the last ventricular complex before the offset. In Figure 5 A the first cycle after the offset has a P-R interval of 0.38

Fig. 4.—Lead III. Taken Jan. 7, 1920. Shows sequence of events following interruption of fast rhythm. Deep breath taken at point indicated by arrow. Held until patient was aware of changed rhythm. Long pause of 4.5 seconds followed by ventricular escape. No regular rhythm established. Long P-R interval at onset of tachycardia.

Fig. 5 A.—Lead II. Taken Jan. 5, 1920. Interruption of tachycardia. Last ventricular complex before offset does not show deformity due to P wave. Next contraction shows long P-R interval. The long pause lasts 4.9 seconds.

Fig. 5 B.—Lead III. Taken immediately after Figure 5 A. Interruption of tachycardia. Last ventricular complex does not show deformity due to P wave.

second. In Figure 4, during the interruption, the longest P-R interval observed in cycles of sinus origin is 0.28 second. The tachycardia is initiated again by a cycle with a P-R interval of 0.37 second.

COMMENT

The Slow Rhythm.—While frequent alternations of the pacemaker back and forth between the sinus and auricular nodes has been observed in several cases, usually, as in those reported by Lewis⁸ and Laslett,¹³ the rates of stimulus production in the two centers are fairly close, so that Laslett's suggestion of variations in vagal tone as the determining factor in the shift, might well apply. In our case, attempts at stimulation of the inhibitory apparatus appeared to have some effect, but not a constant one, in bringing about a few cycles of sinus rhythm, or at least hastening the recurrence of such a period; however, the sinus rhythm would not persist whether the stimulation be continued or not. On the other hand, attempts to remove inhibitory control by atropin did not abolish the shifting back and forth of the pacemaker. The very slow rate following the sinus rhythm revealed the fact that the ability to produce stimuli in the sinus node must have been almost, if not completely, exhausted temporarily, and despite any nervous influences that could be brought to bear, required at least some rest to bring about a restoration of the function. The main effect of nervous influences was apparently to alter the length of the period before the node became able to discharge impulses again.

Changes in relation of auricular systole to ventricular systole during auriculoventricular rhythm have been studied experimentally by several observers. Hering¹⁴ suggested that they were due to migration of the pacemaker. Zahn¹⁵ and Meek and Eyster⁶ demonstrated that migration of the pacemaker was accompanied by change in As-Vs intervals. According to the latter, coronary sinus rhythm was accompanied by only slight shortening of As-Vs time, but when the impulse arose in the lower or ventricular portion of the node, the ventricles might contract even before the auricles. Lewis, White and Meakins¹⁶ have shown that reversed block might also account for changes in As-Vs relations during auriculoventricular rhythm, not only to the extent of producing Vs-As intervals, but to failure of transmission of impulse back to the auricles. White¹⁷ was able to increase R-P intervals in his case by vagal stimulation and by digitalis, and to decrease the intervals lengthened by digitalis through

13. Laslett: *Heart* **6**:81, 1915.

14. Hering: *Arch. f. d. ges. Physiol.* **136**:446, 1910.

15. Zahn: *Ibid.* **151**:247, 1913.

16. Lewis, White and Meakins: *Heart* **5**:289, 1914.

17. White: *Arch. Int. Med.* **16**:517 (Oct.) 1915.

the administration of atropin. These effects were produced presumably by modifying reversed conduction. In those clinical cases without depression of conductivity, spontaneously changing As-Vs relations are probably dependent largely if not wholly on migration of the pacemaker. Weil¹⁸ ascribed the alterations in As-Vs relations observed in his tracings to this factor.

In our case, the facts suggest that both migration of the pacemaker and depressed conductivity are involved in the changing As-Vs relations, as seen in Figure 2 A and B. Explanation on the basis of independent auricular and ventricular contractions falling near each other is easily excluded, for even if it were granted that both auricles and ventricles might be contracting at the same slow rate, it would be unreasonable to expect them to maintain their relations in view of the irregularities in rhythm that occur.

P-R intervals up to 0.27 second during the sinus rhythm, evidence well marked depression of conductivity. The fact that P-R intervals during the early cycles of auriculoventricular rhythm may still be 0.20-0.21 second (Fig. 2 A and B) demonstrates that the principal defect in conductivity lies beyond the point of formation of impulses, consequently a slight forward block is evident. But even complete recovery of conductivity could not account for the development of the R-P intervals. On the other hand, numerous tracings taken later, when conductivity was improved, as in Figure 2, A and B, show not only shorter P-R intervals at the beginning of the auriculoventricular rhythm but no R-P relations at all. Consequently, the only explanation that seems adequate to account for the facts observed is the assumption that the region of the defect of conductivity is within the auriculoventricular node; at the beginning of auricular rhythm, it lies beyond the pacemaker and slight block is present; but by the migration of the pacemaker to lower levels of the node, it is passed and slight reversed block occurs.

The Tachycardia.—The tracings shown in Figure 3 admit of two possible interpretations: (1) paroxysmal junctional tachycardia with R-P intervals, or (2) paroxysmal auricular tachycardia with delayed conduction. The explanation is furnished by the tracings showing interruption and onset of the rhythm (Figs. 4 and 5, A and B). The last ventricular complex before offset shows no P wave as it would if the rhythm were of junctional origin, while the onset is initiated by a cycle with a P-R interval of 0.37 second, demonstrating that conduction is delayed sufficiently for the auricular systole of a particular cycle to fall within the ventricular systole of the preceding cycle during the tachycardia.

18. Weil: Deutsch. Arch. f. klin. Med. **116**:486, 1914.

The fact that conduction is so much more delayed during the tachycardia than occurred with sinus rhythm, is not to be explained in this case on the basis of depression of conductivity due to more frequent transmission of impulses. The first cycle of tachycardia shows the same marked delay in comparison with the contractions of sinus origin that occur just before it (Fig. 4). The same delayed conduction is seen when auricular extrasystoles interrupt either sinus or auriculoventricular rhythm (Fig. 2, A and B). A possible explanation is furnished by the work of Eyster and Meek,⁶ who show that conduction from the sinus to the auriculoventricular node occurs normally not by spreading of the impulse through the mass of auricular tissue, but by specialized conduction paths; while conduction by the auricle may be accomplished, it is difficult and prolonged. Consequently, whether ectopic auricular impulses are carried to the auriculoventricular node through the mass of auricular tissue, or back to the sinus node and thence through the sinoventricular conduction paths the As-Vs time might be prolonged.

The mechanism by which the patient was able to interrupt the tachycardia was not clear. It has long been known that stimulation of the inhibitory apparatus may interrupt a paroxysm of tachycardia, but as shown in the case studied by Cohn and Fraser,¹⁹ this usually occurs promptly. We were unable to initiate the change by pressure on either vagus or by ocular pressure. Carter and Wedd²⁰ exhibit tracings in which the tachycardia was thought to be interrupted by the aid of some voluntary effort on the part of the subject. In our case the fact that holding the breath for from ten to fifteen seconds at least, was necessary, raises the question as to whether slight asphyxial influences might temporarily depress the irritability of the ectopic focus in a heart with evident advanced myocardial disease and doubtless a precarious blood supply.

The unusual behavior of the heart following the interruption of the tachycardia, which, so far as we are aware, is unique, was probably due, in part at least, to the easy exhaustion of stimulus production in the sinus node, which was pointed out above as the principal factor concerned in the shifting back and forth of the pacemaker during the periods of slow rhythm. Whether still further depression of the sinus node was caused by the same mechanism that depressed the irritable focus in the auricle could not be determined. Evidently, however, the automaticity of the auriculoventricular node was markedly disturbed and required time for its recovery so that a regular auriculoventricular rhythm could be established only during the longer periods of interruption of the tachycardia.

19. Cohn and Fraser: *Heart* **5**:93, 1913.

20. Carter and Wedd: *Arch. Int. Med.* **22**:571 (Nov.) 1918.

SUMMARY

A case is reported with a history of attacks of paroxysmal tachycardia occurring over a period of more than forty years; an ability to interrupt the fast rate with temporary cessation of cardiac activity; periods of slow rhythm with interspersed short runs of about the normal rate; abundant clinical evidences of myocardial disease and decompensation.

The electrocardiographic tracings showed left ventricular preponderance, depressed conductivity, periods of sinus rhythm alternating with auriculoventricular rhythm, and paroxysmal auricular tachycardia.

Observations were made in respect of the alternations of the pacemaker between the sinus and auriculoventricular nodes, variations in rate, relations of auricular systole to ventricular systole. The significance of these phenomena is discussed.

The paroxysmal tachycardia was determined as auricular in origin by analysis of curves of offset and onset. The method by which the patient obtained interruption of the tachycardia is described, and an attempt made to evaluate the factors responsible for the behavior of the heart during the period of interruption.