REPORT OF A CASE OF PAROXYSMAL TACHYCARDIA
CHARACTERIZED BY UNUSUAL CONTROL
OF THE FAST RHYTHM *

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In view of the fact that the subject of this report possessed to a remarkable degree the power of control over his pathologic rhythm, the following instance of paroxysmal tachycardia in a young adult seemed to us of sufficient interest to report in some detail. It is greatly to be regretted that we had but a single opportunity to observe the individual for a few hours, as he left the city permanently the day after these observations were recorded.

Apart from the control of these ectopic rhythms occasionally seen following vagus stimulation either by direct pressure or reflexly as a result of various methods of clinical procedure, their voluntary control is seldom met with.

Although it is impossible in the present instance to determine with absolute exactness the means by which such voluntary inhibition is exercised, we have assumed that our use of the term "voluntary control" implies essentially the power on the part of the individual to bring about an abrupt change in his cardiac rate that is undoubtedly induced by some indirect vagal stimulation.

The subject of this note had for many years experienced attacks of abrupt, extreme acceleration of his cardiac rate, brought on at widely varying intervals by some unusual slight exertion, or often without any definitely known exciting cause, these attacks always being accompanied by the usual symptoms of distress referable to his disturbed circulation. Four or five years previously he first noticed that he had acquired the power to cut short these attacks, as he expressed it, by voluntary effort. This was accomplished apparently by a mental concentration associated frequently with an effort to yawn, or by a forced deep inspiration momentarily held.

When seen in September, 1917, by one of us, he seemed to have the power of initiating the onset of his tachycardia in much the same

* Submitted for publication July 11, 1918.

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way that he now controls its offset, without any evident muscular effort. As, however, satisfactory records were not obtained at this time we cannot say definitely that this was a correct interpretation.

At present, February, 1918, he can induce the onset of his fast rhythm by various slight abrupt muscular efforts, but no similar effort had the slightest effect in controlling the offset of the new rhythm, this being accomplished solely by some conscious subjective effort while sitting quietly with his hands and foot in the immersion electrodes. Neither yawning nor any disturbance of his respiratory rhythm alone was sufficient to abort the attack, some other factor seeming to be necessary.

Fig. 1.—Normal electrocardiogram. Lead II. Rate 108 per minute. The P-R interval measures 0.15 second. Note the premature atrial systole.

Fig. 2.—Lead II. First half of the figure during the pathologic tachycardia, rate 200 per minute. At the point marked by the arrow he was asked to stop the paroxysm. The change from the fast to the slow rate is apparent, in this instance, without any long pause. The P-R interval measures 0.15 second. Note the marked inequality in the height of the R peaks, and with the onset of the slow rate the very suggestive "staircase" phenomenon.

His history is briefly as follows:

F. P. L.,¹ aged 29, student, a Russian Jew. The family history was negative.

Personal History.—There was no history of rheumatic fever, scarlet fever or diphtheria. He had an acute short attack of influenza in 1912, but no history of any other illness. He denied any venereal infection. He had used alcohol and tobacco in moderation. He thought smoking increased the frequency of the attacks and at one time stopped smoking for nine months, but noticed no material improvement.

1. We are indebted to Dr. Harold Feil for the opportunity to observe this patient.

Present Illness.—Occurrence of attacks of sudden acceleration of his heart rate dated from 1905, when 17 years of age. These attacks lasted from a few minutes to twelve hours, the longer ones always accompanied by a sense of fulness and distress in the head. These attacks commonly followed some unusual though slight muscular effort and were much more apt to occur if he missed his midday meal or at any time if he felt hungry.

Physical Examination.—The man was of slight frame, fairly well nourished; muscular development slight; no pathologic deformities noted. The upper border of cardiac dulness was at the third interspace; the point of maximum impulse 10 cm. from the midsternal line in the fifth interspace; right border at the right sternal edge; heart sounds clear; first sound at the apex slightly accentuated with every fourth or fifth systole; no murmurs heard; pulse apparently regular in rate and volume, though varying between the two slow rhythms 85 and 106 at different times. Blood pressure 112 and 80. The man gave one the impression of being a very high strung individual with an unstable nervous balance so characteristic of his race.

ANALYSIS OF THE ELECTROCARDIOGRAMS

In a detailed study of the figures accompanying this report, and additional similar records not reproduced, a number of interesting facts are apparent.

Fig. 3.—Lead II. Fast rate 200. At a point three cycles to the left of the first cycle of this figure he was asked to stop the tachycardia. Note the abrupt change to the slow rate. The last P-R interval before the first long pause is slightly increased. The R-R interval of the first long pause measures 1.20 seconds, the R-R interval of the second pause measures 1.04 seconds. The P-R interval with the long pauses measures 0.16 second, that with the premature systole at x measures 0.320 second.

The dominant rate of the pathologic rhythm remained constantly at approximately 200 per minute, varying between 196 and 204, a rate that, according to Lewis,² approaches the borderline in the arbitrary classification between a true paroxysmal tachycardia and atrial flutter. As a simpler and more logical distinction between these two forms of tachycardia, we have considered that the failure of ventricular response, in the presence of these borderline speeds, constituted the essential distinguishing difference, a distinction endorsed by Hart,³

2. Lewis: Clinical Electrocardiography, Shaw & Sons, London, 1913.

3. Hart: Abnormalities of Myocardial Function, The Rebman Co., New York, 1917.

who further places the arbitrary difference in rate between the two at the still higher atrial speed of 250 per minute. In none of our records, however, was there at any time a failure of ventricular response during the presence of the atrial tachycardia.

THE VENTRICULAR COMPLEX

At times, but only for short intervals, the cardiac rate slowed down to 85 per minute, the dominant slow rate ranging between 106 and 108, with a normal electrocardiogram in all three leads and with a normal conduction time. The form of the ventricular complex remains the same at all speeds of the heart before, during and after atropin, showing no pathologic alteration except the alternation noted later.

In the presence of the pathologic tachycardia it is possible that confusion might arise in an analysis of the galvanometric curves as to the presence of a negative P-wave with a lengthened conduction time, were the evidence always as suggestive as is seen in the distinctly negative deflection of the string following R in Figure 9. Elsewhere

Fig. 4.—Lead II. Abrupt stoppage of the fast rate. Signal given four cycles to the left of the first cycle of the figure. R-R interval measures 1.42 seconds.

in all our records there was no such suggestive negative deflection, and we have assumed that T and P are fused, P appearing as a positive wave, as is so evident with the occurrence of isolated premature atrial contractions.

In all the records of our series there is a conspicuous variation in the height of the R peaks, amounting in many instances to a definite alternation. In Figure 2, illustrating the first recorded transition from the fast to the slow rate, the first four cycles of the slow rhythm show a very suggestive "staircase" phenomenon, which may be looked on as additional evidence of a vagus reflex.⁴

THE P-R INTERVAL

With the onset of the fast rhythm the P-R interval remains practically unchanged. With the abrupt offset of the pathologic tachy-

4. Vinis: *Heart*, 1912, **4**, No. 2, p. 123. Ritchie: *Quart. Jour. Med.*, 1912, **6**, No. 21, p. 47.

cardia to the slower rate of 106 following voluntary effort, there is but a slight change in the conduction time preceding the transition, the P-R interval lengthening for the last cycle of the fast rate to shorten again with the establishment of the slower rhythm after the pause.

When, however, he was able to slow the dominant rate of 106 down to 85 per minute, the transition was invariably accompanied by a conspicuous lengthening of the P-R interval, associated with an isolated extrasystole of atrial origin, followed by a long pause (Fig. 8). At x (Fig. 8) the P-R interval measures 0.28 sec. In another similar instance it was increased to 0.30 sec.

Fig. 5.—Lead II. Rate 200. Right vagus stimulation during the tachycardia. Vagus pressure applied at point marked by arrow. No change in rate.

Fig. 6.—Lead II. Rate 200. Left vagus stimulation during the tachycardia. Vagus pressure applied at the point marked by arrow. No change in rate.

The P-R interval then measures 0.16 sec. during the slowest rate of 85, the dominant slow rate of 106, in the presence of the pathologic cardia to the slower rate of 106 following voluntary effort, there is tachycardia and in the escape under atropin, only lengthening conspicuously, as is to be expected, with the transition from a fast to a slower rate.

EFFECT OF MECHANICAL VAGUS STIMULATION

Repeated attempts by pressure applied over both the right and left vagus failed utterly to induce the slightest change in either the rate or the cardiac rhythm. This failure to demonstrate any alteration in the cardiac response to mechanical stimulation of the vagi seems

curiously inconsistent with the results that one would have expected in an instance of the sort under consideration. That in many instances of paroxysmal tachycardia vagal stimulation is without the slightest effect is too well known to need comment, but just how to reconcile the man's own power of subjective control over his fast rhythm, so evidently of vagal origin, in the face of our failure to influence it in the slightest by vagal pressure, is not so simple.

Cohn and Fraser,⁵ in a study of paroxysmal tachycardia and the effect of vagus pressure, suggest that when vagal stimulation by pressure fails to control the paroxysmal attack and other methods succeed, the results may well be attributed to the stimulation of the inhibitory mechanism by the successful procedure and failure to do so by all other methods, an explanation that is after all not wholly satisfactory.

In a study of certain cases of rhythmic irregularities Robinson and Draper⁶ regarded the inability to produce any change in the

Fig. 7.—Lead II. Twenty-five minutes after 1.3 mg. of atropin. Rate 144 per minute. Fifteen minutes after atropin, rate 135; an hour after atropin, rate 120. Two hours after atropin the heart had escaped from atropin effect. Note the alternation.

cardiac rhythm by vagal pressure as due to an increased vagal tone; the heart in one case described by them escaping under atropin and attaining a rate greatly in excess of the rhythmic tachycardia, when the individual was no longer able to induce the changes in rhythm brought about by deep forced inspiration. They conclude that in this case the vagus tone "controlled a heart which would otherwise have beat at an abnormally rapid rate."

In this connection the experimental observations of Rothberger and Winterberg⁷ on the influence of the vagus and accelerator nerves on the galvanometric curves from the dog's heart are of interest. These observers were able to show that in the experimental animal stimulation of the vagus and accelerator yielded curves essentially the

5. Cohn and Fraser: *Heart*, 1913, **5**, No. 1, p. 93.

6. Robinson and Draper: *Heart*, 1912, **4**, p. 97.

7. Rothberger and Winterberg: *Centralbl. f. Physiol.*, 1910, **24**, 790. *idem.*, *Pfluger's Arch. f. d. ges. Physiol.*, 1910, **135**, 506.

antithesis of each other; vagus stimulation giving a low voltage P and T with a conspicuous R deflection, while stimulation of the sympathetic gave a lower R with conspicuous P and T waves. There are admittedly many difficulties in applying the experimental evidence in this connection too dogmatically to the clinical picture, and yet in those instances in which the cardio-inhibitory or accelerator influence is definitely subject to marked voluntary control the suggestive comparison of the experimental and clinical galvanometric curves should not be lost sight of.

Flavell and White,⁸ in reporting a case of voluntary acceleration of the cardiac rate not associated with any apparent change of the pacemaker, referring to the observations of Lewis and Cotton⁹ say that "the changes in the electrocardiographic deflections during the acceleration are those which are known to occur when the sympathetic is stimulated experimentally." Further, as evidence that the voluntary increase of the heart rate is the result, in great part at least, of the

Fig. 8.—Lead II. After escape from atropin effect. Note the voluntary control of the slow rate. First half of figure, rate 108; second half following the long pause, 86 per minute. The P-R interval measures 0.16 second for the first three cycles. With the extrasystole at x the P-R interval measures 0.26 second. The R-R interval following the extrasystole measures 0.96 second. The P-R interval of the next cycle measures 0.14 second and then reverts to 0.16 second.

action of the accelerator mechanism, they cite the fact that "the escape under atropin in large doses produces not one-half of the increase in pulse rate that results from voluntary acceleration." A fact, however, of even greater significance, it seems to us, in the case reported by Flavell and White, is the ability on the part of the subject of their note to break through the rate under the escape from atropin and induce a higher rate, an ability not possessed by the subject of this report.

In discussing this question as to why stimulation of the vagi by pressure is effectual in some cases of paroxysmal tachycardia and not

8. Flavell and White: *Heart*, 1917, **6**, No. 3, p. 175.

9. Lewis and Cotton: *Proc. Phys. Soc.*, June 28, 1913.

10. Robinson, G. C.: *THE ARCHIVES INT. MED.*, 1915, **16**, 967.

in others, Robinson¹⁰ says that it seems probable that this difference is due to the character of the cardiac derangement responsible for the tachycardia, depending on the anatomic relation between the distribution of the vagi and the point taking up the rôle of hyperactive pacemaker. This conclusion, so readily applicable in many instances, does not, however, aid in the explanation of the failure of vagal pressure to control the fast rate in our case, which seems so curiously paradoxical.

ESCAPE UNDER ATROPIN

Following 1.3 mg. of atropin the ventricular escape reached as its highest rate, 144 per minute (Fig. 7). The degree of ventricular escape amounted to approximately the average normal figure, being on the basis of the dominant rate, an increase of 35 per cent.

We have not assumed that the mere removal of vagal control in this instance would *per se* tend to increase the incidence of the paroxysmal attacks, but because of the very evident subjective control over the pathologic tachycardia, and in spite of the fact that it was constantly so uniformly rapid, we hoped that under the influence of atropin it might be possible to induce the pathologic rhythm with even greater facility than before its administration. While under the influence of atropin, however, he was utterly unable to break through the rate due to the cardiac escape and induce the higher speed of the ectopic rhythm. That all inhibitory vagal control had been removed was evidenced by the utter failure to control subjectively the rate under atropin, and by the conspicuous physiologic effects of the drug. Had the pathologic tachycardia been of a lower rate than that under the escape following atropin, the explanation for the failure to induce the ectopic rhythm after removal of vagal tone would be less difficult. That, however, the administration of atropin or belladonna in some form over a variable period of time to persons subject to attacks of paroxysmal tachycardia does not increase the incidence of such attacks is also a matter of observation.

The reaction to atropin in this case is further proof that we were not dealing with an ectopic focus wholly removed from neurogenic control as in the high speed atrial rate of true flutter. Following partial recovery from the atropin effect, two hours after its administration, he was again able to induce the pathologic rhythm and could again retard his slow rate of 106 down to 85 per minute (Figs. 8 and 9).

THE INCIDENCE OF PREMATURE CONTRACTIONS

As is to be expected, the incidence of single premature atrial contractions increased with the slower rates, disappearing entirely in the presence of the tachycardia. But of greater interest is the fact that

they do not disappear entirely under atropin, evidence, we consider, of definite pathologic significance. When these occur, as in Figure 1, free from any conscious effort on the part of the individual to slow his heart, the atrial pause is less than compensatory by 0.16 sec. Under the influence of conscious effort to slow the cardiac rate, as in Figure 8, the atrial pause is greatly increased, being overcompensated by 0.26 sec. There is, further, a curiously constant relationship in the time of the appearance of the atrial extrasystole to the preceding ventricular complex. Extra systoles of ventricular origin were at no time recorded.

VOLUNTARY CONTROL OF THE TACHYCARDIA

In every instance when asked to stop the pathologic tachycardia the result was accomplished within a few cycles of the command, and in every record but one (Fig. 2) the transition occurred with one or more long pauses during which the R-R interval measures all the way from 0.96 sec. to 1.42 sec. The pause involved a complete standstill of the

Fig. 9.—Lead II. Rate 200. After the escape from atropin effect. The pathologic tachycardia. Note the alternation and the very suggestive negativity of the string following the R-complex. See reference in text.

heart and frequently measured more than twice the R-R interval of the dominant rhythm of 106 to the minute. In Figure 4 it exceeds even twice the R-R interval of the slowest rate of 86 to the minute. This fact, together with the lengthening of the P-R interval that invariably accompanied isolated premature systoles, points to the predominant effect of vagus control in cutting short the fast rhythm and in slowing down the predominant slow rate of 106 to 85. That the abeyance of vagus control had not the slightest influence in inducing the paroxysmal rhythm seems as equally well established as is the evidence of the man's voluntary control over his inhibitory mechanism.

When asked to cut short the paroxysmal attack while connected with the galvanometer it was interesting to watch his method of procedure. He closed his eyes, inclined his head backward and slightly to the left and then would apparently concentrate his mind on the effort to check the tachycardia without any visible change in the respiratory rate.

SUMMARY

In view of the constant similarity of the ventricular complex, the constancy of the conduction time, except during the transition from a higher to a lower rate, together with the constant time relationship of the extrasystoles to the preceding ventricular contraction and the direction of the P-wave, we have assumed that we were dealing with an ectopic rhythm originating in the upper level of the atrium near the pacemaker, conspicuously under the control of subjective inhibitory reflexes, but utterly uninfluenced by vagal stimulation.

There is no demonstrable evidence of any accelerator influence or control over the tachycardia, in spite of the general inferences supporting such a view and the similarity of the ventricular complexes to the experimental observations alluded to.