The Syndrome of Short P-R Interval, Normal QRS Complex and Paroxysmal Rapid Heart Action

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A short A-V conduction time, whether present with normal or with abnormal QRS complex, is associated with an increased incidence of paroxysmal rapid heart action. There are a considerable number of patients who have a short P-R interval, normal QRS complex and bouts of tachycardia. They are usually females, in middle life, devoid of organic heart disease and exhibit a snapping apical first heart sound. They do not demonstrate any of the features of anomalous A-V conduction.

Evidence is presented suggesting the operation of endocrine and autonomic nervous system factors in the genesis both of the short P-R interval and the tachycardia.

In 1930 Wolff, Parkinson and White described a distinctive electrocardiographic pattern in certain patients with benign paroxysmal tachycardia. This pattern, now widely recognized, consists of a short P-R interval, usually less than 0.10 second in duration, and a prolonged QRS complex slurred on the upstroke. It was also observed that occasionally the P-R intervals and QRS complexes simultaneously revert to normal. This may occur either spontaneously or as a result of vagal suppression induced by atropine or exercise. The genesis of this syndrome is at present accounted for by the presumed existence of single or multiple accessory auriculo-ventricular conduction pathways. The short P-R interval is believed to be the result of the passage of the atrial impulse along such anomalous channels with a short-circuiting of the normal pathways. The altered configuration of the QRS reflects the aberrant sequence of depolarization of the ventricular myocardium. The extensive literature which now exists on this subject includes instances which, though possessing all other features of this syndrome, have P-R intervals of 0.12 second or more and examples with QRS complexes which, though slurred in their initial portion, are of normal duration.

In recent years we have been impressed by the frequent association of paroxysms of rapid heart action and short A-V conduction time in individuals with QRS complexes of normal duration and configuration. However, to date only 11 such cases have been reported. They have usually been regarded, without adequate justification, as variants of the Wolff-Parkinson-White syndrome. The present study has the following three objectives: first, to ascertain the incidence of paroxysmal tachycardia in patients with short P-R intervals and normal QRS complexes; second, to clarify the relation of such cases to those of the Wolff-Parkinson-White syndrome; and third, to determine whether these patients have distinct features in common which will facilitate their clinical recognition.

Incidence of Paroxysmal Tachycardia in Short as Compared with Normal P-R Groups

Material and Methods

This study is based on 200 patients with a P-R interval of 0.12 second or less, referred to as the short P-R group, and an identically selected group with normal P-R durations serving as a control. Half of the 200 cases in the control series had A-V conduction times of 0.16 and the other half of 0.18 second. Our material was obtained from 13,500 consecutive electrocardiograms taken at the Peter Bent Brigham Hospital from 1947 through 1950.
SYNDROME OF SHORT P-R INTERVAL

this time the length of P–R (or P–Q) intervals has been ascertained and noted routinely on all tracings. Our cases were obtained from such lists and all P–R durations were remeasured in the accepted manner. Case histories were not perused until after the measurement of the P–R duration; thus subjective factors were kept at a minimum during the phase of material selection. If, on remeasurement of the A–V conduction time, a patient had at least one electrocardiogram with a P–R duration of 0.16, 0.18, or 0.12 second or less, his record was studied.

The diagnosis of 700 patients showing the designated values for A–V conduction were examined. All patients with conditions that may either affect P–R duration or induce rapid heart action were eliminated. The purpose was to isolate a group in which no known factors appear to be involved that may modify the type of electrocardiogram and a tendency to tachycardia. The conditions which were thus excluded were:

1. Rate greater than 100 on all electrocardiograms with selected P–R interval.
2. No electrocardiogram available with patient off digitalis and quinidine.
3. Rapid heart action only while digitalized.
4. Rheumatic heart disease and acute rheumatic fever.
5. Thyrotoxicosis and patients on thyroid medication.
6. Coronary artery disease manifested by angina pectoris or myocardial infarction, old or recent.
7. Eisenmenger’s complex and tetralogy of Fallot.
8. Bronchogenic carcinoma.
10. Inadequate history.

Since tachycardia tends to shorten the P–R interval, patients with rates over 100 in all their tracings, even though they possessed the selected P–R’s, were eliminated from this study. The failure to obtain an electrocardiogram while a patient was not taking digitalis or quinidine or the development of rapid heart action while taking digitalis constituted a basis for exclusion. This was done not only because digitalis and quinidine affect A–V conduction time but also because of the accumulating evidence that digitalis in overdosage may induce paroxysmal auricular tachycardia. Conditions such as rheumatic fever, valvular disease and thyrotoxicosis, known either to predispose to or induce auricular arrhythmias, were eliminated. Since nodal rhythm manifested by an upright P wave in lead aVR or an inverted P in leads II and III frequently arises in a background of coronary artery disease, cases with these abnormalities were not included in the short P–R group.

Employing this process of selection, 200 patients with short P–R and 200 control patients with normal P–R intervals were chosen. Once a patient was included in the study, the medical history was carefully examined to determine the presence or absence of paroxysmal tachycardia. On the basis of these findings each case was further classified into one of three categories: (1) Those with definite paroxysmal rapid heart action, wherein electrocardiographic or medical substantiation of the recurrent tachycardia existed; (2) those with suggestive histories of bouts of rapid heart action but with inadequate medical corroboration—the mere presence of palpitation was not a sufficient criterion for inclusion in this group; and (3) those without rapid heart action.

Results

The incidence of paroxysmal rapid heart action in the short and normal P–R groups is summarized in table 1. Among the 200 cases with P–R intervals of 0.12 second or less there were 23 patients with proved paroxysmal tachycardia, while among the 200 patients with P–R intervals of 0.16 and 0.18 second there was only one such case. Histories suggestive but not diagnostic of rapid heart action were also twice as common in the short P–R as compared with the control series.

In table 1, cases in the short P–R group are also classified on the basis of the presence or absence of anomalous A–V conduction of the Wolff-Parkinson-White variety. Our definition of the Wolff-Parkinson-White syndrome included not only patients with typical “Eiffel Tower” QRS configuration, but also those with the slightest slurring of the ascending foot of the QRS irrespective of its duration and those with bundle branch block and P–R intervals of 0.12 second or less. There were four cases with bouts of rapid heart action among the 16 cases considered to have the Wolff-Parkinson-White syndrome as compared with 19 among the 184 with short P–R and normal QRS. Five of the 16 cases designated as having the Wolff-Parkinson-White syndrome had bundle branch block in conjunction with P–R durations of 0.12 second. Such cases may not be instances of anomalous A–V conduction but merely represent the superimposition of bundle branch block on a pre-existing condition of rapid A–V impulse transmission. In the former QRS prolongation is contingent upon P–R shortening, while in the latter the two are but coincidentally associated. In a
short P–R population selected as indicated, there are five times as many cases with paroxysmal rapid heart action with QRS complexes free of aberration as in a population with the Wolff-Parkinson-White configuration; this notwithstanding the higher relative incidence of tachycardia in the latter group.

The 200 short P–R and 200 control cases were compared to assess the presence of factors other than A–V conduction which may play a role in the genesis of the tachycardia. The only difference encountered in the two populations that may be of significance was a disparity of sex distribution and an unequal incidence of psychoneurosis and Addison’s disease. There were 55 per cent females in the short P–R compared with 45.5 per cent in the normal P–R group. Psychoneurosis was nearly twice as prevalent in the short P–R than in the control series, while Addison’s disease had a fourfold higher incidence in the control group. From the comparison of these populations two other differences emerge which further characterize the short P–R group. First was the relative constancy, over the course of years, of the A–V conduction time in the short P–R cases, and second was their possession of a snapping apical first heart sound. In patients with more than one electrocardiogram P–R interval variations up to 0.02 second were encountered in 20 per cent of the short P–R against 65 per cent of the control group. The intensity of the first apical sound, when commented upon, was noted as accentuated in 87 per cent of the short P–R as compared with 21 per cent of the control series.

If among patients with accelerated A–V conduction time there is an increased occurrence of paroxysmal tachycardia, a converse relation should also be apparent, namely, an augmented incidence of short P–R intervals among similarly selected patients with recurrent bouts of rapid heart action. The investigation of the validity of such a relation requires a knowledge of the usual P–R distribution in our routine electrocardiographic population. In determining this distribution we followed the criteria given above and excluded conditions which are known either to affect A–V conduction or to predispose to paroxysmal tachycardia. In 539 consecutive patients selected in this manner 6 per cent had P–R intervals of 0.12 second, 6 per cent of 0.13 and 88 per cent of 0.14 second or more. This distribution approximates that reported in the literature.18

We thereupon chose 50 consecutive cases of paroxysmal supraventricular tachycardia from the records of the Peter Bent Brigham Hospital. Again following the criteria presented above, we excluded 13 because of coronary artery disease manifested either by angina pectoris or previous myocardial infarctions. Twelve were eliminated because of continued digitalization or the absence of an electrocardiogram after cessation of the bout of rapid heart action. Ten other patients excluded had

### TABLE 1. Over-all Results: Incidence of Paroxysmal Rapid Heart Action in the 200 Cases with Short P–R Intervals and 200 Controls

<table>
<thead>
<tr>
<th>Cases with Normal QRS Complexes</th>
<th>Short P–R</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid heart action</td>
<td>19</td>
<td>1</td>
</tr>
<tr>
<td>Suggestive</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>No rapid heart action</td>
<td>190</td>
<td>192</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cases with Abnormal QRS Complexes (Wolff-Parkinson-White)</th>
<th>Short P–R</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid heart action</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Suggestive</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>No rapid heart action</td>
<td>11</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PR Interval</th>
<th>Number of Cases</th>
<th>Observed Incidence</th>
<th>Predicted Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.12 sec. or less</td>
<td>6</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>0.13 sec.</td>
<td>5</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>0.14 sec. or over</td>
<td>4</td>
<td>13</td>
<td></td>
</tr>
</tbody>
</table>

Wolff-Parkinson-White syndrome—0
either thyrotoxicosis, rheumatic heart disease or carcinoma of the lung. The results of the distribution of A-V conduction time in the remaining 15 with bouts of rapid heart action are shown in table 2. Though the sample is small, nevertheless the results are statistically significant. While the 6 per cent incidence of P-R intervals of 0.12 second duration would predict one of the 15 to have a short P–R, the actual finding was six. Similarly, instead of the anticipated one patient with a P–R of 0.13, there were five such cases. While 13 of the 15 should have had P–R intervals exceeding 0.14 only four were observed to fall in this range and it is of interest that three of these four were over 70 years of age. Since this series is small, it precludes definite judgment; it suggests, however, that shortened A-V conduction time bears intimate relation to the occurrence of paroxysmal rapid heart action.

**Comparison of Patients with Short P–R, Normal QRS and Paroxysmal Tachycardia with Those with the Wolff-Parkinson-White Syndrome**

**Material and Methods**

Thirty-four patients with paroxysmal rapid heart action, short P–R duration and normal QRS complexes were compared with 55 with Wolff-Parkinson-White configuration who also exhibited recurrent tachycardia. Nineteen of the 34 patients were obtained from our present study while 15 were found in the private files of one of us (S. A. L.). The two groups with short P–R and normal QRS were comparable in nearly every respect. To permit comparison the Wolff-Parkinson-White patients were chosen in a manner similar to those who had a normal QRS complex. The 55 with Wolff-Parkinson-White were selected from 89 cases with paroxysmal tachycardia along the criteria mentioned above. Twenty-nine of the 89 came from our files while 60 were chosen from cases reported in the literature. In order not to bias sex distribution, reports emanating from veterans hospitals or the armed forces were excluded as a source of Wolff-Parkinson-White material.

**Results**

The significant differences between patients having paroxysmal tachycardia with normal QRS complexes and those with Wolff-Parkinson-White configuration are shown in table 3. The patients with normal QRS complexes, unlike those with anomalous A-V conduction, had the onset of tachycardia a decade later in life. The most striking clinical difference in the two groups was the sex distribution. While two-thirds of the patients with the Wolff-Parkinson-White syndrome were males, two-thirds of cases with normal QRS patterns were females. The female preponderance was of the same magnitude both in the 15 private and in the 19 hospitalized patients. Among those who had descriptions in their case records of the intensity of the first apical heart sound, it was characterized as accentuated in nearly 90 per cent of those with the normal QRS complexes in contrast to 16 per cent of those with the Wolff-Parkinson-White syndrome. Premature beats, both auricular and ventricular, were

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**Table 3. Cases with Paroxysmal Rapid Heart Action.**

<table>
<thead>
<tr>
<th>Comparison of the Wolff-Parkinson-White Syndrome with Short P–R-Normal QRS Cases.</th>
<th><strong>Short PR Normal QRS (34 cases)</strong></th>
<th><strong>Wolff-Parkinson-White (55 cases)</strong>*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset of tachycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>33.5</td>
<td>22.5</td>
</tr>
<tr>
<td>Range</td>
<td>10-61</td>
<td>1-54</td>
</tr>
<tr>
<td>Sex, Female</td>
<td>70.9%</td>
<td>32%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mitral first sound accentuated</th>
<th>87% of 33 cases</th>
<th>16% of 14 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature beats</td>
<td>34% of 34 cases</td>
<td>13.5% of 37 cases</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECG</th>
<th>Slurring of R on upstroke</th>
<th>absent</th>
<th>present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of R (range)</td>
<td>0.02-0.04 sec.</td>
<td>0.03-0.011 sec.</td>
<td></td>
</tr>
<tr>
<td>Onset of R (average)</td>
<td>0.026 sec.</td>
<td>0.074 sec.</td>
<td></td>
</tr>
<tr>
<td>QRS duration (range)</td>
<td>0.04-0.08 sec.</td>
<td>0.09-0.16 sec.</td>
<td></td>
</tr>
<tr>
<td>QRS duration (average)</td>
<td>0.07 sec.</td>
<td>0.13 sec.</td>
<td></td>
</tr>
<tr>
<td>Usual PR</td>
<td>0.12 sec.</td>
<td>0.09 sec.</td>
<td></td>
</tr>
<tr>
<td>Constancy of PR</td>
<td>6% varied more than 43.7% reverted to normal conduction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Usual PJ interval</td>
<td>0.19 sec.</td>
<td>0.22 sec.</td>
<td></td>
</tr>
</tbody>
</table>

* Selected from 29 cases in present series and 60 cases in the literature. See text.
also more frequent in the group having a normal QRS.

The electrocardiographic pattern provided the main distinguishing features between the short P–R-normal QRS entity and the Wolff-Parkinson-White syndrome. In the normal QRS group the onset of the peak of the R wave was early and devoid of slurring, normalization was absent, the duration of P–R time was greater while that of the QRS and P–J interval was less than among cases with anomalous A–V conduction.

The configuration of the QRS complex was the critical point of distinction between the two groups. The presence in any case of a delta,²⁹ that is, a slurring of the initial portion of the QRS, irrespective of the length of this complex, sufficed for its inclusion into the Wolff-Parkinson-White category. We found that the measurement of the duration from the onset of the R wave to its peak served as an index of the extent of the slurring. When this measurement was 0.04 second or less no deformity of the ascending limb of the QRS was visible. The average duration from the beginning to the peak of the R wave was 0.074 second while the total QRS measured 0.13 second in the 55 patients with the Wolff-Parkinson-White syndrome. In contrast, in the 34 with normal QRS complexes the respective values were 0.026 and 0.07 second.

In selecting the short P–R patients from whom the normal QRS group with tachycardia was obtained, we arbitrarily chose 0.12 second as the upper limit for A–V conduction. It is of interest that, within this limitation, the cases with a normal QRS and tachycardia had a very narrow range of P–R distribution. Only one of the 34 patients had an A–V conduction time of 0.10 second and in no instance was it shorter. The cases with the Wolff-Parkinson-White syndrome, however, rarely had P–R intervals which equaled 0.12 second; usually their P–R intervals were 0.10 second or less.

The constancy of P–R interval on repeated electrocardiograms in the same individual is a feature of the patients with normal QRS complexes and bouts of rapid heart action. Only six of the 34 patients under study showed a spontaneous variation in the P–R interval greater than 0.01 second on repeated electrocardiograms. In some instances, the time interval between tracings was as long as 10 years. Such stability was absent in patients with the Wolff-Parkinson-White syndrome, 48 per cent of whom showed “normalization.” This refers to the reciprocal relation between the P–R and QRS durations manifested by the simultaneous lengthening of the former interval when the latter is shortened. In patients of the normal QRS group the P–R varied independently of the QRS which usually remained fixed in duration.

The P–J interval is another feature reflecting the difference between the two categories of short P–R. The P–J interval is measured from the inception of the P wave to the junction between either the R or S wave and the base line. The P–J interval in patients with Wolff-Parkinson-White syndrome is of normal duration and relatively constant irrespective of whether the underlying mechanism is one of either normal or anomalous conduction. In the normal QRS group the P–J was short, with an average value of 0.19 second. In the 34 cases it never exceeded 0.20 second, while patients showing Wolff-Parkinson-White syndrome usually had P–J intervals above this figure. In any one case with a normal QRS the P–J seldom varies; this is due to the relative constancy of the P–R interval.

The electrocardiogram of the patient with short P–R, normal QRS and paroxysmal rapid heart action is easily distinguished from that of the patient whose tracing shows the features characteristic of the Wolff-Parkinson-White syndrome. An occasional case defies categorization. Figure 1 represents the spectrum of QRS variation encountered among patients with short P–R intervals and paroxysms of rapid heart action. The tracing at A is a typical example of the syndrome under discussion, distinguished by a normal QRS complex devoid of slurring and a short P–R interval. The tracing at B demonstrates the characteristic “Eiffel Tower” pattern of the Wolff-Parkinson-White syndrome. The distinct delta in the tracing at C defines it as a case of anomalous
A-V conduction, notwithstanding the fact that the duration of the P-R and QRS are 0.11 and 0.09 second respectively. The tracing at D represents a case of short P-R (0.12 second) in combination with right bundle branch block. While this type of configuration has been regarded as a variant of the anomalous A-V conduction pattern, it may be an example of the entity being described wherein bundle branch block has supervened. This is the type of pattern which, in the absence of evidence of a reciprocality between P-R and QRS dura-

Fig. 1. Four patients with tachycardia and short P-R but with differing QRS configurations. A. Normal QRS complex. The patient was a 39 year old woman who experienced repeated paroxysms of auricular tachycardia. Electrocardiograms between bouts of rapid heart action taken 10 years apart were nearly identical. B. Typical example of the Wolff-Parkinson-White syndrome. In lead III spontaneous normalization for one beat is present. C. QRS of normal duration, but with a definite delta. D. Right bundle branch block.

tions, eludes classification. The large majority of patients with short P-R and tachycardia, however, can easily be grouped into either the Wolff-Parkinson-White or the normal QRS category.

Clinical Features of Patients with Short P-R, Normal QRS and Paroxysmal Tachycardia

In a group of subjects with short P-R intervals and normal QRS complexes, selected as indicated, 10.4 per cent had recurrent rapid heart action. The majority of the patients with tachycardia were females in middle life who were precipitated by noncardiac ailments, trauma or anesthesia; prior to the onset of tachycardia, however, they already exhibited short P-R intervals in their electrocardiograms. In the majority of the patients under 40, no precipitating causes or predisposing factors for the tachycardia were evident. In one patient a severe emotional shock launched the first bout of rapid heart action, but subsequent episodes came without any preceding emotional upheavals. In another patient tachycardia occurred only during menstruation and in three rapid heart action appeared to be triggered by asthmatic attacks. Familial factors existed in two patients: one had a sibling who also showed first apical heart sound. While the average age of onset of tachycardia was 33.5 years, in a significant number the inception was late in life. In 14 of the 34 the first manifestation of tachycardia occurred after the age of 40. In eight of these rapid heart action was first noted after the age of 60. While palpitation was the chief and at times the sole complaint in 20 or 58 per cent of the patients, in the nine who had the onset of tachycardia after the age of 40 no preceding history of palpitation was obtained. In this older group bouts of rapid heart...
recurrent rapid heart action; the other, a middle-aged woman, had a son with tachycardia and a short P–R interval.

The tachycardia, once it appeared, recurred sporadically with intervals between attacks varying from a few hours to many years. The longest history of recurrent rapid heart action was 60 years and was noted in two patients. The most frequent arrhythmia was paroxysmal auricular tachycardia. Twelve patients exhibited this type of rapid heart action, six experienced bouts of auricular fibrillation, four had flutter and one had nodal tachycardia, while another had paroxysmal auricular tachycardia with block. Three patients had more than one type of rapid heart action. The remaining patients had supraventricular tachycardia of unspecified type. The majority of patients tolerated the rapid heart action well. This was due, in all likelihood, to the absence of underlying organic heart disease. Two patients, however, died suddenly. Both of these were subject to paroxysms of auricular fibrillation. No postmortem examinations were available to ascertain whether death was due to pulmonary embolism.

The customary methods of prevention and control of attacks were employed in these cases. Prevention proved more difficult than control. Maintenance of digitalis or constant quinidine administration decreased the incidence of paroxysms in many instances. The greatest harm in the care of these patients is the frequent attribution of coronary artery or arteriosclerotic heart disease as the cause of the recurrent tachycardia. The realization that the tachycardia is self-limited in duration, of benign genesis and usually without deleteri-

Fig. 2. Electrocardiograms of a 50 year old woman (Peter Bent Brigham Hospital #172332) who complained of palpitation for 10 years, demonstrated to be due to paroxysmal auricular tachycardia. Except for a P–R interval of 0.12 second her electrocardiogram is within normal limits.

Fig. 3. Electrocardiogram of a 13 year old girl (S.A.L. # A8660) who had paroxysmal auricular tachycardia for three years. The P–R had a duration of 0.12 and the QRS of 0.06 second.
of any slurring on its upstroke is corroborative.

Discussion

The difference in incidence of rapid heart action in the series of patients with short P-R intervals and in the control series with normal P-R intervals, even after the exclusion of all cases with the Wolff-Parkinson-White syndrome from the former, is statistically significant. The question still arises whether the two populations compared are not artefacts of selection. A concrete answer to the problem is contained in the excluded population. Three hundred sixty patients were excluded from this study because they had conditions which either modified the P-R duration or predisposed to rapid heart action. About half of these patients had short P-R and half had normal P-R intervals. If defective methods of selection had been employed, by means of which most of those with paroxysmal rapid heart action and short P-R were included while the majority of those with normal P-R and rapid heart action were excluded from this study, then the incidence of recurrent tachycardia in the excluded control population would have exceeded significantly that in the excluded group with short P-R intervals. This excess would have roughly approximated the preponderance of patients with tachycardia in the short P-R as compared with the control group under study. This was not the case. The frequency of paroxysmal rapid heart action was similar in the two excluded groups irrespective of the length of the P-R interval. The elimination of known causes of rapid heart action, therefore, accentuates the relation between shortened A-V impulse transmission and recurrent tachycardia. We dwell on this point because to date, except for the special phenomenon of anomalous A-V conduction, no general recognition of such a relation exists. The reason for this has been failure to exclude all conditions which may precipitate tachycardia when the role of short P-R is under investigation. In our study, among 380 cases with short P-R intervals and among an equal number of controls there were 74 patients with paroxysmal rapid heart action, 46 in the former and 28 in the latter category. In the selected group under study, however, among the 200 patients with short P-R intervals, there were 23 with tachycardia compared with one among the 200 patients with normal P-R durations. Obviously when exclusion of other causes of tachycardia is not carried out, the role of the P-R interval is inundated among a host of other factors.

Our data do not establish whether the high incidence of tachycardia in patients with P-R durations of 0.12 second or less is a special feature for this range of A-V time or is merely a reflection of an augmented frequency of tachycardia as the P-R shortens. We arbitrarily chose 0.12 second as the upper limit for our short P-R category. For adults this is regarded by Scherf as the lowest normal value. It may be that a similar incidence of tachycardia exists for patients with P-R intervals of 0.13 second. The evidence available at present suggests that with shortening of the P-R interval there is an increasing incidence of rapid heart action. Among 200 patients with P-R intervals of 0.16 and 0.18 second the frequency of bouts of tachycardia is 0.5 per cent. Among 184 patients with P-R intervals ranging from 0.10 to 0.12 second it is 10 per cent, while the reported incidence of recurrent tachycardia in large series of subjects with the Wolff-Parkinson-White syndrome, generally having P-R intervals of 0.08 to 0.11 second, is given as 70 per cent. It may be that the tachycardias occurring in patients with Wolff-Parkinson-White syndrome are not special results of anatomic anomalous A-V pathways, but merely the accentuated manifestation of the operation of the same physiologic factors as in those instances where A-V conduction time is shortened.

Until this present study 11 patients have been described in the literature with short P-R intervals, normal QRS complexes and recurrent tachycardia. In a paper on paroxysmal tachycardia by Wedd, which appeared in 1921, a typical case with this syndrome is presented. This article is of historic interest since it reports the second case with the distinctive features which have now come to be recognized as characteristic of the Wolff-Parkinson-White syndrome, and, to our knowl
edge, the first case of the entity which we are describing.

In 1938 Clerc and co-workers\textsuperscript{9} described three such patients with short P–R, normal QRS and rapid heart action. These three were obtained from 21 cases with short P–R intervals who were free of stigmas of relevant heart disease. This is approximately the same incidence of tachycardia which we found in our much larger series of patients with short P–R intervals. Over half of the 21 patients of Clerc and associates suffered from palpitation. Seven who were available for long-term follow-up studies showed no alteration of their A–V conduction time. These authors also noted that in two instances vagal stimulation and exercise and in one case digitalis did not alter the P–R duration or the QRS configuration. The seven other patients described in the literature\textsuperscript{10–14} have been considered by the authors as variants of the Wolff-Parkinson-White syndrome. They have been categorized as examples of either the normal or the anomalous conducting phase of Wolff-Parkinson-White syndrome. The following facts, however, are against their being manifestations of the “normalized” phase: (1) the failure to observe the aberrant stage of conduction in those patients followed for many years; (2) the lack of reciprocal changes in the QRS duration in those few patients exhibiting P–R interval prolongations; (3) the higher incidence of patients with short P–R, normal QRS and tachycardia compared with those with the Wolff-Parkinson-White syndrome. One would anticipate a random distribution of P–R durations during the “normalized” phase of the Wolff-Parkinson-White syndrome, and therefore only about 6 per cent would have P–R intervals of 0.12 second.

An alternate explanation is that these are manifestations of the Wolff-Parkinson-White syndrome during the aberrant phase of A–V conduction. Burch\textsuperscript{32} suggests that the accessory bundle in these patients terminates in the interventricular septum near its base or in the bundle of His. On the basis of earlier views\textsuperscript{2, 3} of the pathogenesis of anomalous conduction, it was possible by “positioning” the single bundle of Kent to explain nearly all short P–R tracings as those of the Wolff-Parkinson-White syndrome. Current views, however, cast doubt on the existence of a single, discrete, short-circuiting path resulting in pre-excitation with ensuing asynchrony in both action potential production and mechanical systole of the two ventricles. Glomset and Glomset\textsuperscript{32} have shown that most mammalian hearts have multiple muscular bridges between auricles and ventricles. Rosenbaum and his associates\textsuperscript{38} by means of chest and esophageal leads were the first to suggest that pre-excitation occurs simultaneously in both ventricles. Grishman and co-workers\textsuperscript{39} with the aid of intracardiac and esophageal electrocardiography confirmed and further extended these findings. Their observations indicate that the abnormally propagated impulse reaches simultaneously the anterior surface of the right ventricle and the posterior epicardial and endocardial surfaces of the left ventricle. Conduction through the normal pathways apparently does not occur. Hemodynamic studies\textsuperscript{41} in patients with Wolff-Parkinson-White syndrome, by means of right heart catheterization, show a delay in the interval between the Q wave of the electrocardiogram and both the inception of right ventricular systole and the onset of the pressure rise in the brachial artery. Electrokymographic studies\textsuperscript{33, 34} further confirm these findings by the demonstration of delayed and synchronous pulmonary and aortic systolic impulses in patients with the Wolff-Parkinson-White syndrome. The paradoxical clinical finding of a normal or muffled first apical heart sound in the Wolff-Parkinson-White syndrome, notwithstanding the ultra short P–R, is in accord with the above observations. Normally the P–R duration is closely correlated to the intensity of the first heart sound.\textsuperscript{37–39} The shorter the interval, the more intense is the first heart sound. In the Wolff-Parkinson-White syndrome, notwithstanding the short P–R, there is a delay in ventricular systole caused by the aberrant slow ventricular depolarization. The delayed conduction permits the A–V valves to float up, with a maintenance of the normal intensity of the first heart sound. Current theory holds, therefore, that the Wolff-Parkinson White syndrome is due to a filtering down of
auricular impulses along many tracts with pre-excitation of both ventricles. The impulses are propagated aberrantly through the ventricles and result in simultaneous but delayed ejection of blood into the pulmonic and aortic circuits.

Wolff and White\textsuperscript{1} have regarded these cases of short P–R and normal QRS as instances of anomalous conduction wherein the speed of impulse transmission in the accessory tracts approximates that of the normal pathway. The fulfillment of such a condition would obviate the slurring of the ascending limb of the QRS as well as the reciprocal changes in P–R and QRS duration observed in patients with the Wolff-Parkinson-White syndromes. To sustain such a supposition three further assumptions are necessary: first, that the abnormal pathways have relatively slow rates of conduction; second, that these rates of conduction are nearly identical in all the different muscular connecting bridges; and third, that the normal pathways have relatively rapid conduction times approximating those of the accessory tracts. However, the independence of relation between A–V conduction and QRS duration in some of these patients indicates the operation of a mechanism other than anomalous conduction. In a patient currently under study (not included in the series), vagal stimulation by carotid pressure, parasympathomimetic drugs, atropine, exercise and quinidine caused no significant alteration in the P–R time. With a marked overdosage of digitalis, A–V time was lengthened to 0.15 second; however, the QRS remained fixed in duration. Immediately after a bout of paroxysmal auricular tachycardia with a rate of 190, the P–R interval was observed to be 0.14 second; again the QRS remained unaltered.

A further difficulty in the categorization of these patients as having Wolff-Parkinson-White syndrome is the short duration of their QRS complex. If these cases had usual P–R intervals of 0.13 or 0.14 second and an abnormal pathway conducting at 0.12 second, one would expect the QRS in a large series of these patients to be slightly longer than normal. During aberrant impulse propagation the increment of P–R shortening would be added to the QRS length. The 34 patients with short P–R, normal QRS and tachycardia had an average duration of the QRS complex of 0.071 second. For the 165 patients with short P–R but without tachycardia it was 0.073, while for the 200 patients with normal P–R intervals it was 0.079 second. If the condition of short P–R and normal QRS is due to the presence of congenital aberrant auriculoventricular communication, tachycardia might be expected to appear early in life as in the Wolff-Parkinson-White syndrome. This, however, was not the case. In nearly half it occurred after the fortieth year and in one-fourth of the patients after the sixtieth year of life. The incidence of males with Wolff-Parkinson-White is 70 per cent, while the incidence of males with short P–R, normal QRS and tachycardia is 30 per cent. This striking sex discrepancy further points to a fundamental difference between the two conditions.

Our conclusion, therefore, is that the combination of short P–R, normal QRS and bouts of rapid heart action constitutes a distinct entity and is not an expression of anomalous conduction. The increased incidence of tachycardia in both conditions may, however, be a reflection of those changes brought about by, or associated with, the shortened A–V conduction time.

Some authors have considered electrocardiograms with positive P waves in the limb leads and P–R intervals of 0.12 second or less as a type of coronary sinus nodal rhythm.\textsuperscript{40–42} Scherf and others\textsuperscript{43, 44} regard this as regular sinus rhythm and require deeply inverted P waves in leads II and III for the definition of coronary sinus rhythm. However, even if the former criteria for coronary sinus rhythm are entertained, patients with this form of nodal rhythm differ in many respects from the patients with short P–R intervals and tachycardia. This type of nodal rhythm usually is associated with a P–R of less than 0.10 second, the heart rate is under 50, the nodal rhythm almost invariably is transient and when permanent reflects serious myocardial injury; furthermore, it is not accompanied by paroxysms of rapid heart action.

A study of the conditions that predispose
to shortened A-V conduction time sheds some light on the possible background factors of the syndrome of short P-R, normal QRS and recurrent rapid heart action. During infancy the P-R interval is short and assumes an adult pattern at about the time of puberty.\textsuperscript{45, 46} Söderström\textsuperscript{13} in 30 patients with short P-R intervals observed an increased occurrence of hyperthyroidism and acute myocardial infarction. In the latter condition the short P-R interval was observed during the early stages of the infarction and subsequently it increased in length. In 84 patients with acute coronary thrombosis whom we recently studied\textsuperscript{47} we noted that nearly one-fourth of the patients had transient shortening of A-V time to levels of 0.12 to 0.13 second. This occurred during the first week of the illness. Scherf\textsuperscript{48} found hypertension in 29 out of 49 patients with P-R intervals of 0.11 second or less. Two of the patients with short P-R had hyperthyroidism. He concludes that a short P-R is associated with a "marked hypermotility and hypercontractility of the heart."

Thorn, Dorance and Day\textsuperscript{49} have shown that 21 per cent of patients with Addison’s disease who show some abnormality in the electrocardiogram have first degree heart block. This has been confirmed by Sommerville and associates.\textsuperscript{50} In a preliminary study we find that nearly all addisonian patients have P-R durations of 0.16 second and over; while patients with Cushing’s syndrome have P-R’s of 0.14 or less. Furthermore, in a number of patients now under observation, the administration of cortisone or adrenocorticotropic hormone (ACTH) has caused a shortening of the P-R interval. In the treatment of acute rheumatic fever with cortisone or ACTH one of the earliest changes in the electrocardiogram is a decrease in the A-V conduction time. The conditions which are reported to be associated with a short P-R are known to exert potent stress on the adaptive processes of the body. In some patients anxiety neurosis is accompanied by evidence of increased adrenocortical activity manifested by a chronically depressed fasting eosinophile count.\textsuperscript{51} This is of interest in the light of Ruskin’s observation\textsuperscript{52} that there is a frequent co-existence of mental disease and a short P-R interval without P-wave abnormality. In a recent report Rovrik and Aarstrand\textsuperscript{53} have concluded that shortening of the P-R interval is one of the characteristics of the electrocardiogram in the neurotic female. This is in agreement with our observation. In the short P-R series neurosis was twice as common as in the control population. Since the incidence of neurosis among the males was the same in the two groups, the increase is accounted for by a marked preponderance of females with neurosis and short P-R.

Another mechanism invoked to explain the co-existence of short P-R and neurosis is increased sympathetic tone which is a resultant of the state of chronic tension. Such an explanation may be utilized also to account for the association of short P-R and paroxysmal rapid heart action. It is fairly well established that sympathetic stimulation is associated with a shortening, while parasympathetic stimulation is associated with a lengthening, of the P-R duration.\textsuperscript{54, 55} Standing, which increases sympathetic tone, may shorten the P-R duration in some individuals\textsuperscript{56, 57} and may precipitate paroxysmal tachycardia which is relieved by lying down.\textsuperscript{58} Cervical and upper dorsal sympathectomy has been utilized to do away with troublesome recurrent supraventricular tachycardias.\textsuperscript{59} Forty years ago Rothberger and Winterberg\textsuperscript{60} induced auricular fibrillation by the simultaneous stimulation of the sympathetics and the vagus. The intravenous administration of small amounts of acetylcholine and adrenaline will likewise produce auricular fibrillation in a high percentage of cases.\textsuperscript{61} Many of the conditions reported to shorten the P-R duration such as hypertension, thyrotoxicosis and anoxia increase sympathetic tone as a part of the homeostatic response of the individual. Classic physiologic studies have furnished evidence that there is a sympathetic center in the hypothalamus. Bronk and his colleagues\textsuperscript{62} have induced impulses in efferent sympathetic nerves during hypothalamic stimulation and conversely showed rhythmic variations in potential in the hypothalamic nuclei by the stimulation of efferent nerves possessing sympathetic fibers. Deduc-
tions from animal experiments have led to the supposition that emotional instability is associated with hypothalamic overfunction due to a release from higher cortical centers.\textsuperscript{62} The presence of "autonomic imbalance" has therefore been utilized to account for the short P–R interval in mental disease.

Hume and Wittenstein\textsuperscript{41} have presented evidence that the hypothalamic centers have a regulatory influence over pituitary adrenocorticotropic activity. Thus the intermediary pathways of hypothalamic activity, be they endocrine or sympathetic nervous system in mechanism, tend to shorten the P–R interval. Furthermore, there is a great deal of data, both of a clinical and experimental nature, to suggest that the hypothalamus may initiate some cardiac arrhythmias.\textsuperscript{65–66} The hypothalamic lead of the electroencephalogram is abnormal in patients with paroxysmal rapid heart action when there is no heart disease; however, when there is organic disease of the heart and tachycardia the electroencephalogram is normal.\textsuperscript{69} The evidence presented invites the speculation that in certain individuals chronic stress with resultant hypothalamic discharge activates the adrenal cortex and the sympathetic nervous system. These in turn sustain shortened A–V conduction times and with periodic augmentation of stress these mechanisms trigger outbursts of tachycardia. While such a hypothesis is highly presumptive, it opens possibly fruitful avenues for the investigation of the genesis of the syndrome of short P–R, normal QRS and paroxysmal rapid heart action.

**Summary and Conclusion**

1. In a selected group of 200 patients with short P–R intervals there were 23 patients or 11 per cent with paroxysmal tachycardia compared with 1 or 0.5 per cent in a similarly selected control group of 200 with normal P–R durations.

2. Among the 200 patients with a short P–R interval, 184 had normal QRS complexes and 16 had Wolff-Parkinson-White configurations. The incidence of paroxysmal tachycardia was 10.4 per cent in the former group and 25 per cent in the latter.

3. Of the 23 patients with recurrent bouts of rapid heart action and short P–R, 82 per cent had normal QRS complexes while 18 per cent possessed the features of the Wolff-Parkinson-White syndrome.

4. The patients with a short P–R interval, normal QRS complex and bouts of rapid heart action exhibit a different electrocardiographic pattern from patients with the Wolff-Parkinson-White syndrome. Their QRS, unlike that of Wolff-Parkinson-White, is devoid of slurring and does not exceed 0.08 second in duration; they do not show normalization and have shorter P–J and P–R intervals which are strikingly constant over the course of years.

5. Short P–R, normal QRS and recurrent tachycardia is a distinct and easily recognizable clinical entity. It occurs predominantly in the female sex, the tachycardia in half the patients begins after the fourth decade of life, a third of the patients have premature beats and the majority show an accentuation of the first apical heart sound. In these respects, this entity further differs from Wolff-Parkinson-White syndrome.

6. Evidence is presented that the mechanism underlying short P–R, normal QRS and tachycardia is not one of anomalous A–V conduction and pre-excitation as is believed to be the case in the Wolff-Parkinson-White syndrome.

7. The implication from this study and from pertinent medical literature would lead one to explore the possible relationship between the endocrine system (particularly the adrenals) and autonomic nervous system on the one hand and the shortening of the P–R interval and paroxysms of tachycardia on the other.

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