Studies on the Mechanism of Ventricular Activity

XIV. Clinical and Experimental Studies of Accelerated Auriculoventricular Conduction

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Seven clinical cases with electrocardiographic patterns of constant or variable short P-R interval are reported. The QRS complex was either normal or aberrant. These abnormalities were reproduced experimentally in dogs by the injection of various drugs into the region of the A-V node. The theories advanced to explain these phenomena are discussed. The results indicate that accelerated A-V nodal conduction is responsible for the short P-R interval. The form of the ventricular complex may be dependent upon synchronous or asynchronous activity of the A-V node. A classification of A-V nodal dysfunction is presented.

Prolongation of the P-R interval was observed in the early days of electrocardiography, and the mode of occurrence has long since been established. The short P-R interval, however, was clinically recognized only recently and there is still disagreement as to its underlying mechanism.

The major portion of the P-R interval has been demonstrated by Osborne and associates to be a function of the A-V node; that is, the node normally delays the transmission of the impulse from auricle to ventricle. The most commonly recognized example of the short P-R interval is the Wolff-Parkinson-White (W-P-W) syndrome. In 1944, Ohnell summarized the theories that had been presented to explain the short P-R interval seen in the Wolff-Parkinson-White syndrome. At present, the theories most commonly advanced are (1) the existence of a muscular or neuromuscular pathway, or pathways, between the auricle and the ventricle, (2) pre-excitation of a ventricular focus produced by electrical or mechanical stimulation from the auricles, and (3) acceleration of conduction time through the A-V node.

We have recently observed cases in which the P-R interval was abnormally short, of constant or varying duration, and associated with normal or abnormal QRS complexes; the abnormal QRS complexes were narrow or wide. These cases are presumably not too uncommon, since six of the seven cases herein reported were observed in the ordinary course of private practice within a period of a few months. These cases are obviously not of the classic type of Wolff-Parkinson-White syndrome. Their clinical recognition and experimental reproduction constitute the subject of this paper.

Clinical Data

Short P-R Interval, of Varying or Constant Duration, with Normal QRS Complexes

Case 1. D. P., a 16 year old boy, was seen for cardiac evaluation because of palpitation. The only positive findings disclosed by physical examination were a sinus arrhythmia and a grade I apical systolic murmur varying with respiration. Roentgenographically, the heart was well within normal limits. The electrocardiograms (fig. 1A and B) revealed a sinus arrhythmia, with the rate ranging between 82 and 88 per minute. The P-R intervals varied

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from 0.06 to 0.14 second. There was no relationship between P-R duration and R-R interval. The duration of the QRS was 0.07 second and configuration remained unchanged despite variations of the P-R interval. At the beginning of deep inspiration (fig. 1B) two A-V nodal beats with retrograde auricular conduction and inverted P waves were recorded in lead II, followed by resumption of the auricular pacemaker.

Case 2. W. T., a 49 year old white woman, was seen because of palpitation and backache. The past history, family history, functional inquiry and physical examination were noncontributory. Emotional instability, attributed to menopause, was noted.

Standard, unipolar and precordial leads (fig. 2) revealed a normal sinus rhythm (rate, 94 per minute) with P-R intervals unvaryingly of 0.11 second and QRS complexes of 0.04 second.

Case 3. J. P., a 56 year old white woman, was admitted to the hospital because of myocardial infarction. The patient had experienced chest pain on exertion, extending to the left arm and neck, for about 14 years. On the day of admission she had sudden onset of severe, persistent chest and neck pain, extending down the left arm, which necessitated admission to another hospital. Two weeks later she was transferred to this hospital. The only positive findings were an accentuated second aortic sound and a sedimentation rate of 28 mm. in 1 hour (normal, 0 to 20 mm.).

The electrocardiogram taken before myocardial infarction (fig. 3A) revealed normal sinus rhythm (rate, 75 per minute). The P-R interval was 0.16 second and the QRS complex 0.05 second. Shortly after infarction the electrocardiogram (fig. 3B) revealed sinus rhythm (rate, 68 per minute); the P-R interval varied from 0.12 to 0.04 second and the QRS was 0.06 second. Two weeks after myocardial infarction the electrocardiogram (fig. 3C) showed a normal sinus rhythm (rate, 75 per minute); the P-R interval was 0.14 second and the QRS 0.06 second.

Case 4.* W. S., a 21 year old white soldier, was

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* We are grateful to General Elbert DeCoursey, Director of the Armed Forces Institute of Pathology, for permission to report this case.

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Fig. 1 (Case 1). Electrocardiograms of a 16 year old boy with no evidence of organic heart disease. (A) Leads I, II and III recorded at paper speed of 25 mm. per second. The P-R intervals vary from 0.06 (X) to 0.15 (XX) second. The QRS duration is within normal limits (0.06 second) and configuration is not altered by the short P-R intervals. Marked sinus arrhythmia. The rate varies between 52 and 88 per minute. (B) Lead II, after deep inspiration. The third and fourth complexes (N) are of upper nodal origin with retrograde auricular conduction. They are followed by resumption of the auricular pacemaker.
admitted to Walter Reed Army Hospital from Camp Drum Army Hospital on June 28, 1952, with the complaint of difficulty in hearing since November 1950. Physical examination at Camp Drum had disclosed a cardiac arrhythmia. On admission to Walter Reed the patient stated that at the age of 17 he first experienced precordial aching associated with dyspnea, both of which followed exertion and disappeared with rest. On two occasions in 1950, associated with a severe “chest cold,” he had hemoptysis of one cupful of bright red blood. At the time of physical examination the patient was in no apparent distress. The heart was not enlarged. The second pulmonic sound was split and louder than the second aortic sound. The blood pressure was 115/75. Results of the remainder of the physical examination were essentially normal. Laboratory tests, including urinalysis, serologic test for syphilis, blood count and sedimentation rate, were all within normal limits. Chest x-ray and fluoroscopy showed “minimal overlapping of the spine, in the left oblique, by the left ventricle, indicating slight enlargement of the ventricle.” The heart size was increased 15 per cent over normal. The pulmonary artery shadow was prominent.

The numerous electrocardiograms (some of which are shown in fig. 4) were described as abnormal. Figure 4A reveals a sinus arrhythmia (rate, 97 to 110 per minute); the P-R interval is 0.20 second and the QRS 0.06 second. Figure 4B shows, in lead I, a sinus rhythm; the P-R interval varies from 0.06 to 0.21 second, and the QRS is of normal duration. In lead II, the second to seventh beats are nodal in origin and are associated with retrograde P waves and gradual lengthening of the R-P interval (reversed Wenkebach phenomenon). The initial beat in lead II shows a P-R interval of 0.12 second. The P-R interval following the return to sinus rhythm is 0.24 second. Lead aVf is similar to lead II.

While in the hospital, the patient suddenly died. Autopsy revealed hypertrophy of the right ventricular wall (7 mm.) and dilatation of the right ventricle immediately inferior to the pulmonary valve. The left ventricular wall measured 15 mm. in thickness. There were no valvular abnormalities. The pulmonary conus showed a minimal degree of dilatation. Microscopically the only finding in the myocardium was multiple small areas of infiltration from recent hemorrhage, with no associated inflammatory reaction. Examination of the lungs disclosed generalized atelectasis. There was extensive hyaline thickening of alveolar septal walls. Marked thickening of some of the arteriolar walls, principally intimal, was noted; in many instances the lumina of these arterioles were almost completely obliterated. The lesions were extremely patchy in distribution. The pathologist’s diagnosis was “early primary pulmonary arteriosclerosis.”

The foregoing cases illustrate that the P-R interval may be abnormally long (heart block) or abnormally short. The QRS complexes may be of normal duration and/or configuration.

**Fig. 2.** (Case 2). Electrocardiograms of a 49 year old woman with no evidence of organic heart disease. Leads I, II, and III recorded at paper speed of 25 mm. per second. The P-R intervals are short (0.11 second) and of constant duration. The QRS is of normal duration (0.06 second) and configuration. Normal sinus rhythm; rate, 94 per minute.

**Fig. 3 (Case 3).** Electrocardiograms of a 56 year old woman before, during and after posterior myocardial infarction. (A) Lead II, before myocardial infarction. The P-R interval is 0.16 second and the QRS duration 0.05 second. Normal sinus rhythm; rate, 75 per minute. (B) Six hours after myocardial infarction. The P-R interval varies from 0.12 to 0.04 second. QRS duration is 0.06 second. Sinus rhythm; rate, 68 per minute. (C) Two weeks after myocardial infarction. The P-R interval is 0.14 second. QRS duration is 0.06 second. Normal sinus rhythm; rate, 75 per minute.
The duration of the short P-R interval may be variable (cases 1, 3 and 4) or constant (case 2). It is of interest that abnormally short P-R intervals may at other times be associated with partial heart block and A-V nodal rhythm in the same patient (case 4). The electrocardiographic deviations from normal in these cases may result from acquired disease (cases 3 and 4). Such deviations may also occur in the absence of any clinically demonstrable cardiac pathology as in cases 1 and 2. In these two instances there may have been a functional abnormality of the A-V node without structural changes.

Short P-R Interval, of Constant or Varying Duration, with Aberration of the QRS Complex

Case 5. R. C., a 22 year old white woman, was seen on Nov. 30, 1953, for cardiac examination. She
stated that several years previously, when she was being investigated for a gynecologic disorder, she was told that she had "heart disease." Since then she had had occasional episodes of palpitation. The past history, family history and functional inquiry were noncontributory. Chest x-ray and routine laboratory tests were all within normal limits.

The electrocardiogram shown in figure 5A, recorded two minutes after exercise, shows sinus tachycardia (rate, 125 per minute). The P-R interval is 0.14 second and the duration of the QRS complex 0.06 second. At X there is a single beat in which the P-R interval is 0.10 second and the QRS is 0.11 second and of abnormal configuration.

In figure 5B, recorded shortly after the electrocardiogram reproduced in figure 5A, lead II shows an alternating rhythm; each normal complex is followed by a QRS of abnormal configuration. The P-R intervals of the normal beats vary from 0.14 to 0.16 second and the QRS measures 0.06 second. In the abnormal complexes (X) the P-R interval is sometimes not measurable, because the P wave blends with the upstroke of the QRS complex. When measurable, the interval varies from 0.06 to 0.10 second, and the QRS complexes are obviously wider.

The tracings shown in figure 5C and D (lead II) exhibit normal sinus complexes (S), A-V nodal complexes (N) and alternating, premature ventricular complexes (V). Conducted auricular beats show a P-R interval of 0.21 second. Many of the premature complexes are followed by an upright P wave.

Case 6. R. H., a 50 year old white man, was admitted to the hospital because of sudden onset of severe, oppressive, substernal pain, associated with profuse sweating and profound weakness, which began one hour prior to admission. The blood pressure was 170/120, and there were moist rales at both lung bases posteriorly. The results of the remainder of the physical examination were negative. Urinalysis showed a specific gravity of 1.022, 3 plus acetone and 3 plus albumin; there were 1 to 2 white
blood cells, 1 to 2 hyaline casts and a few red blood cells per high power field. The blood count showed 20,100 white cells, with a differential count of 90 per cent polymorphonuclear leukocytes, 5 per cent lymphocytes and 2 per cent monocytes. The sedimentation rate was 13 mm. in 1 hour (normal 0 to 9 mm.), and the fasting blood sugar level was 140 mg. per 100 cc. A diagnosis of acute myocardial infarction was made.

The electrocardiograms, of which two leads are illustrated (fig. 6A), reveal shortening of, and variation in, the P-R interval, associated with aberration of the QRS complex. The rate is 68 per minute; the P-R interval is 0.08 to 0.18 second, and the QRS is 0.06 to 0.12 second. Figure 6B reveals an alternating rhythm with short P-R intervals of varying duration, and aberrant QRS complexes of the alternate beats. The normal P-R interval is 0.16 second and QRS duration is 0.06 second. The P-R interval of the aberrant complexes varies from 0.07 to 0.13 second, and the QRS is 0.12 second. This rhythm reverted to normal after a short period of time and remained normal.

Several months later, while in another city, the patient had another episode of coronary occlusion, which resulted in his death. We were fortunate in being able to obtain the heart for histologic examination, which was done by the Lev technic. The pathologist* reported that, in the node, "the amount

* The slides were examined by Dr. Harry Goldblatt, to whom we express our thanks.
associated with profuse sweating and weakness. The pain was relieved by administration of 200 mg. of Demerol, and he was admitted to the hospital. Physical examination revealed tachycardia (rate, 116 per minute) and numerous ventricular extrasystoles. The blood pressure was 110/80. There were no other abnormalities noted on physical examination. Routine laboratory tests were within normal limits.

The electrocardiograms (fig. 7A) reveal short P-R intervals of varying duration (0.09 to 0.16 second), associated with wide and aberrant QRS complexes (0.12 second). In the electrocardiogram taken after recovery (fig. 7B) the P-R interval and QRS complex have reverted to normal (P-R, 0.16 to 0.20 second and QRS, 0.08 second).

The cases in this group illustrate that a short P-R interval may be associated with aberrant QRS complexes. In case 5 there were short P-R intervals of varying duration with normal and aberrant QRS complexes, nodal beats and ventricular extrasystoles, without any other clinical evidence of myocardial disease. At other times this patient manifested evidence of partial heart block with prolongation of the P-R interval. In cases 6 and 7, short P-R intervals with aberrant QRS complexes were noted in association with myocardial infarction. These three cases illustrate that the phenomenon may be associated with acquired cardiac disease (cases 6 and 7) or may exist in the absence of any clinically demonstrable heart disease (case 5).

The short P-R intervals with normal or abnormal QRS complexes observed in the clinical cases reported herein were produced in dogs by experimental alteration of the A-V node. Nodal rhythms and heart block, which occurred in some of the patients, were also produced by altering the A-V node in dogs. It is suggested, therefore, that the phenomena found in these patients may result from A-V nodal disturbances.

Experimental Study

Materials and Methods

Experiments were carried out in 40 dogs. Each animal was anesthetized with Pentothal or Nembutal administered intravenously. The animal was placed in a supine position and a control electrocardiogram taken. Tracheotomy was then performed. In some experiments the chest was opened through a right lateral incision in the third or fourth intercostal space. A bilateral incision was employed in other

![Fig. 8. Illustration of technic of injection into the A-V node through the body of the right auricle. For orientation the needle is advanced through the tricuspid valve until contact is made with the interventricular septum (I.V.S.). It is then withdrawn into the auricular cavity and directed caudally toward the coronary sinus (C.S.) until contact is made with the A-V node (A.V.N.).](http://circ.ahajournals.org/)

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experiments. After the chest was opened and the anterior pericardium removed, a second control electrocardiogram was made.

All tracings were made on a direct-writing Sanborn Poly-Viso electrocardiograph. Continuous recordings were made throughout each experiment, usually at a paper speed of 50 mm. per second. In addition, recordings were made on the Sanborn Twin Beam electrocardiograph, which has a frequency response of 500 cycles per second. The results were the same as with the Poly-Viso electrocardiograph.

In the earlier experiments, lead II was recorded simultaneously with aVR or a direct auricular lead. Four simultaneous leads were recorded during the later experiments, usually including leads I, II, III and aVR or a direct auricular lead. In several instances lead II and a direct auricular lead were registered simultaneously with direct right and leftventricular leads. The direct auricular leads were recorded with a cotton-tipped electrode sutured to the posterior auricular wall in the region of the coronary sinus.

The experimental procedure consisted of injecting the region of the A-V node with cocaine, formaldehyde or acetylstrophanthidin. These were chosen for the purpose of anesthetizing, destroying or depressing A-V nodal tissue. Two techniques of injection were used:

1. A 22 or 23 gage needle, attached to a tuberculin syringe, was inserted through the hind portion of the right auricle and advanced through the tricuspid valve to the right side of the interventricular septum. The needle was then withdrawn into the auricular cavity and directed caudally and posteriorly until the A-V node was located (fig. 8).

2. The needle was inserted into the cavity of the right auricle through a purse string opening in the right auricular appendage. Then, by directing the needle cephalad from the coronary sinus, the A-V node could be reached.

Contact with the node was manifested by the occurrence of transient heart block of first, second or third degree, or nodal arrhythmias of short duration. After the position of the needle had been verified by these electrocardiographic changes, approximately 0.25 to 0.50 cc. of drug was injected into the A-V node. Upon completion of each of the experiments, the animal was sacrificed and the heart opened in order to verify further the site of injection. In al

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**Fig. 9.** Effect of injection of cocaine in the A-V node of a dog. (A) Control. Sinus rhythm, rate, between 96 and 106 per minute. The P-R interval is 0.12 second and the QRS 0.03 second. Paper speed, 50 mm. per second. (B) Continuous strip. Lead II. After injection of 3 per cent cocaine in the region of the A-V node. There is gradual lengthening, shortening and then lengthening of the P-R interval. The rate is 84 per minute. The P-R interval ranges from 0.04 to 0.13 second and the QRS is 0.04 second. Paper speed, 50 mm. per second.
experiments reported, a grossly edematous and hemorrhagic area approximately 5 mm. square was visible in the immediate vicinity of the A-V node between the coronary sinus and the posterior cusp of the tricuspid valve. Sections from five hearts were examined by the pathologist, using Lev's technic. These microscopic examinations confirmed the gross observation that the chemicals had been injected into the A-V node.

Thirteen of the 40 dogs received 20 per cent, 5 per cent or 3 per cent cocaine hydrochloride. In 7 of the 13, 20 per cent formaldehyde was injected after the effects of cocaine had disappeared. Twenty-one dogs were given 20 per cent formaldehyde alone. In the remaining six animals, 0.33 to 1.0 cc. (1 to 3 cat units) of acetylstrophanthidin was injected into the A-V node. Ten of the animals were used as controls; cocaine, formaldehyde or acetylstrophanthidin, in concentration similar to that injected into the A-V node, was injected peripherally into the free wall of the ventricles and into various parts of the interventricular septum.

Results

Control electrocardiograms with the chest closed or open were essentially normal, except that inversion of the T wave followed opening of the chest. After injection of cocaine or formaldehyde into the A-V node, variable shortening of the P-R interval was frequently observed. The P waves in standard limb leads were usually identical with, or only slightly different from those in the control electrocardiograms. The shortening of the P-R interval was correlated with changes in the P-R segment rather than with changes in duration of the P waves.

The results obtained in only 11 of the dogs are reported; the remaining animals developed heart block, nodal rhythms or nodal arrhythmias, or the short P-R intervals were not persistent for a long enough period of time to be considered significant. In the group reported, the pattern of varying short P-R intervals appeared within several seconds to very few minutes after the injection into the node and persisted from one to several minutes. After normal sinus rhythm recurred, the short P-R intervals could often be reproduced by a second injection.

Cocaine. The seven dogs that received injections of cocaine into the A-V node presented short P-R intervals of varying duration with QRS complexes of normal duration. In two of these animals (fig. 9A and B) gradual shortening, lengthening and then shortening of the P-R interval occurred within a period of several beats. There is a definite similarity between the pattern occurring in this animal and that in case 1 (fig. 1). The remaining five dogs presented short P-R interval variations with no apparent pattern. Although the duration of the P-R interval usually changed with each cardiac cycle, occasionally it remained constant for a few beats (fig. 10A and B). In three of the seven animals, the QRS complex was normal in configuration as well as in duration. In four animals the shape and amplitude of the QRS were altered (fig. 10A and B), but the duration remained within normal limits.

In two animals, 2:1 heart block followed the injection of cocaine into the A-V node. In one of these the QRS was of normal duration and
configuration, while in the other the QRS was aberrant. The P-R interval of the conducted beat was extremely short in both animals (figs. 11 and 12).

Another point in figure 12 is noteworthy. By examining only lead II, one might diagnose the abnormality as first degree heart block with extremely long P-R intervals. It is only by examining the auricular lead that one observes a P wave immediately preceding the ventricular complex. The P wave is responsible for the initial portion of the R wave in lead II. Because the P-R interval is so short, the auricular and ventricular complexes in this lead appear fused. An important way of determining that the aberrant ventricular beat is preceded by a P wave is by accurate measurement of the P-P interval. It will then be found that the ventricular complex starts with a P wave.

Formaldehyde. Among the three animals that received formaldehyde injections and exhibited short P-R intervals, the shape of the QRS complex was normal in one and altered in two. Although the P wave in standard limb leads always remained upright, it was lower than normal in two dogs and higher than normal in one. In two dogs the P-R interval was short and varying in duration and was associated with an aberrant QRS complex (fig. 13); note the similarity to case 6 (fig. 6A, lead V6).

Acetylstrophanthidin. One of the animals that received acetylstrophanthidin presented a very short P-R interval of unvarying duration,
followed by second and first degree heart block. As the heart block became less severe it was frequently associated with isolated beats, runs of beats or alternating complexes manifesting short P-R intervals, of constant or varying duration, and associated with either normal or aberrant QRS complexes.

**DISCUSSION**

In our experiments, alteration of the A-V node resulted in three phenomena: (1) heart block, (2) short P-R intervals and (3) nodal rhythms and arrhythmias. The short P-R interval was of constant or of varying duration, associated with normal or abnormal QRS complexes. These phenomena are comparable to those seen in the clinical cases. The pattern of constant short P-R interval was experimentally reproduced in one dog by injection of acetylstrophanthidin in the region of the A-V node. The varying type followed injections of cocaine or formaldehyde into the same region. These results are consistent with the clinical observation that short P-R intervals are frequently associated with acute myocardial disease involving the A-V node, such as acute rheumatic myocarditis and acute posterior myocardial infarction. Three cases have been reported in which acquired and permanent Wolff-Parkinson-White syndrome was associated with histopathologic changes in the region of the A-V node. Since there was no obvious cardiac pathology in some of our cases (cases 1, 2 and 5), it would appear that a dysfunction of the A-V node could be responsible for the electrocardiographic phenomena even in the absence of a definite lesion of this structure. In the one case in this series in which autopsy was done, the alterations in the A-V node were considered to be at the upper limit of normal. However, the autopsy was performed several months after the episode that was manifested by short P-R intervals in the electrocardiogram. It is probable that more significant pathologic changes might have been present during the acute episode.

**Causes of Short P-R Interval**

Various theories have been proposed to account for the occurrence of a short P-R in-
interval (0.12 second or less) in the clinical electrocardiogram. These theories will be discussed in relation to the results reported in this paper.

Autonomic Influences. In the first report of an electrocardiogram showing a short P-R interval associated with an abnormal QRS complex,\(^9\) the phenomenon was considered to be due to vagal influence. This explanation was also advanced by Wolff, Parkinson and White, in their original article.\(^1\) Since then there have been published many reports of the occurrence of short P-R intervals with normal QRS complexes in a variety of clinical syndromes: hyperthyroidism,\(^9\) hypertension,\(^1\) acute coronary thrombosis,\(^12\) the neurotic state\(^13,14\) and associated with paroxysmal tachycardia.\(^15\) The variation of the vago-sympathetic tone was considered responsible for the shorter auriculoventricular conduction time.

It is well known that hypothalamic or drug-induced sympathetic stimulation results in sinus tachycardia and relative shortening of the P-R interval. In our clinical experience, however, when there is variation of what is referred to as “vago-sympathetic tone,” the P-R interval generally remains longer than 0.12 second. None of the drugs used in our experimental procedures has a known sympathomimetic effect; the effect of digitalis is mainly vagotonic. There was no indication in our experiments that the autonomic nervous system factor was responsible for the shortening of the P-R interval.

Ectopic Auricular Focus. Some workers have attributed the short P-R interval with a QRS complex of normal duration to the presence of a low ectopic auricular focus. Various labels have been used, among them, paranoval rhythms,\(^16\) parasinus rhythms,\(^17\) and coronary sinus rhythms.\(^18\) All these terms imply that the impulse arises near the A-V node rather than the S-A node. Since such impulses have a short distance to travel before reaching the A-V node, these workers ascribe the shortening of the P-R interval to the shortened conduction time. The theory of the low ectopic auricular focus is based on the belief that the configuration of the P wave in the standard leads is not influenced by the location of the pacemaker in the auricle, but rather by the direction of spread of the impulse from the ectopic focus to the A-V node.

It has been clearly demonstrated that a caudad auricular pacemaker would be manifested by an inverted P wave in leads II and III.\(^7\) Thus the upright P waves in our cases, both clinical and experimental, are not of low auricular origin. Moreover, since the rate of aurricular depolarization averages about 1000 mm. per second,\(^19\) it is estimated that activation of the A-V node occurs 0.05 to 0.06 second after depolarization of the sinoauricular node. Thus, shortening of the P-R interval by more than 0.04 to 0.05 second could not occur even if the impulse were initiated as close to the A-V node as possible.

A wandering auricular pacemaker, whether or not associated with transient nodal rhythm, will manifest itself by different configuration of the P waves in the standard leads and by shortening of the P-R interval. A wandering pacemaker obviously did not occur in our cases, as the P wave remained unaltered.

Ventricular Type of Short P-R Interval. Others have stressed the possibility that a hyperexcitable septal or ventricular focus is prematurely discharged under the mechanical or electrical influence of auricular systole.\(^3\) This would explain the short P-R interval and the QRS complex of long duration and abnormal configuration, comparable to an extrasystolic complex. This theory is based on neurophysiologic principles and is supported by experimental observations in animals and in humans. Such complexes have been produced by injection of alcohol, adrenaline or silver nitrate into the septum,\(^20\) by mechanical stimulation of the anterobasal part of the septum,\(^21\) and by injection of strychnine into the wall of the ventricular myocardium;\(^22\) in fact, they have been produced by electrical, mechanical and chemical stimulation of any part of either ventricle.\(^7\) In human subjects anomalous atrioventricular excitation has been produced by stimulation of the ventricular septum during cardiac catheterization.\(^23\)\(^24\)

Variants of the theory of the idioventricular complex have been proposed, such as iso-
rhythmic dissociation, 25 "phénomène d’accrochage"25b and regular interference between auricular and ventricular foci. 26

There is no question that complexes of this type occur when the ventricle is stimulated from any ventricular focus. In our experience this is of frequent clinical occurrence. In these cases, a very long electrocardiographic strip must be taken. Although many, or most, of the aberrant beats will be preceded by P waves, one will find QRS complexes without preceding P waves. In other words, these patients frequently have ventricular extrasystoles and ventricular tachycardia. These aberrant QRS complexes will have the same configuration as the QRS complexes associated with the short P-R interval. The configuration of the aberrant QRS complex depends upon which ventricle is stimulated first: In lead I, the major ventricular deflection will be inverted if the left ventricle is stimulated and upright if the right ventricle is stimulated. It is possible that the aberrant QRS complexes and the short P-R interval in case 7 were of this type, because in other electrocardiograms similar aberrant QRS complexes occurred without preceding P waves.

In the experiments reported in this paper, great care was taken not to stimulate the ventricle during the procedure, and all recorded QRS complexes were preceded by P waves.

Aberrant Anatomic Pathways. For more than 20 years, the existence of one or more muscular or neuromuscular accessory bridges, extending from the auricle to the ventricular myocardium, has been the most generally accepted physiopathologic explanation for the occurrence of short P-R intervals with normal or abnormal QRS complexes.

The termination of the aberrant pathway in the ventricular myocardium would short-circuit the delaying A-V node and cause preexcitation of the affected ventricle. The terminal phase of ventricular systole would be the result of the impulse traveling along the normal auriculoventricular pathway. A shorter P-R interval associated with an aberrant QRS complex would thus be produced.

The pattern of short P-R intervals with QRS complexes of normal duration is identified as a variant Wolff-Parkinson-White syndrome by only a few workers. 3, 27, 28 They base their interpretation on a few scattered observations of abnormal pathways terminating in the interventricular septum, 3, 29, 30 so located as to permit the impulse to bypass the A-V node and motivate the common bundle of His. Stimulation of the ventricles thus would follow the normal sequence, yielding a QRS complex of normal duration.

The following evidence has been presented in support of the theory of anomalous atrioventricular conduction: Histologic examination of the auriculoventricular groove in a few clinical cases of Wolff-Parkinson-White syndrome revealed the presence of aberrant muscle bundles connecting the auricle and the ventricle. 3, 31, 32 These reports constitute the major basis for the wide acceptance of the theory of anomalous atrioventricular conduction. The ingenious experiments of Butterworth and Poindexter 33 suggested the functional significance of such an aberrant pathway. They established an electrical circuit between the auricle and the corresponding ventricle in dogs and cats; a typical Wolff-Parkinson-White complex was produced by the application of electrical current to the ventricle.

It should be pointed out that, in some clinical cases of Wolff-Parkinson-White syndrome, no aberrant pathways could be demonstrated histologically. 34, 35 On the other hand, aberrant connections have been found in the normal heart in a variety of species. 38 In a patient with the Wolff-Parkinson-White syndrome, there is, thus far, no way of ascertaining that an anomalous anatomic connection, if present, is responsible for the syndrome. Despite the lack of direct evidence, the theory has gained wide acceptance, principally because it explains beautifully the abnormal complexes.

Accelerated Conduction. As a result of the demonstration that the function of the A-V node is to delay the transmission of the impulse from auricle to ventricle, 2 it was theorized that, if this function could be altered, the impulse might not be delayed to the same
extent; the P-R interval would thereby be shortened.7

The A-V node was altered by a continuous, subthreshold, direct electrical current applied to the node. It was observed that the P-R interval became shortened and the QRS complex aberrant and widened. Since the QRS complex was abnormal but the last portion usually occurred at the normal time, it was suggested that only part of the node discharged prematurely and the remainder after a normal delay. As the essential disturbance was premature transmission of part of the impulse through the A-V node, the term accelerated auriculoventricular conduction, or accelerated conduction, was utilized to describe this phenomenon.7

Antagonism of Complete Heart Block and Accelerated Conduction

Complete heart block should be a means of determining the validity of the aberrant bundle theory or the acceleration conduction theory. According to the theory of accelerated auriculoventricular conduction, complete heart block and accelerated conduction are mutually exclusive. According to this theory, the abnormality lies in the specific auriculoventricular pathway. If this pathway is completely destroyed, as in complete heart block, accelerated conduction obviously cannot occur. According to the bundle of Kent theory, complete heart block should not interfere with, but should actually facilitate, the production of accelerated conduction. The explanation is that, since the normal pathway is destroyed, the aberrant pathway should function unopposed.

It has been clearly shown in animals that complete A-V block and accelerated conduction cannot occur at the same time: (1) In a previous study in seven animals, Wolff-Parkinson-White complexes could not be produced by electrical stimulation of the endocardial surface of the right ventricle after the production of complete heart block by destruction of the bundle of His (ventricular type of Wolff-Parkinson-White mechanism).7 (2) In the present study, accelerated conduction complexes could not be reproduced by any of the methods after complete heart block had developed as a result of manipulation of the A-V node. (3) After the animal recovered from complete heart block, accelerated conduction complexes occasionally occurred.

Clinically, also, it appears that complete heart block and a Wolff-Parkinson-White complex are mutually exclusive. Two cases in point have been reported. The first case, reported by Coelho,36 concerns a 62 year old woman with hypertension. The electrocardiogram taken after posterior myocardial infarction revealed 2:1 heart block associated with short P-R intervals and abnormal QRS complexes. Subsequently complete heart block developed, after which accelerated conduction or Wolff-Parkinson-White complexes were no longer recorded. In the second case, reported by Fox and associates,37 Wolff-Parkinson-White syndrome was diagnosed in a 70 year old woman. After the intravenous administration of 300 mg. of Pronestyl, 2:1 A-V block developed, associated with Wolff-Parkinson-White complexes of the conducted impulses. After an additional intravenous dose of 200 mg., complete heart block occurred and the Wolff-Parkinson-White beats disappeared.

In view of the above experimental and clinical evidence, it seems probable that the short P-R interval results from accelerated conduction rather than from an anomalous anatomic auriculoventricular pathway. Further observations of clinical cases manifesting short P-R intervals which become complicated by complete heart block are desirable.

It may be recalled that Kent claimed, "When in the heart of the mammal one severs all the structures which connect the auricles to the ventricles with the exception of a strip of tissue on the right lateral aspect of the organ, spontaneous beats arising in the auricle still pass through the ventricle and evoke ventricular beats."73 Erlanger, whose classic studies38 early in the century established the nature of heart block, strenuously objected40 to Kent's idea of a functional aberrant connection between auricle and ventricle. He clearly showed that when the bundle of His was crushed, though all other connections were untouched, heart block occurred. When the crush was placed elsewhere, heart block never occurred.
In this laboratory, in this and in previous studies over the past four years, A-V nodal function has been completely destroyed in more than 60 animals. We have never failed to produce complete heart block. As Erlanger has commented, if there were anomalous connections, permanent anomalous ventricular activation should have been established immediately after the node was destroyed. It should also be pointed out that extensive post-mortem observations over the past 50 years have shown that chronic A-V block in man occurs only in the presence of a lesion in the region of the A-V node and bundle of His.

Kent’s physiologic observations, as far as we know, have never been confirmed. Recently Frau, Maggi and Agostini repeated Kent’s experiments in the rat. They also severed all connections between the auricle and the ventricle except for a strip of tissue on the right lateral aspect of the heart. Their results were entirely negative, and they concluded that there was no functioning anomalous anatomic pathway.

From extensive physiologic and pathologic data accumulated during the past half-century, as well as some clinical data, it becomes apparent that the theory of anomalous functioning A-V connections has comparatively little evidence to support it. Furthermore, observations on complete heart block clearly indicate that the A-V node and the bundle of His are necessary not only for transmission of the normal impulse from auricles to ventricles, but for the production of the accelerated conduction complexes in animals and probably in man.

Partial Heart Block and Accelerated Conduction

In distinction to complete heart block, it has been shown that partial heart block and accelerated conduction can occur simultaneously. Figures 11 and 12 illustrate concomitant 2:1 heart block and accelerated conduction. Several reports of identical manifestations in clinical cases have been published.

In the present series of experiments we have observed repeatedly, after injecting the A-V node, complete heart block without accelerated conduction complexes. After absorption of injected material, there is a gradual return to lesser degrees of heart block, occasionally associated with accelerated conduction complexes. After absorption of injected material, there is a gradual return to lesser degrees of heart block, occasionally associated with accelerated conduction complexes. A clinical case with similar manifestations has been reported. The patient was a 59 year old man with known hypertension of many years’ duration. Electrocardiograms showed a normal P-R interval. Following a second episode of posterior myocardial infarction, the electrocardiograms showed second degree heart block, which was succeeded by first degree heart block during the convalescent stage. The P-R interval eventually reverted to normal. Several months later, in the absence of any new clinical manifestations, the electrocardiogram exhibited accelerated conduction, with the P-R interval measuring 0.09 second. Autopsy disclosed extensive replacement fibrosis in the region of the A-V node.

The presence of partial heart block indicates disturbance of A-V nodal function. Since partial heart block and accelerated conduction complexes were observed in the same animal, the indication is that a single lesion in the A-V node may result in both types of disturbance. Partial heart block and accelerated conduction may occur in the same patient (cases 4 and 5). If the A-V node is completely destroyed, complete heart block results. If it is partially disturbed, partial heart block results. However, the disturbance may on occasion be such, as in some patients and in these experiments, that the function of normal delay is impaired and accelerated conduction results. Thus, if the node is diseased but not completely destroyed, the impulse may at times be delayed and at other times accelerated. The mechanism by which the A-V node at one time delays and at other times accelerates conduction in the same heart is unknown.

The A-V Node, the “Central Nervous System” of the Ventricle

It has been shown that when a constant subthreshold stimulus was applied to the A-V node, ventricular aberration of a constant type occurred. When other parts of the node were similarly stimulated, entirely different types
of ventricular complexes resulted. This suggested that the node is a sort of "central nervous system" and that, from a physiologic viewpoint, certain parts of the node supply specific parts of the ventricle. Mechanical stimulation of the A-V node has yielded aberrant ventricular complexes similar to both right and left ventricular extrasystoles and bundle-branch block. Further evidence supporting this idea was obtained by Smith and co-workers, who showed that strategically placed cuts in the endocardium could produce segmental block with no change in the rest of the ventricles.

In this study, after chemical modification of the node, it was observed that the QRS complex associated with the short P-R interval was either of normal duration or aberrant and widened. It is suggested that when it is widened, one part of the node is discharged early while the rest of the node is discharged at its normal time. The part of the node which is discharged early determines the form of the initial portion of the QRS complex. The normal QRS, it is postulated, occurs when the entire node discharges synchronously, resulting in normal sequence of activation of the ventricles. Whether the nodal changes produced in our experiments represented specific effects of the drugs injected or nonpharmacologic effects cannot be stated at present. In either event, the results of the experiments are attributed to alteration of nodal function.

Indirect evidence has been obtained that afferent impulses from any part of either ventricle may be conducted through the A-V node to the auricle. The ventricular type of accelerated conduction can be explained by this mechanism. Retrograde conduction may be considered a crude example of this phenomenon. More work on the physiology of the A-V node is obviously necessary.

**Classification of A-V Nodal Abnormalities**

The multiplicity of disturbances produced by experimental procedures directed at the A-V node suggests that this region has a more important role in regulating the heart beat than hitherto supposed. A tentative classification of A-V nodal dysfunctions appears in table 1. Normally the A-V node delivers the auricular impulse to the ventricles after a delay of 0.07 to 0.15 second. An alteration of this function would produce either of two main electrocardiographic abnormalities: (1) various degrees of A-V block, or (2) accelerated conduction with shortening of the P-R interval. It is postulated that simultaneous activation of the entire node yields normal QRS complexes, while premature or delayed activation of parts of the node produces QRS aberrations similar to those produced by

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**Table 1: Disturbances of A-V Node**

<table>
<thead>
<tr>
<th>Disturbance</th>
<th>Description</th>
<th>Diagram</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant heart block</td>
<td>(1) Delay</td>
<td></td>
</tr>
<tr>
<td>1st degree</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2nd degree</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd degree</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inconstant heart block (Wenkebach)</td>
<td>(2) Acceleration</td>
<td></td>
</tr>
<tr>
<td>Normal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nodal rhythm</td>
<td>(1) Nodal rhythm</td>
<td></td>
</tr>
<tr>
<td>Normal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nodal extrasystoles</td>
<td>(2) Ectopic focus</td>
<td></td>
</tr>
<tr>
<td>Normal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nodal tachycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nodal flutter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal QRS</td>
<td></td>
<td></td>
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<tr>
<td>Abnormal QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nodal fibrillation</td>
<td></td>
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</tbody>
</table>

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*Drawings and tables are not included in the text and should be inserted manually or as an image.*
ventricular pacemakers. Occasionally the A-V node conducts in a retrograde fashion. This may be associated with delay of the impulse and with some degree of retrograde block (case 4). The A-V node may also initiate the impulse as in A-V nodal rhythm and nodal arrhythmias. It is postulated that these disturbances may occur without histologically demonstrable lesions of the nodal tissue, as in one of our cases, or may be associated with demonstrable pathology, as in three previously reported cases? It is suggested that the well-known Wolff-Parkinson-White syndrome is an example of accelerated conduction with ventricular aberration. This condition is often associated with nodal beats and nodal arrhythmias. Whether or not organic changes are present in such cases is not yet known.

It seems remarkable that by altering the A-V node—a tiny structure less than 2 mm. in diameter—by chemical, mechanical or electrical stimulation, so many abnormalities may be produced. It is perhaps not quite so remarkable if one compares the heart with the central nervous system, where very small lesions, if strategically located, may cause profound changes. Much larger lesions in so-called silent areas in the brain or in the heart may cause no signs or symptoms.

Summary and Conclusions

1. Seven clinical cases with electrocardiographic patterns characterized by a short P-R interval are reported. The short P-R interval was of constant or of varying duration. The QRS complex was either normal or aberrant. The abnormality can occur in normal subjects with no evidence of organic heart disease or may occur as a result of organic heart disease. Nodal rhythm and partial heart block occurred in some of the cases. These cases are not the classic Wolff-Parkinson-White syndrome.

2. In 40 dogs, the A-V node was injected with cocaine, formaldehyde or acetylstrophanthidin. Eleven of the dogs developed exactly the same variety of P-R and QRS abnormalities seen in the seven clinical cases. Heart block and nodal rhythm occurred in the other animals.

3. In 10 animals, control injections into other parts of the heart did not produce any of the above-mentioned electrocardiographic phenomena.

4. The normal function of the A-V node is to delay passage of the impulse from the auricle to the ventricle. This delay accounts for the major portion of the normal P-R interval. It is thought that the injections interfered with this function, allowing acceleration of conduction from auricle to ventricle with resultant shortening of the P-R interval. The abnormality has been termed accelerated conduction.

5. Since the electrocardiograms in the patients and in the dogs were identical, it is proposed that the abnormality in the patients may also have been due to disturbance of the A-V node.

6. Evidence has been presented that the A-V node is a sort of "central nervous system" of the heart and that, from a physiologic viewpoint, certain parts of the node supply specified parts of the ventricle.

7. In cases with short P-R intervals, it is postulated that when the entire A-V node is discharged prematurely, the QRS is narrow and normal. If only part of the node is discharged prematurely, and the rest discharged later, then the QRS is wide and aberrant.

8. The two major theories of the short P-R interval phenomenon are (1) anomalous anatomic pathways and (2) accelerated conduction through the normal pathway. The phenomenon of complete heart block should be a means of determining which theory is valid. If the short P-R interval is due to anomalous pathways, then, after destruction of the A-V node, the abnormal beats should be accentuated and permanent. If the abnormal beats are due to dysfunction of the normal conducting system, destruction of the normal system should eliminate these beats.

9. It has been demonstrated that complete heart block can always be produced by a small lesion in the A-V node in both man and animals. It seems unlikely that the aberrant anatomic pathways which have been described can have a physiologic function, since they do not function even after the A-V node is destroyed.
10. In all experiments in this series, complete heart block eliminated all the accelerated conduction phenomena in the dogs. Two clinical cases have been reported in which Wolff-Parkinson-White complexes disappeared after production of complete heart block. This is evidence in favor of the accelerated conduction theory and against the anomalous anatomic pathway theory.

11. Complete heart block and accelerated conduction are mutually antagonistic. This is not true of partial heart block, which may coexist with accelerated conduction. This has been found in our experimental animals and has been reported to occur in patients. It is postulated that in such cases the A-V node is diseased, blocking some beats and transmitting others prematurely.

12. As a result of simultaneous recording of direct auricular and limb leads, it was found that the P-R interval in the limb lead may be so short that the P wave is buried in the initial portion of the upstroke of the QRS complex. Such beats may be erroneously diagnosed as idioventricular beats, ventricular extrasystoles or ventricular tachycardia. In fact, such beats may be manifestations of extreme accelerated conduction.

13. A clinical classification of disturbances of the A-V node is presented. It is suggested that the well-known Wolff-Parkinson-White syndrome is due to an abnormality of the A-V node resulting in short P-R intervals, aberrant QRS complexes, and occasionally nodal rhythms and arrhythmias.

14. The A-V node, a very minute structure, thus can have three different types of disturbances: (a) heart block, (b) accelerated conduction and (c) nodal rhythms and arrhythmias. Dysfunction of the A-V node may also completely change the duration and configuration of the QRS complex.

SUMMARIO IN INTERLINGUA

Es reportate 7 casos de configurationes electrocardiographic a breve intervallos P-R de natura o constante o variable. Le complexo QRS esseva o normal o aberrante. Iste anormalitates esseva reproduce experimentalmente in canes per le injection de varie drogas a in le region del nodo atrioventricular. Es discutite le theorias que ha essite formulate pro explicar le presente phenomenos. Le resultatos indica que un acceleration del conduction in le nodo atrioventricular es responsabile pro le breve intervallo P-R. Le forma del complexo ventricular pote depender del activitate synchrono o asynchrono del nodo atrioventricular. Nos presenta un classification de dysfuncionamentos del nodo atrioventricular.

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Studies on the Mechanism of Ventricular Activity: XIV. Clinical and Experimental Studies of Accelerated Auriculoventricular Conduction

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