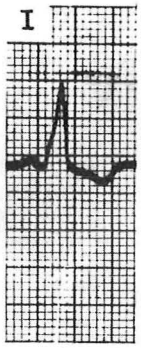
# The American Heart Journal

VOL. V AUGUST, 1930 No. 6

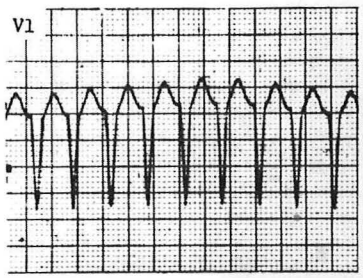

## Original Communications

### BUNDLE-BRANCH BLOCK WITH SHORT P-R INTERVAL IN HEALTHY YOUNG PEOPLE PRONE TO PAROXYSMAL TACHYCARDIA


LOUIS WOLFF, M.D., BOSTON, MASS., JOHN PARKINSON, M.D., LONDON, ENG., AND PAUL D. WHITE, M.D., BOSTON, MASS.


*THE WOLFF PARKINSON WHITE SYNDROME*




PARKLAND MEMORIAL HOSPITAL

Medical Grand Rounds

April 16, 1970

TABLE OF CONTENTS

THE WOLFF PARKINSON WHITE SYNDROME


I INTRODUCTION
II ELECTROCARDIOGRAPHIC CRITERIA
A. Classical WPW
B. Variants of WPW
III SYNONYMS
IV INCIDENCE
V ASSOCIATED CONDITIONS
VI FAMILIAL INCIDENCE
VII PATHOPHYSIOLOGY
VIII ARRHYTHMIAS AND SURGICAL TREATMENT
IX MEDICAL TREATMENT
X PROGNOSIS
XI CASE HISTORIES

## I. INTRODUCTION[1]

"The combination of bundle branch block, abnormally short P-R interval, and paroxysms of tachycardia (also paroxysmal auricular fibrillation and perhaps flutter) in young, healthy patients with normal hearts is distinctive, and worthy of recognition as a mechanism heretofore undescribed as such. The reversion to normal ventricular complexes and longer (normal) P-R interval, spontaneously or by vagal release following exercise or atropinization is characteristic. The paradoxical effect of vagal stimulation on the P-R interval is noteworthy."

On April 2, 1928, a 35-year-old man was referred to Dr. Paul D. White's laboratory because of a history of paroxysmal atrial fibrillation. The patient was a vigorous 35-year-old athletic director whose only complaint was intermittent palpitation of ten years duration. In the times between the paroxysms of atrial fibrillation the electrocardiogram revealed abnormal ventricular complexes, a P-R interval of 0.10 second and normal P waves. A review of the hospital records uncovered three more patients with this electrocardiographic abnormality. Later that year Dr. White took a European trip and took with him these unusual electrocardiograms. (2) Response by several European cardiologists was less than enthusiastic but Dr. John Parkinson was interested in the observation and offered to review his own files. He found seven cases in London which added to four cases from Boston comprised the report which was published in 1930. (1) Eleven patients demonstrating an abnormally short PR interval associated with a prolonged QRS containing a delta wave (an initial slurring with a prolonged QRS complex) who were prone to episodes of paroxysmal tachycardia were described. The syndrome bears the name of the three authors who cited these eleven patients and will I am sure, continue to be called the Wolff Parkinson White syndrome. However, it would be less than charitable to earlier investigators if it were not recognized that six previous publications clearly described this syndrome (3-8) although the authors did not fully grasp the implications of their observations. *(Fig. 1)*

Many questions are raised by this syndrome, such as the nature of the mechanism responsible for the peculiar electrocardiogram, the etiology of the syndrome, its association with congenital heart disease, the mechanism of the paroxysmal tachycardia, the rationale of medical and surgical therapy, and its prognosis. Recently the Wolff Parkinson White Syndrome has been referred to as the Rosetta Stone of electrocardiography (9) since an unravelling of its hieroglyphics could lead to a much fuller understanding of many principles of electrophysiology. Accordingly an archeological expedition is not inappropriate for Grand Rounds this morning.

## II. ELECTROCARDIOGRAPHIC CRITERIA

### A. Classical WPW:

The features of the WPW anomaly are:

(1) Normal P Waves:

(2) P-R interval less than 0.12 seconds in 85 per cent of cases and usually less than 0.10 seconds. (10)

(3) A normal P-J interval (measured from the onset of the P Wave to the end of the QRS)

(4) and a heavily slurred initial deflection of the QRS- the delta wave. (Eiffel Tower QRS).

On the basis of inscription of the delta wave, the WPW pattern has been divided into two types, A and B. (11-15) Type A is the rarer of the two types. In it there are large often slurred R waves in V1-V6 with positive delta waves in the same leads. *(Fig. 2)* Type B is characterized by a large negative QRS deflection in V1-V2 and predominantly positive QRS complexes in the left precordial leads. *(Fig. 3)* In summary then a positive delta wave in V1 and V2 characterizes Type A and a negative delta wave in V1 and V2 characterizes Type B.

Type A, with positive delta waves in V1 and V2, has been said to represent early activation of the left ventricle with the subsequent initial excitation wave moving anteriorly accounting for a positive delta wave in V1 and V2. Similarly Type B has been said to represent early activation on the right ventricle with the subsequent posteriorly directed excitation wave accounting for a negative delta wave in V1 and V2.

### B. Variants of WPW:

*(1) Concertina Effect: The concertina effect consists of a progressive lengthening of the P-R interval and concomitant narrowing of the QRS complex which assumes a more normal contour. (Fig. 4) The delta wave encroaches, sometimes more and sometimes less, on the preceding P-R Segment. This phenomenon has been attributed to variation in the amount of ventricular musculature undergoing premature excitation. (16-17)*

*(2) Lown Ganong Levine Syndrome: (18-21) This variant is characterized by an electrocardiogram displaying a short P-R interval and a normal QRS complex with paroxysms of tachycardia.*

*This syndrome differs from that described by Wolff Parkinson and White because no delta wave is seen and the QRS complexes are normal. Seventy per cent of the patients in most series of WPW syndrome are male. Age at onset of the tachycardia is frequently greater than age 40 (42%). The first heart sound is accentuated in The Lown Ganong Levine Syndrome but decreased in WPW syndrome.*

TABLE I COMPARISON OF LOWN GANONG LEVINE SYNDROME AND WOLFF PARKINSON WHITE SYNDROME

| | LGL | WPW |
|---|---|---|
| Age at onset of tachycardia | | |
| Average | 33.5 | 22.5 |
| Range | 10-61 | 1-54 |
| Sex, Female | 70.9% | 32% |
| M1 Loud | 87% | 16% |

Several authors (22-23) feel this syndrome is not a separate clinical entity. They explain the absence of the delta wave by variation in cardiac position, or union between normal and abnormal conducting pathways. This syndrome will be discussed further under the section on Pathophysiology.

III. SYNONYMS:

Since the description of the WPW Syndrome in 1930 there have been a number of authors who have renamed it. Much of the confusion in terminology reflects the speculation the authors have had concerning the mechanism of the electrocardiographic changes. A listing of some of the synonyms for the syndrome appears in Table II.

TABLE II SYNONYMS FOR WOLFF PARKINSON WHITE SYNDROME

Bundle of Kent Syndrome (Scherf and Schoenbrunner) (24)
Pre-Excitation Syndrome (Oehnell) (25)
Anomalous Atrioventricular Excitation (Rosenbaum) (26)
Aberrant Atrioventricular Conduction (Fox) (27)
By-pass A-V Conduction (Wiggers) (28)
Antesystolie (Holzmann) (29)
Accelerated Atrioventricular Conduction (Prinzmetal) (30)
False Bundle Branch Block (Zao) (31)
Paladino-Kent Bundle Syndrome (108)

In the section on pathophysiology which appears below, some of these theories will also be discussed.

## IV INCIDENCE:

The true incidence of this abnormality is not easy to determine since most surveys have built in biases which invalidate the data. (32-44) The incidence in hospitalized patients reflects not only the incidence in the general population but is heavily weighted as it also includes patients admitted because of associated paroxysmal arrhythmias. In addition since the electrocardiographic abnormalities often mimic a myocardial infarction, some patients with WPW syndrome and chest pain of other etiology will be admitted to the hospital with a diagnosis of acute myocardial infarction.(45-47) In the more common Type B WPW pattern, QS deflections occur commonly in leads II, III, AVF, V1 and V2. With alteration of ventricular depolarization by the pre-excitation wave there is also alteration of repolarization leading to secondary ST segment and T wave abnormalities. These changes may be misinterpreted for the primary changes of injury or ischemia occurring with infarction.

Many surveys have included screening of air force personnel since all of these individuals receive an electrocardiogram upnn entering this branch of the service. The two largest surveys which include a total of 122,043 individuals place the incidence at 1.5 per thousand.(32-33 ) (Other series range from 0.1 to 3.1 per thousand). The ages of the subjects ranged from 16 to 50 years. Even this survey has the built in bias of representing almost entirely a male population. Part of the reason for the apparent increased incidence of WPW syndrome in men is perhaps due to the inherent biases of the selected population groups which include hospitalized patients with suspected myocardial infarction, insurance examination surveys and military population surveys.

## V ASSOCIATED CONDITIONS:

A number of conditions which appear to be associated with the WPW syndrome have been reported and appear on Table III. Most notable among these are myocardial infarction, corrected transposition of the great vessels and Ebstein's anomaly.

TABLE III CONDITIONS IN WHICH THE WPW ANOMALY IS REPORTED

| ACQUIRED | CONGENITAL |
|---|---|
| Acute rheumatic carditis (48-49) | Atrial septal defect (62) |
| Thyrotoxicosis (50-51) | Ventricular septal defect (63) |
| Acromegaly (52) | Tetralogy of Fallot (49) |
| Central nervous dysfunction (53) | Coarctation of the aorta (49) |
| Psychiatric disorders (50,52) | Idiopathic hypertrophic subaortic stenosis (48) |
| Coronary heart disease (52) | Corrected transposition of the great vessels (52) |
| Myocardial infarction (54-60) | Tricuspid atresia (64) |
| Bundle branch block (61) | Endomyocardial fibroelastosis (65-66) |
| | Ebstein's anomaly (64) |
| | Familial cardiomegaly (48) |
| | Primary myocardial disease (48) |

VI FAMILIAL INCIDENCE:

The WPW syndrome has been described as occurring in siblings and in other family members. (67-75) (The occurrence of this syndrome in several members of the same family (74) as well as its appearance in infancy and childhood (76-80) favors the thesis that the process of the WPW syndrome, whether determined anatomically or electrophysiologically, is an inherited one.) Not all investigators agree however that the WPW Syndrome is an inherited one. Warner and McKusick (81) examined 80 members of 14 WPW families without finding any additional cases. Nor could they establish any increase of consanguinous matings among the parents of these WPW cases. All studies which address themselves to surveying family members of index cases however have the inherent weakness that the electrocardiographic manifestations of this syndrome may be transient, appearing and resolving alternately and intermittently.

VII PATHOPHYSIOLOGY:

Many theories have sought to explain the pathogenesis of this syndrome (82-87). The three most common explanations are:

(1) THE ECTOPIC FOCUS THEORY
(2) ACCELERATED CONDUCTION
(3) ACCESSORY CONDUCTION TISSUE

(1) ECTOPIC FOCUS THEORY (88-92)*( Fig 5 )*

The ectopic focus theory postulates the appearance of fusion beats between synchronous pacemakers, one situated at the sino-atrial node having a normal conduction pathway, and the other located at a ventricular or atrial level, both above the bifurcation of the bundle of His. Sodi-Pallares and his associates produced complexes resembling WPW during cardiac catheterization by stimulating the right ventricular septum. They hypothesized that there were hyperexcitable areas on the right side of the interventricular septum that responded to mechanical contraction of the atrium or to weak atrial action potentials.

(2) ACCELERATED CONDUCTION (93-95)*( Fig 6 )*

Prinzmetal and his associates propose only one conduction system, with normal intratrial and interatrial conduction but decreased A-V nodal delay. They postulate several fibers in the region of the A-V node undergoing continuous depolarization, permitting the accelerated conduction from atria to ventricles.

They observed that artificial stimuli would produce QRS complexes resembling WPW complexes once the His bundle had been cut. Langendorf and co-workers (96) pointed out that the artificially induced QRS complexes were only superficially similar to WPW complexes, since there was usually an absence of the delta wave.

(3) ACCESSORY PATHWAY *(Fig. 7)*

The anomalous electrocardiographic complex has been generally regarded as a fusion beat composed of a normally conducted beat and an associated beat through an accessory pathway. (97-99) According to this interpretation, the sinus impulse is thought to traverse both the normal and the anomalous conduction pathways simultaneously on its way to the ventricles. That portion of the sinus impulse which traverses the anomalous route is thought to depolarize a portion of the ventricular myocardium prematurely, producing the delta wave. The remaining portion of the ventricular myocardium is thought to be depolarized by the impulse that traverses the normal atrioventricular conduction pathways, producing the terminal portion of the QRS. Thus the proposed concept of a fusion complex implies that premature excitation of a portion of the ventricles shortens the P-R interval and distorts and lengthens the QRS complex by the same amount that the P-R interval is shortened. The P-S interval remains unchanged.

In 1893 Kent described muscular bridges connecting the right atrium and right ventricle in a variety of species. (100-101) Later studies by Kent (102-107) described a band of muscle fibers at the right lateral atrio-ventricular junction. He reported that when all other atrioventricular connections were divided the muscular bridge between the atrium and ventricle could conduct impulses in both an antegrade and retrograde direction. It is of interest to note that the Italians refer to the Bundle of Kent as the Paladino bundle as these fibers were described by Paladino in 1878.(108)

Any theory which wishes to explain the WPW syndrome must also explain the increased incidence of paroxysmal arrhythmias that are an integral part of the clinical picture. THE ACCESSORY PATHWAY THEORY MOST CLOSELY EXPLAINS ALL THE FEATURES OF THE WPW SYNDROME (109-115) BUT THE EXPLANATION IS MORE COMPLEX THAN MERELY POSTULATING A BUNDLE OF KENT.

First, a number of cases of proved WPW syndrome have been examined postmortem without demonstration of such bundles, (116) and some normal hearts (ones from patients without known WPW syndrome) have lateral myocardial connections directly between atrium and ventricle. Second, of the available illustrations

demonstrating histology of Kent bundles, all appear to contain ordinary myocardial cells and not Purkinje cells. None of the known characteristics of rapidly conducting cells have been shown to be present in Kent bundles. It is possible that Kent bundles conduct rapidly without anatomic evidence of such function but ordinarily, rapidly conducting fibers are not difficult to discern with appropriate stains. Furthermore any investigation of the WPW Syndrome must explain the known variants of this syndrome.

Although the original description of the WPW syndrome described three features:

(1) Short P-R interval
(2) Prolonged QRS Complex
(3) Delta wave

two variants of this syndrome have also been recognized. One of these (Lown Ganong Levine Syndrome) has a

(1) Short P-R interval
(2) Normal QRS complex
(3) No delta wave

and the other has

(1) Normal or prolonged P-R interval
(2) Prolonged QRS
(3) Delta wave

Recent anatomic studies by James (117) and Lev (118) have afforded possible explanations for these three forms of anomalous AV conduction. A brief review of the possible pathways of atrial and AV conduction provides the anatomic background for a unifying concept of anomalous AV conduction in its various forms.

In the past the usual concept of AV conduction has rested upon the premise that after discharge of the sinoatrial node, the wave of excitation moved over the atrial fibers in a uniform radial and diffuse fashion and arrived at the atrioventricular node. The sinus node, located at the junction of the superior vena cava and right atrium was conventionally regarded as an island of specialized tissue in a sea of atrial muscle.

Recent electron microscopic studies have demonstrated that the sinus node is composed of many small and independent cells of different forms and structure. (119-121) Some of these cells are thought to be primary pacemaker cells while others represent intermediary stages between these pacemaker cells and the ordinary cells of the atrial myocardium. Different groups of pacemaker cells may depolarize at different times leading to a differing time course of atrial depolarization. After a pause and delay (122-123) at the superior atrionodal junction, lasting from 0.04 to 0.06

seconds it was generally believed that the electrical impulse depolarized the AV node followed by the bundle of His, the right and left bundle branches and through the Purkinje fibers, the ventricular myofibrils. Recent anatomical work has shown however that human atria contain 3 specialized conduction pathways separate from atrial musculature. These are not only interatrial, joining' left and right atria, but are also internodal tracts affording direct anatomic connection between SA and AV nodes, and possibly allowing for preferential function via these circuits. (124-128)

The tracts are called *The Anterior Internodal Tract, The Middle Internodal Tract and the POSTERIOR Internodal Tract (Fig 8)* This latter tract terminates with most of the fibers BYPASSING the bulk of the A-V node. The possible functional importance of such tracts i.e. their capability as preferential and faster conduction pathways between the sinus node and the A-V node, as well as the left atrium, is a distinct electrophysiologic possibility. Conduction over such paranodal or "bypass" tracts would result in a P-R interval shorter than normal since the normal conduction delay at the superior AV nodal margin would be avoided. The ensuing QRS complex would be normal.*(Fig 9 )* The Lown Ganong Levine Syndrome could be explained by such a mechanism.

In addition to the "bypass" tract of James, there exist a set of short but direct connections between the lower AV node and the ventricular septum or between the upper part of the bundle of His or each bundle branch and the ventricular septum.*(Fig 10)* These fibers are called Mahaim fibers. (129-130)

Finally Lev (125) has confirmed right and left sided bundles of Kent (neuromuscular bridges across the A-V ring outside the conduction systems directly connecting atria and ventricles).*(Fig 11 )* Hence there are at least three possible pathways of atrioventricular nodal conduction. These are (1) Bypass fibers of James
(2) Mahaim fibers
(3) Bundle of Kent
From this information it is possible to reconstruct the anatomic and electrophysiologic background to explain the various forms of anomalous AV conduction.

(1) In the classic form of the WPW syndrome with short PR, long QRS and delta wave, the bypass or short circuiting may occur in one of two ways; excitation may move across a bundle of Kent, thus avoiding the specific conduction tissues completely or almost completely and bringing pre-excitation or early depolarization to the ipsilateral ventricle and somewhat later depolarization to the contralateral ventricle resulting in a short PR and a long QRS; *(Fig 12)* or excitation may travel over two separate bypass tracts namely the AV bypass of James, which would produce the short PR, and then over Mahaim fibers which would produce the delta wave of early ventricular

invasion and the prolonged QRS.*(Fig 13)* Such a case has been recently reported (Lev 116) and a bypass sequence quite similar to this one has been postulated by Scherf and James.

(2) With a short P-R and a normal QRS (Lown Ganong Levine Syndrome) only the James bypass or paranodal fibers would exist or be utilized. *(Fig 14)* With no other anomalous pathway functioning, a normal ventricular event and QRS would follow.

(3) When a delta wave indicating pre-excitation of the ventricles and a prolonged QRS exist with a normal P-R interval (an unusual variant of the WPW syndrome), it is likely that Mahaim fibers alone and not a bundle of Kent, are being used to bypass normal intraventricular conduction.*(Fig 15)* The excitation enters the upper margin of the AV node from the atria, is susceptible to the normal AV nodal delay and hence produces a normal and not a short PR interval, and moves into the common bundle of His. There it short-circuits into the ventricular system over Mahaim fibers, thus bypassing the lower branches of the bundle of His. If the AV nodal function is impaired by drugs of disease, then the PR interval will of course be longer than normal.

## VIII ARRHYTHMIAS

Since these several bypass paths are probably potentially bidirectional, the arrhythmias so frequently seen in patients with pre-excitation states, can be explained as a re-entry phenomenon. That is to say, if excitation comes down over the usual AV connections it may enter the caudal end of a bypass and return rapidly cephalad to invade the atria or nodal area, thus setting off an ectopic rhythm.*(Fig 16)*

Paroxysms of tachycardia are frequent, especially in patients under thirty years of age. Fifty to 80% of the patients with WPW have paroxysmal atrial tachycardia. (131-133) Table IV summarizes the types of arrhythmias which have been associated with syndrome. In these series 70 per cent of patients developed PAT. Atrial fibrillation was noted in 22 per cent of patients.

TABLE IV TYPES OF ARRHYTHMIAS ASSOCIATED WITH WPW SYNDROME

| Author | PAT | Fibrill. | Supravent | Flutter |
|---|---|---|---|---|
| Bishop (134) | 39 | 6 | | |
| Hunter et al (20) | | 2 | 5 | |
| Wolff & White (22) | 8 | 3 | | 2 |
| Littman & Tarnower (135) | 2 | 2 | | |
| Herrmann et al (136) | 22 | 6 | 7 | 2 |
| Chung et al (137) | 24 | 4 | | 1 |

PAT-Paroxysmal atrial tachycardia; Fibrill.-Atrial fibrillation; Supravent. Supraventricular tachycardia (undefined); Flutter-Atrial flutter.

Analysis of the reported data revealed a high incidence of abnormal ventricular responses to supraventricular arrhythmias.

TABLE V - PERCENTAGES OF DIAGNOSED SUPRAVENTRICULAR TACHYARRHYTHMIAS WITH ABERRATION IN THE WPW SYNDROME

| Author | Total No. of Cases Considered | Cases with Aberration | ECG Diagnosis | %with Aberration |
|---|---|---|---|---|
| Herrmann et al (136) | 20 | 12 | -- | 60 |
| Wolff and White (22) | 13 | 2 | -- | 16 |
| Hunter (20) | 7 | 2 | At. Fib* | 28 |
| Littman & Tarnower (135) | 4 | 1 | PAT* | 25 |
| | | | 2 PAT* | |
| Chung et al (137) | 29 | 4 | 2 At. Fib* | 14 |
| Schiebler et al (138) | 8 | 3 | -- | 37.5 |
| Total | 81 | 24 | | 29.6 |

*PAT-Paroxysmal atrial tachycardia; At. Fib.-Atrial fibrillation.

Herrmann et al (136) noted this aberration in 60 per cent of cases. In 6 of 13 recorded cases of atrial tachycardia, in one of 2 cases of atrial flutter and in all 5 cases of atrial fibrillation the QRS complexes were abnormally widened. Wolff and White (22) did not mention which arrhythmias produced abnormal ventricular responses, but in 2 of their 13 cases this finding was displayed, an incidence of 16 per cent. Chung, Walsh and Massie (137) discuss this problem and reproduce only 4 instances of aberrant ventricular conduction as noted in 29 cases, an incidence of 14 per cent.

The differentiation of supraventricular tachycardia with aberrant conduction from ventricular tachycardia is an extremely difficult one. The finding of independent atrial activation or the finding of fusion beats would support the diagnosis of ventricular tachycardia. Without these two findings the diagnosis of ventricular tachycardia is quite difficult. A review of the literature reveals 16 reported instances of "ventricular tachycardia" associated with the Wolff Parkinson White Syndrome. (139-149) Review of the electrocardiographic presentations included in these reports however reveals no unequivocal case of ventricular tachycardia. The question of ventricular tachycardia and ventricular fibrillation will be explored more fully in the section on Prognosis.

Proponents of the "accessory conduction tissue" theory have an explanation for the origin and propagation of tachycardias. A single impulse conducted through the abnormal pathway to the ventricles may return to the atria retrograde over the normal pathway as a re-entry phenomenon (150-157) and then initiate atrial tachycardia. In atrial tachycardia with normal QRS complexes, the stimulus for ventricular depolarization travels over the normal pathway and re-enters the atria

over the anomalous pathway. P wave changes may be seen depending on the site of stimulus re-entry into the atria and its distance from the A-V node.

Bizarre QRS changes may be seen if the antegrade conduction is through the Bundle of Kent and the retrograde conduction from ventricle to atrium is through the atrioventricular node.

The proponents of the "accelerated conduction" theory have shown that continuous, subthreshold, depolarizing current applied to the A-V node not only produces the WPW pattern but also atrial tachycardia, flutter or fibrillation. "The ectopic focus theory" does not offer a suitable explanation for the rapid rhythms. However, one might postulate that a nonsinus pacemaker accelerates and captures conduction, producing atrial tachyarrhythmias.

The earliest studies involving surface mapping of a WPW heart were performed by Durrer and associates. (158-160) In a patient operated upon for closure of an atrial septal defect, very early excitation occurred, 10 msec. after the end of the P wave, at the right lateral border near the atrioventricular sulcus, an area which is located a relatively large distance from the atrioventricular node. Because the epicardial region closest to this node did not show early excitation, it was concluded that in this heart the node was not involved, but that a muscular bypass between the right atrial muscle and the closely adjacent right ventricular surface was responsible. The right ventricle was activated predominantly or completely by an excitation wave originating in this area.

The viewpoint that the WPW Syndrome might be surgically ablated if an aberrant A-V pathway could be identified by electrophysiological means has gained considerable support in its past two years. The first report was that of Burchell and his associates (161) published in 1967. The transient success of this procedure is indicated in the title of the report "Atrioventricular and Ventriculoatrial Excitation in Wolff Parkinson White Syndrome (Type B). Temporary Ablation at Surgery." In a 43-year-old man with WPW syndrome operated upon for repair of an atrial septal defect epicardial exploration revealed an area of premature excitation at the extreme right border of the right ventricle. A solution of 1% procaine injected into the right ventricular muscle, presumably at the site of a Kent bundle, was followed by disappearance of pre-excitation of the right ventricle. Initially a transient atrioventricular dissociation occurred. A transverse cut, 1 cm., in length, was then made on the inside of the right atrium, close to and parallel to the atrioventricular ring. At the end of the intracardiac repair, the cardiac mechanism was by an atrial pacemaker with a normal P-R interval, without evidence of ventricular pre-excitation. However, just after the

final closing of the chest, the electrocardiogram showed a return of the WPW complexes. The postoperative period was without complication. The day after discharge from the hospital the patient reported that he had a short episode of tachycardia.

The authors conclude that "the incision designed to transect the bundle of Kent seemed unduly timid," and promised that in the next case they would attempt ablation of the anomalous pathway of conduction by completely separating the atrium from the ventricle by an incision near the tricuspid ring in the appropriate area.

The next report dealing with the surgical approach to the WPW Syndrome appeared from Duke (162-163). This patient was a 32-year-old man with Type B WPW and recurrent episodes of tachycardia. Premature ventricualr beats and nodal or atrial premature beats initiated episodes of tachycardia. Retrograde P waves frequently followed the premature beats. Sinus beats with anomalous conduction were never seen to initiate episodes of tachycardia.

At the time of surgery in May, 1968 epicardial surface mapping of the right and left ventricles was performed.*(Fig. 17)* The results revealed that the earliest area of ventricular excitation occurred in a localized area, approximately 1.5 cm in width, at the extreme lower border of the right ventricle. A 5 to 6 cm. incision extending from the base of the right atrial appendage to the right border of the right atrium and completely transecting the communication between the atrium and ventricle, was then made. At the time of the incision the electrocardiogram revealed disappearance of the delta wave and appearance of a normal P-R interval and QRS duration. Re-exploration of the heart with a bipolar electrode following surgery revealed that the earliest area of activation was in a normal location on the right ventricular free wall adjacent to the anterior descending coronary artery.*( Fig. 18)* The wave front then spread to encompass both right and left ventricles in a normal manner. The last area of activation recorded was at the right ventricular free wall along the A-V groove. This was the region which prior to surgery had been the earliest excitation. Postoperatively there has been no recurrence of supraventricular tachycardia.

In neither the Mayo Clinic or Duke cases were Kent bundles actually identified, although it must be added that the inferential evidence is strong.

A third patient was described by Dreifus and co-workers.(164) She was a 55-year-old woman whose electrocardiogram showed a classical Type A WPW pattern. Because of refractory tachycardia and congestive heart failure on operation was performed on May 14, 1967. Transient atrioventricular block was achieved after placement of several sutures ligating the common A-V bundle (bundle of His). Postoperatively atrio-ventricular transmission through the anomalous pathway was again

noted and has continued to the present time. No episodes of paroxysmal tachycardia have occurred however, suggesting that a necessary component of the circus re-entrant cycle had been abolished.

The fourth patient in whom an operative procedure was utilized for relief of paroxysmal tachycardias was that of Edmonds et al. (165) On April 30, 1968 a thoracotomy was performed and through an atriotomy electrocoagulation in the area of the A-V node was carried out. Complete atrioventricular block occurred and the patient had a transvenous pacemaker inserted. No further episodes of tachycardia have occurred. Although the production of third degree heart block is a major side effect of this particular operation, the authors felt that the procedure was indicated because of the life threatening nature of the recurrent PAT's. Certainly, however, surgical division of a Kent Bundle is a much more attractive consideration than division of the His Bundle.

Two case reports which have been reported in abstract form however, point out the difficulty in localizing the Bundle of Kent or "Accessory Tissue" (166). Both patients had endocardial and epicardial mapping, which indicated an area of early right ventricular depolarization. Surgical resection of the areas of early depolarization failed in both cases to normalize the electrocardiogram. In one of the patients however, resection of tissue near the A-V node caused reversion of the ECG to normal and microscopic sections of the resected specimen revealed "P cells", suggestive of conduction tissue.

For the present it would seem prudent for surgical treatment of the WPW syndrome to be restricted to those medical centers in which careful epicardial mapping and other essential electrophysiologic studies can be done.

TABLE VI - SURGICAL RESULTS IN WPW SYNDROME

| Author | Patient | Procedure | Result |
|---|---|---|---|
| Burchell (161) | 1) 43 M | Procaine Injection Right Atrial Incision | Return of WPW pattern and arrhythmias |
| Cobb (162-163) | 2) 32 M | 5-6 cm incision along atrioventricular junction made | Loss of delta wave and no arrhythmias |
| Dreifus (164) | 3) 55 F | Ligation of A-V node | Delta wave persists - no arrhythmias |
| Edmonds (165) | 4) 59 M | Electrocoagulation of A-V node | A-V block No arrhythmias |
| Cole (166) | 5) | Resection of area of early depolarization plus area near A-V node | Loss of delta wave - no arrhythmias |
| | 6) | Resection of area of early depolarization near A-V node | Delta wave persists no arrhythmias |

## IX MEDICAL TREATMENT

Therapy for patients with the WPW syndrome reduces itself to therapy for the paroxysmal arrhythmias. No therapy is indicated for solely the electrocardiographic finding of WPW. Since the incidence of supraventricular tachycardia may be as high as 80% however, most patients do require therapy at some time in their course.

### 1. Digitalis

Although digitalis is the drug of choice in the treatment of ordinary supraventricualr tachycardias, many investigators feel that digitalis may aggravate the paroxysmal arrhythmias in patients with WPW. (167-171) It is felt that digitalis may enhance conduction through anomalous paths and decrease normal atrioventricular conduction. Consequently this medication usually fails to slow the rapid ventricular rate in patients with atrial fibrillation and the WPW syndrome and in some patients may even increase the frequency of arrhythmias.

### 2. Quinidine and Procaine amide (172)

Quinidine has a long history of use in patients with paroxysmal tachycardia and WPW. It is possible that quinidine may have several effects upon paroxysmal tachycardias since it is know to

1) decrease the maximal frequency at which atria follow stimuli
2) increase electrical threshold
3) increase fibrillatory threshold
4) decrease conduction velocity

### 3. Lidocaine

One report of the effectiveness of lidocaine to suppress the paroxysmal arrhythmias in a patient with WPW syndrome has appeared in the literature. (173) It is speculated that the effectiveness of lidocaine in the treatment of this patient was attributable to its myocardial depressant properties. Conduction through an accessory pathway may have been interrupted by lidocaine on the ventricular side of the circuit. The reason for this hypothesis is the lack of effectiveness of lidocaine in supraventricular tachycardias of the usual type. In a reciprocating re-entrant tachycardia of the WPW variety however, lidocaine may have a role in therapy.

### 4. Propranolol

Many studies (174-182) have reported the effectiveness of propranolol upon paroxysmal arrhythmias in patients with the WPW Syndrome. In addition to its beta adrenergic blocking effects, propranolol does have a quinidine like action upon the myocardium

and conducting system. It causes a marked decrease in conduction through the atrioventricular node and through this action may inhibit the retrograde circus movement thought to be responsible for paroxysmal arrhythmias of WPW. In addition it may act as a specific ventricular myocardial depressant and thereby inhibit the ventricular side of the re-entry tachycardia.

5. Cardioversion

External countershock has become accepted therapy for a variety of supraventricular and ventricular arrhythmias. It is not surprising therefore that the tachycardia associated with Wolff Parkinson White Syndrome has also been treated by this mode of therapy. The first case report of treatment of this disorder by external countershock was published in 1963 (183) followed by two others in 1966. (184-185)

6. Atrial Stimulation (186-191)

In a variety of supraventricular tachycardias, atrial stimulation through a catheter pacemaker has resulted in the termination of supraventricular and reciprocal tachycardias. The possible mechanisms are:

1) overdrive and suppression of an ectopic supraventricular pacemaker focus.

2) alteration in the supraventricular arrhythmia, that is, conversion of atrial flutter to atrial fibrillation, an unstable rhythm likely to convert to sinus rhythm.

3) the interruption of a fixed circus circuit by atrial paced beats.

It is likely that the success of this mode of therapy in WPW syndrome is through the interruption of a fixed circus composed of the normal A-V conduction pathways and the anomalous bundle.

7. Ventricular Stimulation (192-193)

The interruption of supraventricular tachycardia by premature atrial stimulation and by ventricular depolarization appears to occur by different mechanisms. Premature atrial stimulation as demonstrated by Durrer and associates rendered the atria refractory to retrograde depolarization by the impulse from the accelerated pathway. Premature ventricular stimulation generates early retrograde activation of the atria with identical conduction (R-P) time as the ectopic rhythm. Then when the impulse from this atrial depolarization travels in the antegrade direction through the normal A-V conduction system, the junctional tissue is refractory and the reciprocal rhythm is blocked.

8. Carotid sinus nerve stimulation (194)

A potential surgical approach to the problem of supraventricular tachycardia is implantation of a carotid sinus nerve stimulator. Stimulation of the carotid sinus nerves causes an augmentation of vagal tone and an inhibition of cardiac sympathetic discharge . Vagal stimulation through the carotid sinus pacemaker can be delivered without the concern of traumatizing the carotid artery by manual compression.

## X PROGNOSIS

By far the greatest number of arrhythmias in the Wolff Parkinson White syndrome seem to be supraventricular in origin with paroxysmal atrial tachycardia, atrial flutter and atrial fibrillation being the three most common. Isolated examples of "ventricular tachycardia" have been reported, but almost all of these are subject to an alternative interpretation, the most common one being paroxysmal atrial fibrillation with a rapid ventricular response and aberrant ventricular conduction. Ventricular tachycardia can be diagnosed with certainty only if premature ventricular beats with a configuration resembling the configuration of the ventricular tachycardia are recognized during a predominant sinus rhythm tracing or if there is the presence of fusion beats. It is evident that in the Wolff Parkinson White syndrome these methods of diagnosing ventricular tachycardia are virtually impossible because the ventricular complexes are a result of fusion of a split supraventricular beat so that they are all a type of "fusion beat." To add to the confusion, varying degrees of fusion (195) may occur in this syndrome so that a patient may demonstrate in successive electrocardiograms, or even in the same electrocardiogram, different configurations of the ventricular complex depending on how much of the impulse arrives at the ventricle by way of anomalous pathway and how much travels by way of the normal A-V nodal pathway. Furthermore, the question may validly be asked why ventricular arrhythmias should be an intrinsic part of an anomaly which involves abnormal conduction from the atria to the ventricles. Certainly, it is not difficult to understand an increased incidence of ventricular tachycardia and fibrillation in patients who have an acquired Wolff Parkinson White syndrome due to coronary heart disease or in those having anomalous conduction and associated cardiomyopathy or congenital Ebstein's disease. Ventricular arrhythmias are common in these entities even inthe absence of pre-excitation; however, since the large majority of individuals with Wolff Parkinson White syndrome are otherwise normal and have no evidence of heart disease, the explanation for ventricular arrhythmias is not clear.

There have been only four published reports of ventricular fibrillation associated with the Wolff Parkinson White syndrome. (196) In 1 case, it followed the administration of tetraethylammonium chloride, which may have been toxic to the myocardium. In the case

reported by Kaplan (197) the patient had no evident heart disease of any kind. It must be admitted that this patient could have acquired the Wolff Parkinson White syndrome as a result of her episode of paroxysmal ventricular fibrillation. This seems unlikely since there was no evidence of coronary disease on the coronary cine-angiograms and because of the past history of multiple episodes of paroxysmal arrhythmia. It is also possible that despite the absence of cardiac disease, an episode of paroxysmal ventricular fibrillation developed reflexly, as must occasionally occur in patients without Wolff Parkinson White disease. Again, this explanation would seem statistically improbable.

It is conceivable that some normal hearts cannot tolerate rapid supraventricular arrhythmias of the Wolff Parkinson White type, especially if repetitive or sustained. Such arrhythmias may lead to foci of hypoxia and, hence, to ventricular fibrillation. It is also possible that as yet undefined metabolic factors may be present during such anomalous conduction.

Yahini (198) et al reported on six patients whose arrhythmias simulated ventricular tachycardia but in whom the diagnosis of Wolff Parkinson White syndrome was established after the paroxysm was terminated.

Data from several large surveys of Naval aviators (199) have supported the concept that there is not an increased risk of mortality in subjects with the Wolff Parkinson White syndrome.

Insurance underwriters however are of the opinion that patients with the WPW anomaly and syndrome have an increased mortality rate as compared to the general population. A summary of the policy of 12 insurance companies is summarized below. (34)

1. There are no insurance statistics that reliably document the long term prognosis of the WPW syndrome. The Aetna Life Insurance Company has a group of 49 people with the WPW pattern that has been under observation for a total of 308 patient years. Two deaths have occurred. As yet, the mortality rate is not significantly increased in this group as compared to the population at large.

2. At the present time, the life-table mortality rate is considered to be increased 25 to 30 per cent for people under the age of 35 with the WPW anomaly without paroxysmal tachycardia. For people over 35, the increase in mortality is of the order of 100 per cent. If paroxysmal tachycardia is coexistent, the mortality rate is increased 60 to 300 per cent, depending on the number, duration and character of the attacks of tachycardia.

In spite of the relatively good prognosis suggested by the large surveys cited, several reports have documented death during the paroxysmal tachycardias. (200-201) In a review of literature by Okel, twenty-two reports of fatality during paroxysmal tachycardia were collected.

Based on these reports of fatalities in patients with this syndrome, it appears likely that there is some increased risk of mortality associated with it.

## XI CASE HISTORIES

### E.O. PMH 297882

E.O. a 20-year-old female was first admitted to PMH on 9/26/65 because of palpitations and chest pain. She had been in good health until 12 hours prior to admission when her symptoms suddenly began. She had one previous episode in 1963 which spontaneously cleared. The electrocardiogram revealed a supraventricular tachycardia with wide QRS complexes at a rate of 200. She converted to a normal sinus rhythm after 200 mg. procaine amide intravenously. Her post conversion ECG was classical for WPW syndrome.

She was next admitted to PMH on 11/12/68 because of palpitations, chest pain and dyspnea, all increasing over a five day period. Her physical examination and chest roentgenogram revealed profound pulmonary edema. Cardioversion was immediately carried out with reversion to sinus tachycardia. She improved over the next six days and was discharged on propranolol 25 mg. tid. She was seen in Medicine Clinic for one follow up visit and was asymptomatic.

Two representative electrocardiograms appear in *Fig. 19 and 20.*

### I.P. PMH 368285

I. P. a 31-year-old male was admitted to the hospital on 10/18/69 after arriving in the Emergency Room apneic and pulseless. An electrocardiogram revealed ventricular fibrillation. He was given DC electroshock x15 and finally settled into a sinus tachycardia. He was admitted to the MICU comatose.

He was previously in good health without any known cardiac disease. On the afternoon of admission he drank a large quantity of brandy. After dinner he was on his way to a dance when he suddenly lost consciousness. He was brought directly to the emergency room. His physical examination was within normal limits but for his comatose state. His electrocardiogram showed the classical findings of type B Wolff Parkinson White Syndrome. He never regained consciousness and died three weeks later of Pseudomonas septicemia.

A representative electrocardiogram appears in *Fig. 21.*

# BIBLIOGRAPHY


1. Wolff, L., Parkinson, J. and White, P.D.: Bundle branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 5:685, 1930.
2. Wolff, L.: Wolff Parkinson White syndrome: Historical and clinical features. Prog Cardiovasc Dis 2:677, 1960.
3. Wilson, F.N.: A case in which the vagus influenced the form of ventricular complex of the electrocardiogram. Arch Int Med 16:1008, 1915.
4. Gilchrist, A.R.: Paroxysmal ventricular tachycardia. A report of five cases. Am Heart J 1:547, 1926.
5. Wedd, A.M.: Paroxysmal tachycardia with reference to normotropic tachycardia and the role of extrinsic cardiac nerves. Arch Int Med 27:571, 1921.
6. Bain, C.W.C. and Hamilton, C.K.: Electrocardiographic changes in rheumatic carditis. Lancet 1:807, 1926.
7. Bach, F.: Paroxysmal tachycardia of forty-eight years duration and right bundle branch block. Proc Roy Soc Med London, Clin Sect 22:415, 1929.
8. Hamburger, W.W.: Bundle branch block. Four cases of intraventricular block showing some interesting and unusual features. Med Clin North Amer 13:343, 1929.
9. James, Thomas N.: The Wolff Parkinson White syndrome. Annals Int Med 71:399, 1969.
10. Wolff, L.: Syndrome of short P-R interval with abnormal QRS complexes and paroxysmal tachycardia (Wolff Parkinson White syndrome) Circulation 10:282, 1954.
11. Rosenbaum, F.F., Hecht, H.H., Wilson, F.N. and Johnston, F.D.: The potential variations of the thorax and the esophagus in anomalous atrioventricular excitation (Wolff Parkinson White syndrome). Amer Heart J 29:281, 1945.
12. Burch, G.C., and Kimball, J.L.: Notes on the similarity of QRS complex configurations in the Wolff Parkinson White syndrome. Amer Heart J 32:560, 1946.
13. Bandiera, G. and Antongnetti, P.F.: Ventricular precontracting area in the Wolff Parkinson White syndrome. Demonstration in man. Circulation 17:225, 1958.
14. Gamboa, R., Penaloza, D., Sime, F. and Banchero, N.: The role of the right and left ventricles in the ventricular pre-excitation (WPW) syndrome. An experimental study in man. Am J Cardiol 10:650, 1962.

15. Holzmann, M.: Das Syndrome von Wolff, Parkinson and White. Ztschr. Kreislfschg 51:275, 1962.

16. Ohnell, R.F.: Preexcitation: A cardiac abnormality. Acta Med Scandinav Suppl 152:117, 1944.

17. Kossman, C.E. and Goldberg, H.H.: Sequence of ventricular stimulation and contraction in a case of anomalous atrioventricular excitation. Am Heart J 33:308, 1947.

18. Lown, B., Ganong, W.F. and Levine, S.A.: The syndrome of short P-R interval, normal QRS complexes and paroxysmal rapid heart action: Circulation 5:693, 1952.

19. Littmann, D.: Aberrant atrioventricualr conduction in a patient with paroxysmal tachycardia, a short P-R interval and a normal QRS complex. Amer J Med 2:126, 1947.

20. Hunter, A., Papp, C. and Parkinson, J.: The syndrome of short P-R interval, apparent bundle branch block and associated paroxysmal tachycardia. Brit Heart J 2:107, 1940.

21. Burch, G.E. and Kimball, J.L.: Notes on the similarity of QRS complex configuration in Wolff Parkinson White syndrome. Am Heart J 32:560, 1946.

22. Wolff, L. and White, P.D.: Syndrome of short P=R interval with abnormal QRS complexes and paroxysmal tachycardia. Arch Int Med 82:446, 1948.

23. Pick, A. and Katz, L.N.: Disturbances of impulse formation and conduction in the pre-excitation (WPW) syndrome - on its mechanism. Am J Med 19:759, 1955.

24. Scherf D. andSchoenbrunner, E.: Beitraege zum problem der verkuerzten vorhofkammerleitung. Ztschr Klin Med 128:750, 1935.

25. Oehnell, R.F.: Pre-excitation, a cardiac abnormality, Acta Med Scandin Supp 152, 1944.

26. Rosenbaum, F.F.: The nature of paroxysmal tachycardia in anomalous atrioventricular excitation. Am Heart J 37:668, 1949.

27. Fox, T.T.: Aberrant atrioventricular conduction in a case showing a short P-R interval and an abnormal but not prolonged QRS complex. Am. J. Med. Sci. 209:199, 1945.

28. Wiggers, C.J.: Physiology in Health and Disease, 5th ed. Philadelphia, Lea and Febiger.

29. Holzmann, M.: Das syndrom von Wolff, Parkinson and White Zcschr Kreislfschg. 51:275, 1962.

30. Prinzmetal, M. and Kennamer, R.: Anomalous atrioventricular excitation. Ann New York Acad Science 65:852, 1957.

31. Zao, Z.Z., Herrmann, G.R. and Hejtmancik, M.R.: A study of the delta wave in"non-delayed" conduction. Am Heart J 56:920, 1958.

32. Averill, K.H. and Lamb, L.E.: Electrocardiographic findings in 67,375 asymptomatic subjects. I Incidence of abnormalities. Am J Cardiol 6:76, 1960.

33. Hiss, R.G. and Lamb, L.E.: Electrocardiographic findings in 122,043 individuals. Circulation 25:947, 1962.

34. Smith, R.F.: The Wolff Parkinson White syndrome as an aviation risk. Circulation 29:672, 1964.

35. Sears, G.A. and Manning, G.W.: The Wolff Parkinson White pattern in routine electrocardiography. Canad Med Assoc J 87:1213, 1964.

36. Hejtmancik, M.R. and Herrmann, G.R.: The electrocardiographic syndrome of short P-R interval and broad QRS complexes. Amer Heart J 54:708, 1957.

37. Graybiel, A., McFarland, R.A., Gates, R.C. and Webster, F.A.: Analysis of the electrocardiogram obtained from 1000 healthy aviators. Amer Heart J 27:524, 1944.

38. Averill, K.II., Rosmoe, R.J. and Lamb, L.E.: Electrocardiographic findings in 67,375 asymptomatic subjects. IV. Wolff Parkinson White syndrome. Amer J Cardiol 6:108, 1960.

39. Littmann, D. and Tarnower, H.: Wolff Parkinson White syndrome. Amer Heart J 32:100, 1946.

40. Graybiel, A., McFarland, R.A., Gates, D.C. and Webster, F.A.: Analysis of the electrocardiograms obtained from 1000 young healthy avaitors. Am Heart J 27:524, 1944.

41. Manning, G.W.: Electrocardiography in the selection of Royal Canadian Air Force aircrew. Circulation 10:401, 1954.

42. Manning, G.W.: An electrocardiographic study of 17,000 fit young Royal Canadian Air Force aircrew applicants. Am J Cardiol 6:70, 1960.

43. Mortensen, V., Nielsen, A.L. and Eskildsen, P.: Wolff Parkinson White syndrome. Acta Med Scandin 118:506, 1944.

44. Heinecker R.: The significance of the Wolff Parkinson White syndrome. Deutsch Med Wschr 93:357, 1968.

45. Wolff, L. and Richman, J.L.: Diagnoses of myocardial infarction in patients with anomalous atrioventricular excitation. (Wolff Parkinson White syndrome) Am Heart J 45:545, 1953.

46. Grayzel, J.: Electrocardiographic criteria in the differential diagnosis of pre-excitation (Wolff Parkinson White syndrome) and arteriosclerotic heart disease. New Eng J Med 259:369, 1958.

47. Wolff, L.: Diagnostic clues in the Wolff Parkinson White syndrome. New Engl J Med 261:637, 1959.

48. Swiderski, J., Lees, J. II. and Nadas, A.S.: The Wolff Parkinson White syndrome in infancy and childhood. Brit Heart J 24:561, 1962.

49. Schiebler, G.L., Adams, P. and Anderson, R.C.: The Wolff Parkinson White syndrome in infants and children. Pediatrics 24:585, 1959.

50. Chung, K.Y., Walsh, T.J. and Massie, E.: Wolff Parkinson White syndrome. Amer Heart J 69:116, 1965.

51. Sanghvi, L.M. and Banerjie, K.B.: Wolff Parkinson White syndrome associated with thyrotoxicosis. Amer J Cardiol 8:431, 1961.

52. Brilmayer, C.: The diagnosis is - laterally spreading recent infarct of the anterior wall. Med Klin 62:770A, 1967.

53. Manck, H.P., Rinko, W. and Hoff, E.C.: Neural pathways involved in the production of Wolff Parkinson White electrocardiographic complexes by mesencephalie stimulation. Circulation 28:756, 1963.

54. Wolff, L.: Anomalous atrioventricular excitation (Wolff Parkinson White syndrome). Circulation 9:14, 1959.

55. Spritz, N., Cohen, B.D., Frimpter, G.W. and Rubin, A.C.: Electrocardiographic interrelation of the pre-excitation (Wolff Parkinson White) syndrome and myocardial infarction. Amer Heart J 56:715, 1958.

56. Rinzler, S.H. and Travell, J.: The electrocardiographic diagnosis of acute myocardial infarction inthe presence of Wolff Parkinson White syndrome. Amer J Med 3:106, 1957.

57. Stein, I. and Wroblewski, E.L.P.: Myocardial infarction in Wolff Parkinson White syndrome. Amer Heart J 42:624, 1957.

58. Wolff, L. and Richman, J.C.: The diagnosis of myocardial infarction in patients with anomalous atrioventricular excitation (Wolff Parkinson White syndrome). Amer Heart J 45:545, 1953.

59. Goldberg, H.H. and Lewis, S.M.: Acute myocardial infarction and Wolff Parkinson White syndrome. Amer Heart J 40:614, 1950.

60. Kistin, A.D. and Robb, G.P.: Modification of the electrocardiogram of myocardial infarction by anomalous atrioventricular excitation. Amer Heart J 37:249, 1959.

61. Castellanos, A., Mayer, J.W. and Lemberg, L.: The electrocardiogram and vectorcardiogram in Wolff Parkinson White syndrome associated with bundle branch block. Amer J. Cardiol 10:657, 1962.

62. Vacheron, P.: Sur la frequence du Wolff Parkinson White chez les nourrissons, a propos d'un cas clinique personell. Arch Mal Coeur 47:345, 1954.

63. Bernreiter, M.L.: The association of Wolff Parkinson White syndrome and paroxysmal tachycardia in two cases of ventricular septal defect. Missouri Med 58:566, 1961.

64. Schiebler, G.L., Adams, P., Anderson, R.C., Amplatz, K. and Lester, R.G.: Clinical study of twenty-three cases of Ebstein's anomaly of the tricuspid valve. Circulation 19:165, 1959.

65. Adams, P. and Anderson, R.C., Schiebler, G.L.: Familial cardiomegaly in association with the Wolff Parkinson White syndrome. Amer Heart J 58:113, 1959.

66. Westlake, R.E., Cohen, W. and Willis, W.H.: Wolff Parkinson White syndrome and familial cardiomegaly. Amer Heart J 64:314, 1962.

67. Ohnell, R.F.: Pre-excitation: a cardiac abnormality. Acta Med Scandinav 117 (suppl 152): 1, 1944.

68. Doumer, E. and Dumez, L.: Syndrome de Wolff Parkinson White familial. Arch mal coeur 44:1134, 1951.

69. Willis, W.H. and Shepard, C.C.: The familial incidence of certain unusual diseases. North New York M.J. 10:19, 1953.

70. Kupatz, H. Aur Klinik des WPW-syndrome im Kindersalter. Neue Osterreichische Ztschr Kinderh 1:59, 1955.

71. McIntire, M.S. and Freed, A.F.: The Wolff Parkinson White syndrome. Report of a case occurring in a mother and infant. Am J Dis Child 89:743, 1955.

72. Averill, J.H.: Wolff Parkinson White syndrome occurring in brothers. Am Heart J 51:943, 1956.

73. Sokmen, C.: A familial case of WPW syndrome. Am Heart J 53:940, 1957.

74. Harnischfeger, W.H.: Hereditary occurrence of the pre-excitation (WPW) syndrome with re-entry mechanism and concealed conduction. Circulation 19:28, 1959.

75. Hecht, H.H., Kennamer, R., Prinzmetal, M., Rosenbaum, F.F., Sodi-Pallares, D., Wolff, L., Brooks, C., Pick, A., Rijlant, P. and Robb, J.S.: Anomalous atrioventricular excitation: panel discussion. Ann New York Acad Sc 65:826, 1957.

76. Hastreiter, A.R. and Miller, R.A.: Management of primary endomyocardial disease. The myocarditis-endocardial fibroelastosis syndrome. Pediat Clin N Amer 11:401, 1964.

77. Paul, O. and Harrison, C.J.: Wolff Parkinson White syndrome in an infant. J A M A 149:363, 1952.

78. Gleckler, W.J. and Lay, J.V.M.: Wolff Parkinson White syndrome and paroxysmal tachycardia in infancy. J A M A 150:683, 1952.

79. Engle, M.A.: Wolff Parkinson White syndrome in infants and children. Am J Dis Child 84:692, 1952.

80. Walsh, S.Z.: Wolff Parkinson White syndrome in a healthy two hour old infant without paroxysmal tachycardia. J A M A 186:14, 1963.

81. Warner, A.O. and McKusick, V.A.: Wolff Parkinson White syndrome: a genetic study. Circulation Res 6:18, 1958.

82. Gamboa, R., Penaloza, D., Sime, F. and Bonchero, N.: The role of the right and left ventricles in the ventricular pre-excitation (Wolff Parkinson White) syndrome: an experimental study in man. Amer J Cardiol 10:650, 1962.

83. Grishman, A., Kroop, J.H. and Steinberg, M.I.: The course of the excitation wave in patients with electrocardiograms showing short P-R interval and wide QRS complexes. (Wolff Parkinson White syndrome). Amer Heart J 40:554, 1950.

84. Grant, R.P., Tomlinson, F.B. and VanBuren, J.K.: Ventricular activation in the pre-excitation syndrome (Wolff Parkinson White). Circulation 19:355, 1958.

85. Bleifer, S., Kahn, M., Grishman, A. and Donoso, E.: Wolff Parkinson White syndrome: a vectorcardiographic, electrocardiographic and clinical study. Amer J Cardiol 4:321, 1959.

86. Tranchesi, J., Gruimaraes, A.C., Texeira, V. and Pileggi, F.: Vectorial interpretation of the ventricular complex in Wolff Parkinson White syndrome. Amer J Cardiol 4:334, 1959.

87. Kisch, B.: Explanation of the Wolff Parkinson White syndrome. Amer Heart J 40:466, 1950.

88. Gottsegen, G. and Bodrogi, G.: On the mechanism of the WPW syndrome. Acta Cardiol 16:529, 1961.

89. Anselmi, A., Anselmi, G., Machado, I. and Anselmi, G.: Estudio experimental del sindrome de Wolff Parkinson White y de las latidos de fusion. Su aplicacion clinca Arch Inst Card

90. Butterworth, J.S. and Poindexter, C.A.: Short P-R interval associated with prolonged QRS complex: a clinical and experimental study. Arch Int Med 69:437, 1942.

91. Fox, T.T.: Morphology of the delta wave in the Wolff Parkinson White syndrome. Circulation 20:696, 1959.

92. Fox, S.T.: On the morphology of the delta wave in the Wolff Parkinson White syndrome. Cardiologia 42:377, 1963.

93. Prinzmetal, M., Kennamer, R., Corday, E., Osborne, J.A., Fields, J. and Smith, L.A.: Accelerated conduction In: The Wolff Parkinson White syndrome and related conditions. New York, Grune and Stratton, 1952.

94. Pallares, D., Wolff, L., Brooks, C., Pick, A., Rijlant, P. and Robb, J.C.: Anomalous atrioventricular excitation: panel discussion. Ann New York Acad Sci 65:826, 1957.

95. Osborne, J.A., Corday, E., Fields, J., Kennamer R., Smith, L.A. and Prinzmetal, M.: Studies on the mechanism of ventricular activity. I. The nature of the P-R interval. Amer. Heart J. 42:503, 1951.

96. Langendorf, R., Lev, M. and Pick, R.: Auricular fibrillation with anomalous A-V excitation (WPW syndrome) imitating ventricular paroxysmal tachycardia: Case report with clinical and autopsy findings and a critical review of the literature. Acta Cardiol 7:241, 1952.

97. Butterworth, J.S. and Poindexter, C.A.: Fusion beats and their relation to the syndrome of short P-R interval associated with a prolonged QRS complex. Am Heart J 28:149, 1944.

98. Linenthal, A.J. and Zoll, P.M.: Ventricular fusion beats during electric stimulation in man. Application to conduction velocity and anomalous A-V excitation. Circulation 31:651, 1965.

99. Anomalous atrioventricular excitation: Panel discussion. In: The electrophysiology of the heart. Edited by Hecht, H.: Ann New York Acad Sc 65:826, 1957.

100. Kent, A.F.S.: Proceedings of Physiol Soc Nov. 12, 1892. J Physiol 14:23, 1893.

101. Kent, A.F.S.: Researches on structure and function of mamalian heart. J Physiol 14:233, 1893.

102. Kent, A.F.S.: Observations on auriculo-ventricular junction of mammalian heart. Quart J Exper Physiol 7:193, 1913-14.

103. Kent, A.F.S.: Structure of cardiac tissue at auriculo-ventricular junction. J Physiol 47:17, 1913-14.

104. Kent, A.F.S.: Right lateral auriculo-ventricular junction of heart. J Physiol 48:xxii, 1914.

105. Kent, A.F.S.: Conducting path between right auricle and external wall of right ventricle in heart of a mammal. J Physiol 48:lvii, 1914.

106. Kent, A.F.S.: Illustrations of right lateral auriculo-ventricular junction in the heart. J Physiol 48:lxiii, 1914.

107. Kent, A.F.S.: Neuromuscular structures in heart. Proc Roy Soc 87:198, 1913-14.

108. Paladino, G.: Contributo all'anatomica, istologia e fisiologia del cuore. Movim. Medico-Chir, Napoli 8:428, 1878.

109. Kimball, J.L. and Burch, G.E.: The prognosis of the Wolff Parkinson White syndrome. Ann Int Med 27:239, 1947.

110. Lev, M., Kennamer, R., Prinzmetal, M. and Demesquita, Q.H.: A histopathologic study of the atrioventricular communication in two hearts with the Wolff Parkinson White syndrome. Circulation 24:41, 1961.

111. Sodi-Pallares, D. and Friedland, C.: A histopathologic study of the atrioventricular communication in a case of WPW with incomplete left bundle branch block. Am Heart J 66:399, 1963.

112. Wood, F.C., Wolferth, C.C. and Geckeler, G.D.: Histologic demonstration of accessory muscular connection between auricle and ventricle in a case of short P-R interval and prolonged QRS complex. Amer Heart J 25:454, 1943.

113. Goldman, I.R., Cosby, R.S. and Griffith, G.C.: The effect of atriopine in the cardiac mechanism in anomalous atrioventricular conduction. Amer Heart J 40:903, 1950.

114. Hoffman, I., Abernathy, R.S. and Haedicke, T.A.: Effect of procaine amide on anomalous conduction and paroxysmal tachycardia in a case resembling the Wolff Parkinson White syndrome. Amer Heart J 44:154, 1952.

115. Wolferth, C.C. and Wood, F.C.: The mechanism of production of the short P-R interval and prolonged QRS complexes in patients with presumably undamaged hearts: hypothesis of an accessory pathway of auriculoventricular conduction (bundle of Kent). Amer Heart J 8:297, 1933.

116. Lev, M., Leffler, W.B., Langendorf, R. and Pick, A.: Anatomic findings in a case of ventricular pre-excitation (WPW) terminating in complete atrioventricular block. Circulation 34:718, 1966.

117. Lev, M.: "The pre-excitation syndrome: Anatomic considerations of anomalous A-V pathways", in Dreifus, L.S. and Koff, W.S. (eds.): Mechanisms and therapy of cardiac arrhythmias, New York: Grune and Stratton, Inc., 1966, pp 665-670.

118. James, T.N.: "Identification of the supraventricular arrhythmias: The specialized conducting itssue of the atria," in Dreifus, L.S., and Koff, W.C. (eds.): Mechanisms and therapy of cardiac arrhythmias, New York: Grune and Stratton, 1966, pp 97-106.

119. Kawamura, K.: Electron microscope studies on the cardiac conduction system of the dog. II. The sinoatrial and atrioventricular nodes, Jap Circ J 25:973, 1961.

120. Trautwein, W. and Uchizono, K.: Electron microscopic and electrophysiologic study of the pacemaker in the sinoatrial node of the rabbit heart, Z. Zellforsch 61:96, 1963.

121. James, T.N., Sherf, L., Fine, G. and Morales, A.R.: Comparative ultrastructure of the sinus node in man and dog. Circulation 34:139, 1966.

122. Hoffman, B.F. and Cranefield P.: The physiologic basis of cardiac arrhythmias. Amer J Med 37:670, 1964.

123. Robb, J.S.: The conduction tissue and cardiac electrophysiology. Ann N Y Acad Sci 65:818, 1957.

124. James, T.N.: The connecting pathways between the sinus node and AV node and between the right and left atrium in the human heart. Amer Heart J 66:498, 1963.

125. Lev, M.: Anatomic considerations of anomalous A-V pathways, in mechanism and therapy of cardiac arrhythmias. Fourteenth Hahnemann Symposium, 1965. Eds.: L.S. Dreifus and W. Likoff. Grune and Stratton, New York, 1966 p 665.

126. Merideth, J and Titus, J.L.: The anatomic atrial connections between the sinus and A-V node. Circulation 37:566, 1968.

127. Truex, R.C.: Anatomical considerations of the human atrioventricular junction in mechanism and therapy of cardiac arrhythmias. Fourteenth Hanemann Symposium, 1965. Eds.: L.S. Dreifus and W. Likoff. Grune and Stratton, New York, 1966 p 333.

128. Sherf, L. and James, T.N. A new electrocardiographic concept: Synchronized sinoventricular conduction. Diseases of the Chest 55:127, 1969.

129. Mahaim, I. and Benatt, A.: Nouvelles recherches sur les connexions superieures de la branche gauche due Faisceau de His-Tawara avec la Cloison Interventriculaire. Cardiologia 1:61, 1937.

130. Mahaim, I. and Clerc, A.: Nouvelle forme anatomique de bloc de coeur, a substituer au bloc dit d'arborisations (bloc bilateral manque). C R Soc Biol (Paris) 109:183, 1932.

131. Rosenbaum, F.F.: The nature of paroxysmal tachycardia in anomalous atrioventricular excitation. Am Heart J 37:668, 1949 (Abstr 3d Inter-amer Cardiol Congr)

132. Hejtmancik, M.R. and Hermann, G.R.: The electrocardiographic syndrome of short P-R interval and broad QRS complex. Am Heart J 54:708, 1957.

133. Willius, F.A. and Carryer, H.M.: Electrocardiograms displaying short P-R intervals with prolonged QRS complexes: An analysis of sixty-five cases. Proc Staff Meet Mayo Clin 21:438, 1946.

134. Bishop, L.F.: Bundle branch block with short P-R interval in individuals without organic heart disease. Amer J Med Sci 194:794, 1937.

135. Littmann, D. and Tarnower, H.: Wolff Parkinson White syndrome. Amer Heart J 32:100, 1946.

136. Herrmann, G.R., Oates, J.R., Runge, T.M. and Hejtmancik, M.R.: Paroxysmal pseudoventricular tachycardia and pseudoventricular fibrillation in patients with accelerated A-V conduction. Amer Heart J 53:254, 1957.

137. Chung, K.Y., Walsh, T.J. and Massie, E.: Wolff Parkinson White syndrome. Amer Heart J 69:115, 1965.

138. Schiebler, G.L., Adams, P. and Anderson, R.C.: The Wolff Parkinson White syndrome in infants and children. Pediatrics 24:585, 1959.

139. Dunn, J.J., Sarrell, W. and Franklin, R.B.: The Wolff Parkinson White syndrome associated with paroxysmal ventricular tachycardia. Amer Heart J 47:462, 1954.

140. Fleishman, S.J.: A case of Wolff Parkinson White syndrome with paroxysmal ventricular tachycardia. Amer Heart J 44:897, 1952.

141. Fox, T.T., Weaver, J. and March, H.W.: On the mechanism of the arrhythmia in aberrant atrioventricular conduction (Wolff Parkinson White). Amer Heart J 43:507, 1952.

142. Cain, E.F.: Wolff Parkinson White syndrome presenting certain unusual features. Amer Heart J 33:523, 1947.

143. Levine, S.A. and Beeson, P.B.: The Wolff Parkinson White syndrome with paroxysms of ventricular tachycardia. Amer Heart J 22:401, 1941.

144. Gilchrist, A.R.: Paroxysmal ventricular tachycardia: a report of five cases. Amer Heart J 1:547, 1926.

145. Cooke, W.T. and White, P.D.: Paroxysmal ventricular tachycardia. Brit Heart J 5:33, 1943.

146. Missal, M.E., Wood, D.J. and Leo, S.D.: Paroxysmal ventricular tachycardia associated with short P-R intervals and prolonged QRS complexes. Ann Int Med 24:911, 1946.

147. Klainer, M.J. and Joffe, H.H.: A case of short P-R interval and prolonged QRS complexes with a paroxysm of ventricular tachycardia. Ann Int Med 24:920, 1946.

148. Palatucci, O.A. and Knighton, J.E.: Short P-R interval associated with prolongation of QRS complex: a clinical study demonstrating interesting variations. Ann Int Med 21:58, 1944.

149. Holzmann, M.: Klinische Elektrokardiographic. Stuttgart, George Thieme Verlag, 1956.

150. Harris, W.E., Semler, H.J. and Griswold, H.E.: Reversed reciprocating paroxysmal tachycardia controlled by guanethidine in a case of Wolff Parkinson White syndrome. Amer Heart J 67:812, 1964.

151. Scherf, D., Blumenfeld, S. and Mueller, P.: A-V conduction disturbance in presence of the pre-excitation syndrome. Amer Heart J 43:829, 1952.

152. De Boer, S.: Die physiologische Grundlage und Klinik des unregelmassigen Herzschlages. Ergebin Inn Med Klinderheilk 29:391, 514, 1926.

153. Rosenbaum, F.F., Hecht, H.H., Wilson, F.N. and Johnston, F.D.: Potential variations of the thorax and the esophagus in anomalous atrioventricular excitation (Wolff Parkinson White syndrome). Amer Heart J 29:281, 1945.

154. Wolff, L.: Anomalous atrioventricular excitation (Wolff Parkinson White syndrome). Circulation 19:14, 1959.

155. Harnischfeger, W.W.: Hereditary occurrence of pre-excitation (Wolff Parkinson White) syndrome with re-entry mechanism and concealed conduction. Circulation 19:28, 1959.

156. Pick, A. and Katz, L.N.: Disturbances of impulse formation and conduction in pre-excitation (WPW) syndrome: their bearing on its impulse mechanism. Amer J Med 19:759, 1955.

157. Moe, G.K. and Mendez, C.: Physiologic basis of reciprocal rhythm. Prog Cardiov Dis 8:461, 1966.

158. Durrer, D., Roos, J.P.: Epicardial excitation of the ventricles in a patient with Wolff Parkinson White syndrome (type B). Circulation 35:15, 1967.

159. Durrer, D., Schoo, L., Schuilenburg, R.M. and Wellens, H.J.J.: Role of premature beats in initiation and termination of supraventricular tachycardia in Wolff Parkinson White syndrome. Circulation 36:644, 1967.

160. Durrer, D.: Electrical aspects of human cardiac activity: a clinical-physiological approach to excitation and stimulation. Cardiovasc Res 2:1, 1968.

161. Burchell, H.B., Frye, R.L., Anderson, W.M. and McGoon, D.C.: Atrioventricular and ventriculoatrial excitation in Wolff Parkinson White syndrome (type B). Temporary ablation at surgery. Circulation 36:633, 1967.

162. Cobb, F.R., Blumenschein, S.D., Sealy, W.C., Boineau, J.P., Wagner, G.S., and Wallace, A.G.: Successful surgical interruption of the bundle of Kent in a patient with Wolff Parkinson White syndrome. Circulation 38:1018, 1968.

163. Sealy, W.C., Hattler, B.G. Jr., Blumenschein, S.D. and Cobb, F.R. Surgical treatment of Wolff Parkinson White (WPW) syndrome. Ann Thorac Surgery 8:1, 1969.

164. Dreifus, L.S., Nichols,H., Morse, D., Watanabe, Y. and Truex, R.: Control of recurrent tachycardia of Wolff Parkinson White syndrome by surgical ligature of the A-V bundle. Circ 38:1030, 1968.

165. Edmonds, J.H., Jr., Ellison, R.G. and Crews, T.L.: Surgical induced A-V block as treatment for recurrent atrial tachycardia in Wolff Parkinson White syndrome. Ibid Circ 39:105, 1965.

166. Cole, J.S., Wills, R.E., Winterscheid, L.C., Reichenbach, D.D. and Blackmon, J.R.: The Wolff Parkinson White syndrome: problems in evaluation and surgical therapy.American Journal of Card 25:90, 1970. (Abstract)

167. Fox, T.T., Travell, J. and Molorsky, L.: Action of digitalis on conduction in the syndrome of short P-R interval and prolonged QRS complex. Arch Int Med 71:206, 1943.

168. Wolff, L.: Wolff Parkinson White syndrome: historical and clinical features. Prog Cardiovas Dis 2:677, 1960.

169. Langendorf, R., Lev. M. and Pick, A.: Auricular fibrillation with anomalous A-V excitation (WPW syndrome) imitating ventricular paroxysmal tachycardia. A case report with clinical and autopsy findings and critical review of the literature. Acts Cardiol 7:241, 1952.

170. Blinder, H., Burstein, J. and Smelin R.: Drug Effects in Wolff Parkinson White syndrome. Amer Heart J 44:268, 1952.

171. Gitsios, C.T.: Restoration of normal conduction following the administration of digitalis in a case of WPW syndrome. Amer Heart J 59:283, 1960.

172. Herrman, G.R., Oates, J.R., Runge, T.M. and Hejtmancik, M.: Paroxysmal pseudoventricular tachycardia and pseudoventricular fibrillation in patients with accelerated A-V conduction: Am Heart J 53:254, 1957.

173. Dye, C.H.: Atrial tachycardia in Wolff Parkinson White syndrome: conversion to normal sinus rhythm with Lidocaine. Am J Cardiol 24:265, 1969.

174. Gianelly,R., Griffin, J.R. and Harrison, D.C.: Propranolol in the treatment and prevention of cardiac arrhythmias. Annals of Int Med 66:667, 1967.

175. Harris, A.: Long term treatment of paroxysmal cardiac arrhythmias with propranolol. Amer J Cardiol 18:431, 1966.

176. Bester, E.M.M. and Friedlander, D.H.: Clinical experience with propranolol. Postgrad Med J 41:526, 1965.

177. Shock, J.P.P.: Beta adrenergic blocking drugs in the clinical management of cardiac arrhythmias. Amer J Cardiol 18:444, 1966.

178. Reynolds, E.W. and VanderArk, C.R.: Treatment of quinidine-resistant arrhythmias with the combined use of quinidine and propranolol. Circulation 36: Suppl II: 221, 1967.

179. Ural, N.: Clinical observations and long-term treatment with the use of beta-adrenergic blocking drugs (propranolol-Inderal) in disorders of cardiac rhythm. Fifth Europ Congr Cardiol Abstracts 1968, p 362.

180. Frieden, J., Enselberg, C.D., Rosenblum R. and Rosenberg, A.S.: Propranolol therapy in patients with chronic intractable supraventricular arrhythmias. Circulation 36: Suppl.II:113, 1967.

181. Harrison, D.C. and Griffin, J.R.: The antiarrhythmic effects of propranolol. Ann N Y Acad Sci 139:997, 1967.

182. Gettes, L.S. and Surawicz, B.: Long-term prevention of paroxysmal arrhythmias with propranolol therapy. Amer J Med Sci 254:257, 1967.

183. Knoebel, S.B., King, H. and Fisch, C.: Termination of supraventricular tachycardia complicating the Wolff Parkinson White syndrome with external countershock . Circulation 28:111, 1963.

184. Castellanos, A. Jr., Johnson, D., Mas, I. and Lemberg, L.: Electrical conversion of paroxysmal atrial fibrillation in the Wolff Parkinson White (pre-excitation) syndrome. Am J Cardiol 17:91, 1966.

185. Meyer, A.D. and Greenberg, H.B.: Cardioversion of recurrent post-operative supraventricular tachycardia in the Wolff Parkinson White syndrome. Amer J Cardiol 18:904, 1966.

186. Zeft, H.J., Cobb, F.R., Waxman, M.B., Hunt, N.C. and Morris, J.J.: Right atrial stimulation in the treatment of atrial flutter. Ann of Int Med 70:447, 1969.

187. Lister, J.W., Cohen, L.S., Bernstein, W.H. and Samet, P.: Treatment of supraventricular tachycardias by rapid atrial stimulation. Circulation 38:1044, 1968.

188. Massumi, R.A., Kistin, A.D. and Tawakkol, A.A.: Termination of reciprocating tachycardia by atrial stimulation. Circulation 36:637, 1967.

189. Haft, J.I., Kosowsky, B.D., Lau, S.H., Stein, E. and Damato, A.N.: Termination of atrial flutter by rapid electrical pacing of the atrium. Amer J Cardiol 20:239, 1967.

190. Hunt, N. C., Cobb, F.R., Waxman, M.B., Zeft, H.J., Peter, R.H. and Morris, J.J., Jr.: Conversion of supraventricular tachycardias by atrial stimulation. Circulation 38:1060, 1968.

191. Lau, S.H., Stein, E., Kowsowsky, B.D., Haft, J.I., Lister, J.W. and Damato, A.N.: Atrial pacing and atrioventricular conduction in anomalous atrioventricular excitation (Wolff Parkinson White syndrome) Amer J Cardiol 19:354, 1967.

192. Ryan, G.F., Easley, R.M., Jr., Zaroff, L.J. and Goldstein, S.: Paradoxical use of a demand pacemaker in treatment of supraventricular tachycardia due to Wolff Parkinson White syndrome. Circulation 38:1037, 1968.

193. Zeft, H.J. and McGowan, R.L.: Termination of paroxysmal junctional tachycardia by right ventricular stimulation. Circulation 40:919, 1969.

194. Braunwald, E.B., Sobel, B.E. and Braunwald, N.S.: Treatment of supraventricular tachycardia by electrical stimulation of the carotid sinus nerves. New Eng J Med 281:885, 1969.

195. Lamb, L.E.: Multiple variation in a case of Wolff Parkinson White syndrome. Am J Cardiol 4:346, 1959.

196. Prinzmetal, M., Kennamer, R., Corday, E., Osborne, J.A., Fields, J. and Smith, L.A.: Accelerated conduction: the Wolff Parkinson White syndrome and related conditions. pp 85, 86, New York, 1952, Grune and Stratton.

197. Kaplan, M.A. and Cohen, K.L.: Ventricular fibrillation in the Wolff Parkinson White syndrome. Amer J Cardiol 24:259, 1969.

198. Yahini, J.H.,Zahavi, I. and Neufeld, H.N.: Paroxysmal atrial fibrillation in Wolff Parkinson White syndrome stimulating ventricular tachycardia. Amer J Cardiol 14:248, 1964.

199. Berkman, N.L. and Lamb, L.E.: The Wolff Parkinson White electrocardiogram, a follow-up study of five to twenty-eight years. New Eng J of Med 278:492, 1968.

200. Kimball, J.L. and Burch, G.: The prognosis of the Wolff Parkinson White syndrome. Ann Int Med 27:239, 1947.

201. Okel, B.B.: The Wolff Parkinson White syndrome. Am Heart J 75:673, 1968.

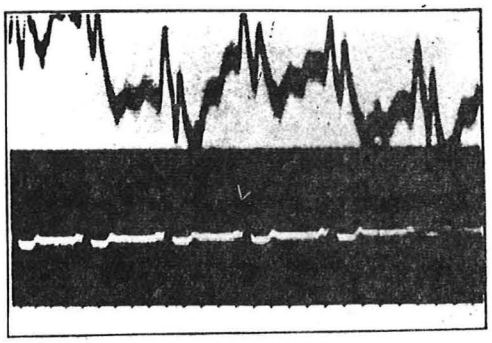


*Figure 1 - Significance of ECG findings was not recognized. Arch. Int. Med. 16:1008, 1915.*

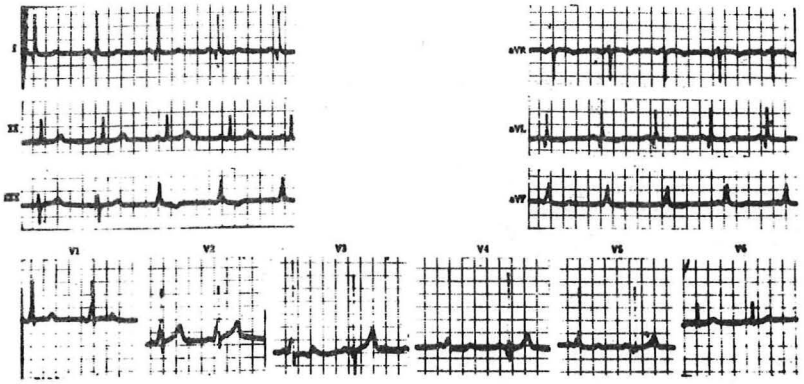


*Figure 2 - Type A WPW. Tall R wave in V1 and V2 with delta wave.*

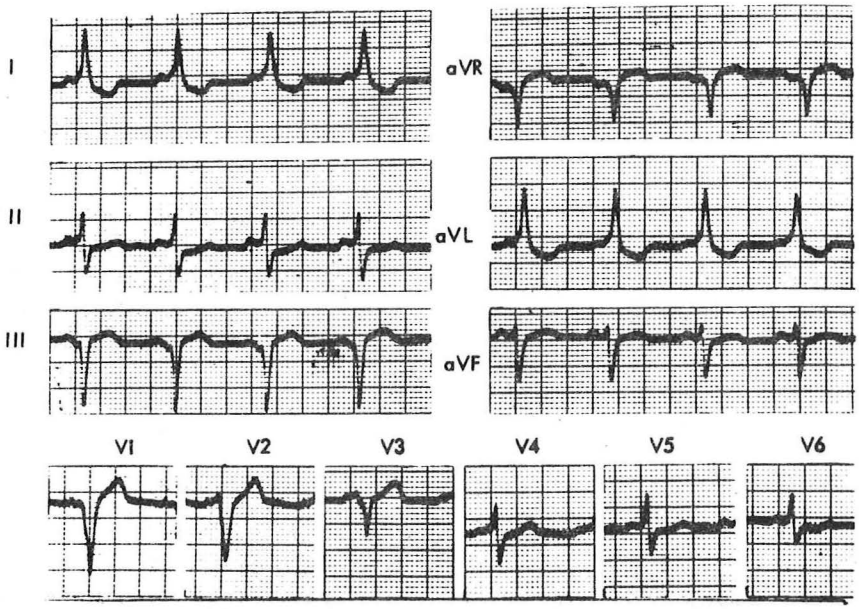



*Figure 3 - Type B WPW. QS in V1 has delta wave.*

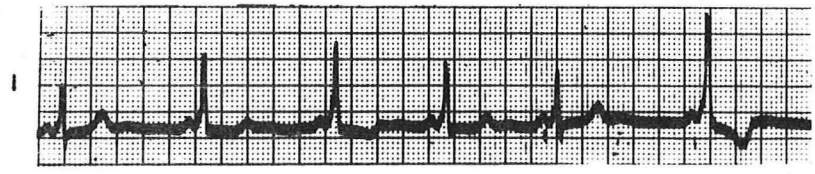



*Figure 4 - "Concertina effect."*

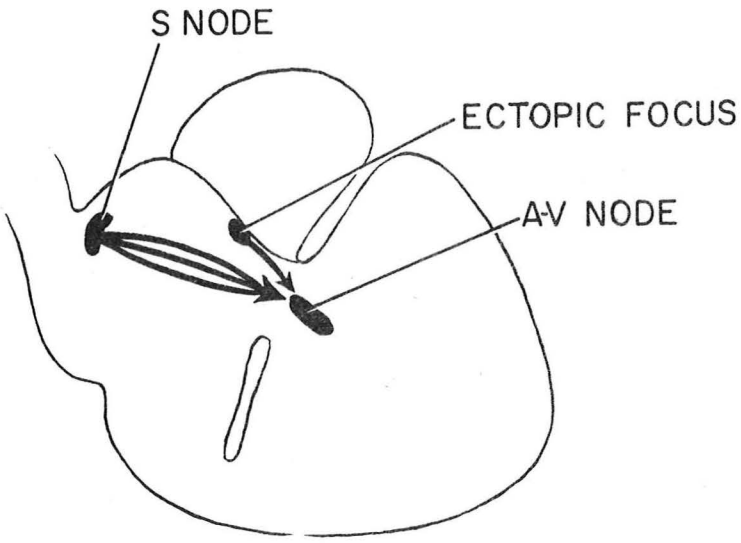



*Figure 5 - Ectopic focus theory*

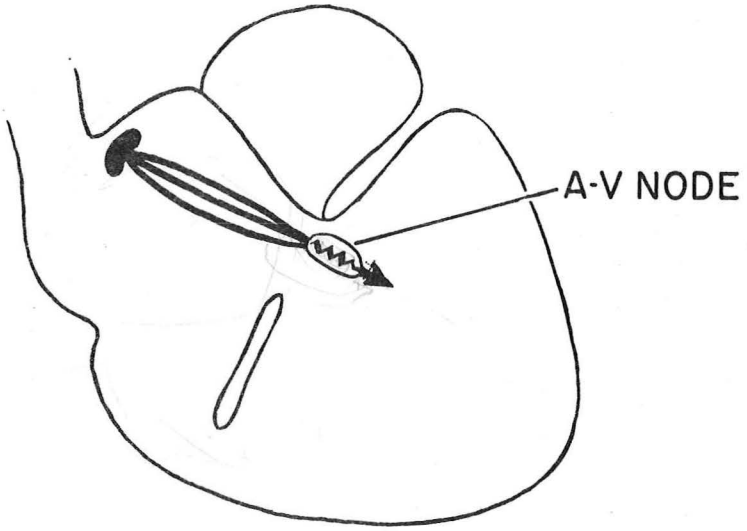



*Figure 6 - Accelerated conduction theory*

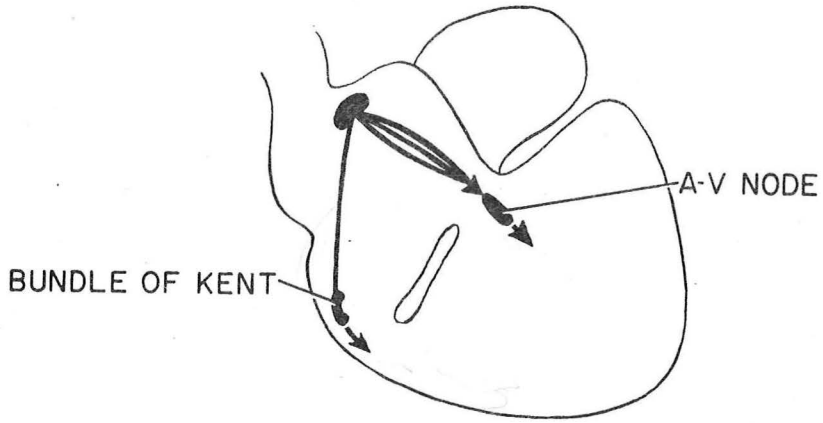



*Figure 7 - Accessory tissue theory*

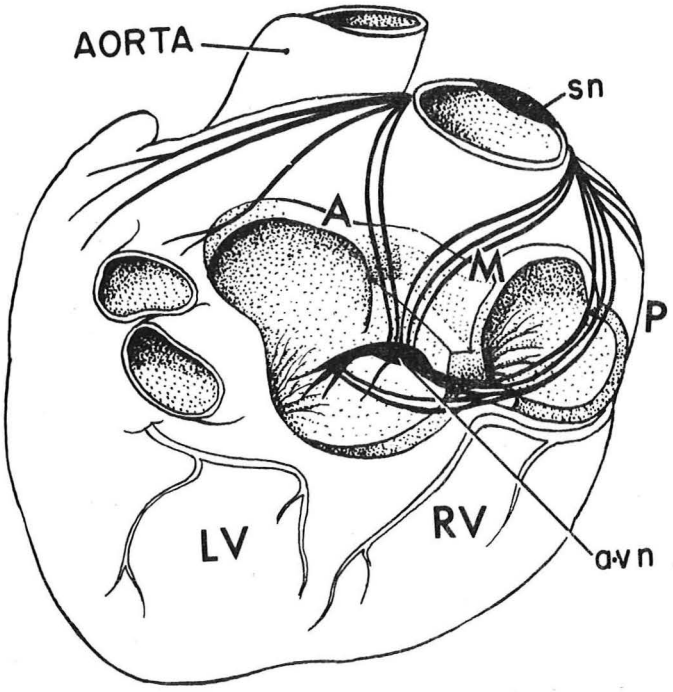
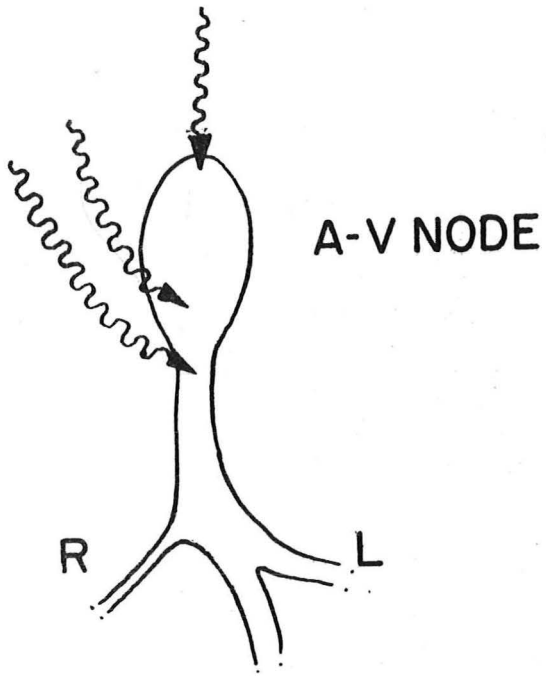
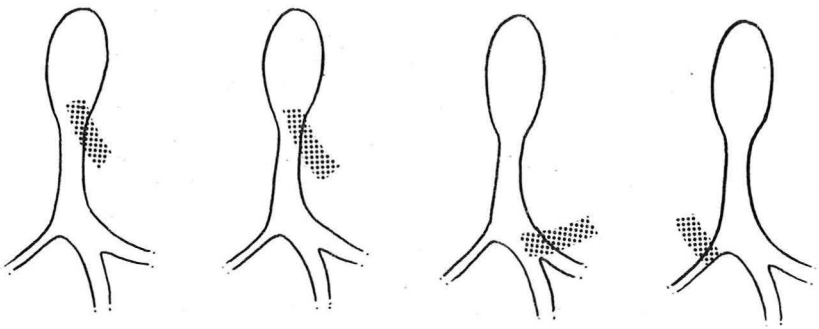



*Figure 8 - Three tracts from Sinus to A-V node.*



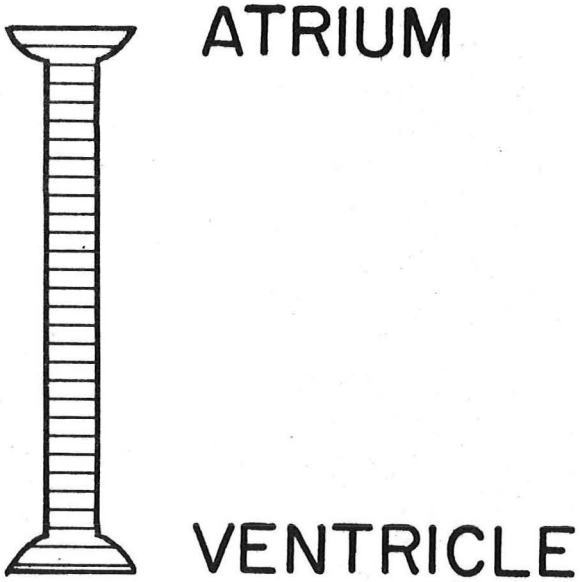

JAMES

*Figure 9 - James' bypass tracts.*



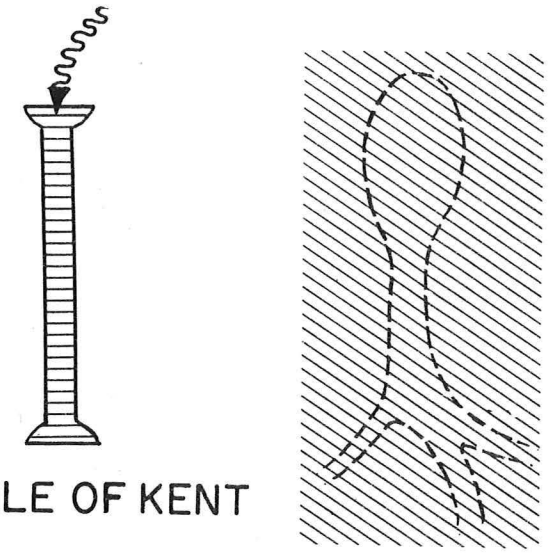
MAHAIM

*Figure 10 - Mahaim fibers between lower A-V node and ventricle.*




KENT

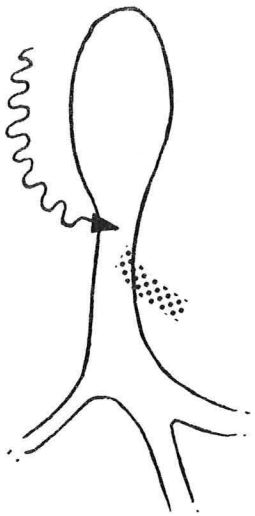
*Figure 11 - Diagram, Bundle of Kent.*



BUNDLE OF KENT

SHORT PR, Δ WAVE
PROLONGED QRS

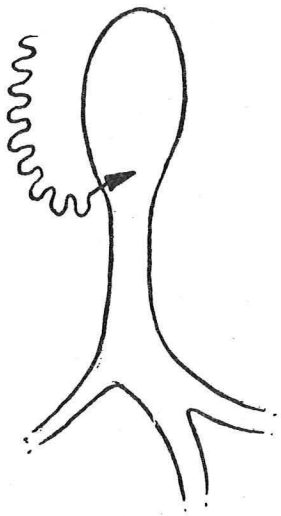
*Figure 12 - Classic WPW Bundle of Kent.*



JAMES AND MAHAIM TRACTS

SHORT PR, ΔWAVE
PROLONGED QRS

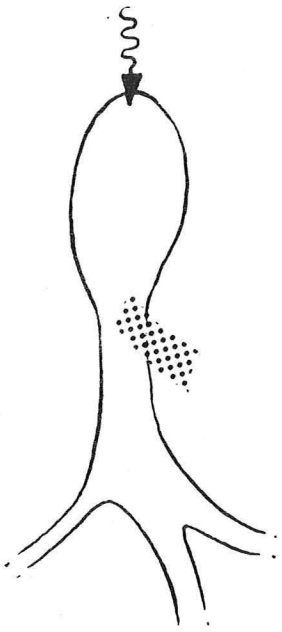
*Figure 13 - Alternate explanation for WPW, James and Mahaim Tracts.*



AVN BYPASS (JAMES) ONLY

SHORT PR,
NORMAL QRS

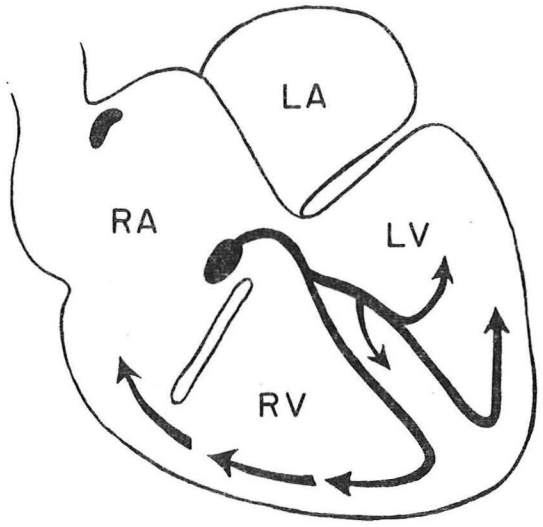
*Figure 14 - Possible mechanism for Lown Ganong Levine Syndrome.*



MAHAIM FIBERS ONLY

NORMAL (OR LONG) PR,
ΔWAVE, PROLONGED QRS

*Figure 15 - Possible mechanism for WPW variant.*




RETROGRADE KENT CONDUCTION
RESULTING IN ATRIAL TACHYCARDIA

*Figure 16 - Mechanism for atrial tachycardia*

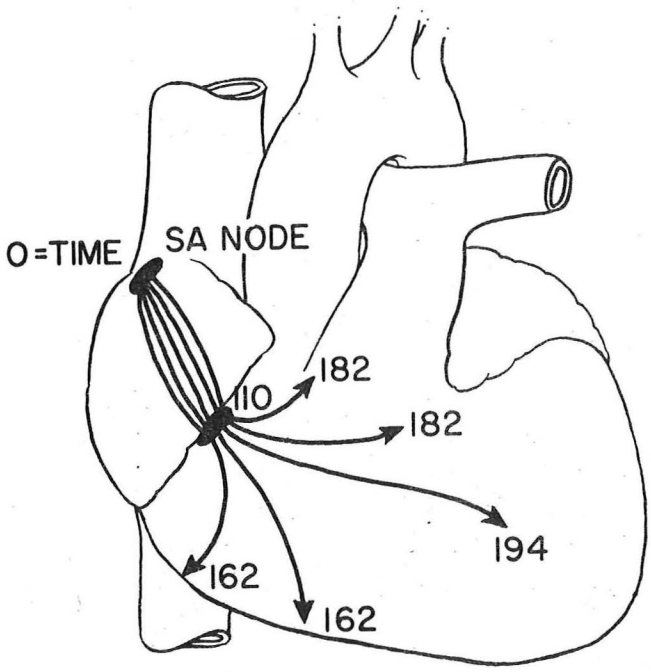



*Figure 17 - Preoperative epicardial mapping. (163)*

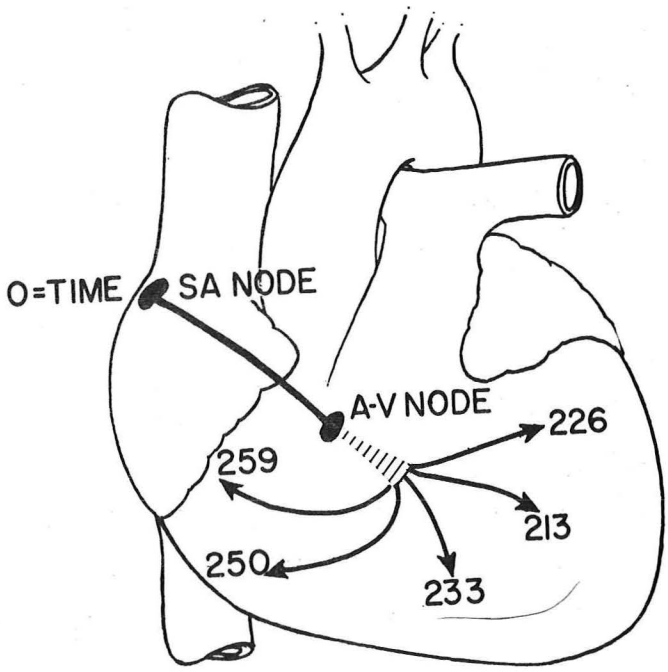



*Figure 18 - Postoperative epicardial mapping (163)*

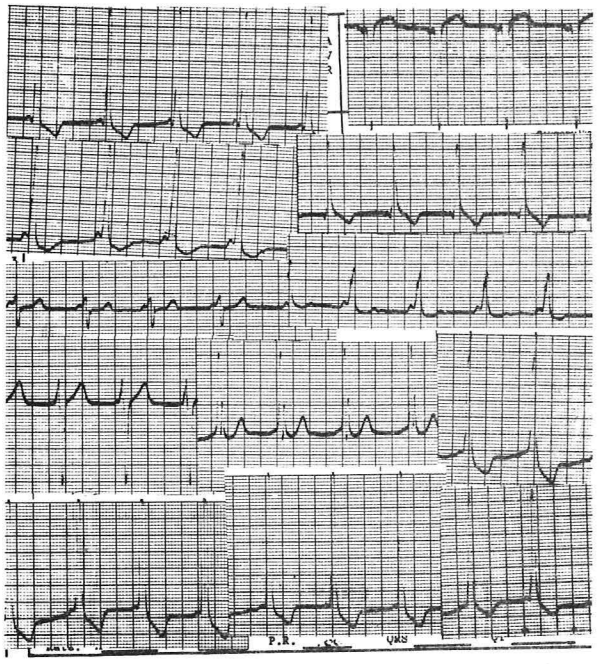


*Figure 19 - E.O. PMH 297882*
*ECG while in sinus rhythm.*

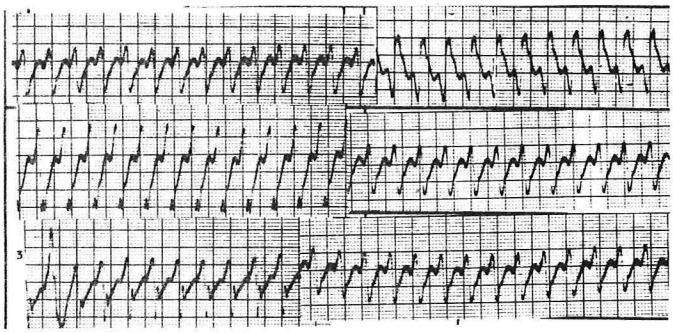


*Figure 20 - E.O. PMH 297882*
*ECG while in atrial tachycardia*

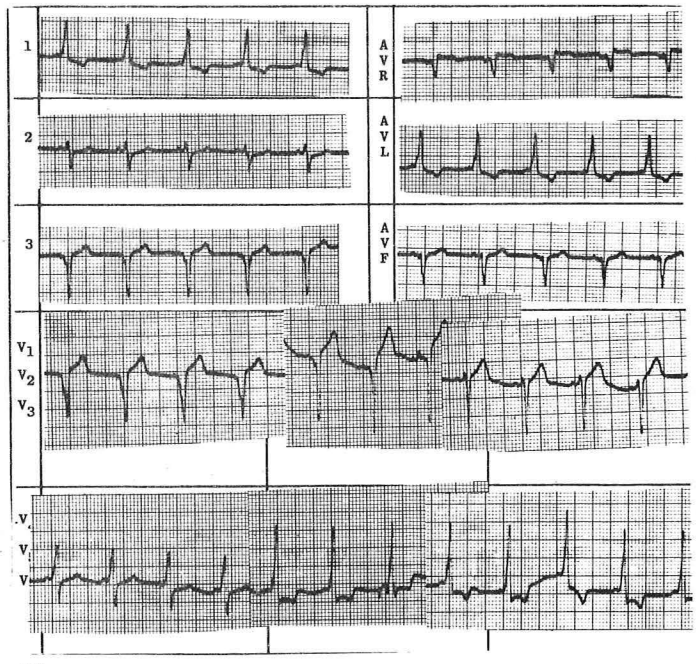



*Figure 21 - I.P. PMH 368285.*
*ECG while in sinus rhythm.*