MODERN MEDICAL MONOGRAPHS

3

ACCELERATED CONDUCTION

The Wolff-Parkinson-White Syndrome And Related Conditions

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GRUNE & STRATTON • New York
1952

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This volume is from the Institute for Medical Research, Cedars of Lebanon Hospital, Los Angeles, California; and the Department of Medicine, University of California Medical School, Los Angeles, California.

The material was presented at McGill and University of Montreal Medical Schools as part of the 13th Louis Gross Memorial Lecture, Montreal, Canada, October 17, 1951.

Preface

THE PROBLEM of the mechanism of Wolff-Parkinson-White aberration has been a topic of lively discussion for many years. Our interest in this subject grew out of certain observations made during the course of studies on the auricular arrhythmias. In examining the ventricular aberrations which sometimes occur with these arrhythmias it was seen that they often bore striking similarities to the ventricular complexes of WPW aberration. Our curiosity was further aroused when WPW complexes were observed to occur in some patients during intrathoracic operations for bronchogenic carcinoma. Moreover, in experiments in which myocardial infarction was produced in dogs by ligating the anterior descending coronary artery, it was noted that WPW complexes sometimes appeared after ligation of the artery. Here were three apparently unrelated and dissimilar situations which were capable of giving rise to ventricular aberration closely resembling the WPW type of complex, and in none did it seem possible to explain the occurrence of this aberration on the basis of any previously advanced hypothesis.

In view of these findings, and because comparatively little actual laboratory investigation of WPW aberration had been carried out, it was decided to undertake an experimental study of the mechanism of these complexes. It was soon discovered that there was no difficulty in producing the aberration in dogs by many different methods.

The most important conclusions derived from these investigations are that in both man and in the experimental animal there exist two main types of WPW aberration, a nodal type, arising from a disturbance in the A-V node, and a ventricular type, arising from a disturbance in the ventricle proper. Of these, the nodal type appears to be more common. In three patients having the nodal type of WPW aberration significant lesions were found about and in the A-V node at autopsy. In both the nodal and the ventricular types of WPW aberration, the auricular impulse was found to reach the ventricles

over the normal conduction system, and not by way of anomalous anatomical A-V connections. Since evidence is presented that the fundamental disorder in all types of WPW aberration is a diminution of the usual, normal delay of the excitation wave at the A-V node, resulting in abnormally rapid transmission of the impulse through the node, the term "accelerated conduction" has been suggested to designate this phenomenon and the aberration resulting from it. This study is the second in a series of investigations entitled, "Studies on the Mechanism of Ventricular Activity".

Since previous consideration of WPW aberration had been confined largely to the classical, congenital syndrome, at first it was rather a surprise when numerous acquired forms of the aberration were found to exist clinically. In fact, the incidence of acquired WPW aberration appears to be greater than that of the congential form. It is hoped that the reader, if he watches for such cases, will also find many more clinical examples of acquired types of WPW aberration than were formerly recognized.

Because "accelerated conduction" in its various forms is not rare and because obviously much remains to be learned about this disorder, it is hoped that other observers will look for and study cases with this disturbance so that the many lacunae in our knowledge of this subject may be filled in.

For their help and cooperation in these investigations and in the preparation of this monograph, the authors wish to thank the following: Dr. Alfred Dantes, for his invaluable aid in the writing and preparation of the manuscript; Drs. John Martin Askey, John J. Sampson, John S. Lawrence, A. C. P. Bakos, A. A. Kattus, for their careful and critical reading of the manuscript; Mr. Louis Fields, for helping develop much of the apparatus used in the experiments, especially the simultaneous photography of heart and electrocardiogram; Dale Gillette, the medical photographer, whose meticulous care and skill is responsible for the electrocardiographic reproductions and the photomicrographs; Mr. Homer Alexander of Brown & Caldwell, engravers, for the color plate (Figure 1) and for Figures 26 and 27.

This study was made possible by the financial support of Morton May, Nathan Dauby and Nathan Loeser (Trustees of the Louis D. Beaumont Trust Fund), Mrs. Anita May, Mr. J. Benny, Mr. H. Borun, Mr. J. M. Schmook, Mr. and Mrs. W. Goetz, Mr. E. Mannix,

Mr. Adrien Pelletier, Mr. I. Berlin, Mr. L. Spitz, and Mr. M. Silberberg.

We wish to express our thanks to the following authors and their respective publishers for allowing us to reproduce their illustrations: Dr. R. Ohnell and Acta Medica Scandinavia for Figures 30A and 31A. Dr. M. Melnick for Figure 39 and kindly loaning us Case #8. Drs. H. D. Levine and J. C. Burge, Jr. and C. V. Mosby Co. for Figure 41. Drs. R. Langendorf, M. Lev and R. Pick for Figure 42. Drs. J. Gould and A. S. Mundal and C. V. Mosby Co. for Figure 43. Drs. S. P. Schwartz and A. Jezer and Lea and Febiger for Figure 44. Dr. W. Dressler and C. V. Mosby Co. for Figure 48. We also wish to thank the Division of Laboratories, Cedars of Lebanon Hospital, for the slides reproduced as Figures 38 and 40.

Without the work of Eleanor Gerlach, our co-worker and secretary, this monograph would not have been possible. We also acknowledge the patience of her husband, George.

Dr. Harry Goldblatt has not only performed an invaluable service to us by his careful study of the histology of the A-V node, but he has read and criticised the manuscript. As Director of the Institute for Medical Research, Cedars of Lebanon Hospital, he has given us the utmost cooperation and guidance. We extend our sincere thanks.

THE AUTHORS

To Blanche

As clinical observers, we study the experiments which Nature makes upon our fellow-creatures. These experiments, however, in striking contrast to those of the laboratory, lack exactness, possessing as they do a variability at once a despair and a delight—the despair of those who look for nothing but fixed laws in an art which is still deep in the sloughs of Empiricism; the delight of those who find in it an expression of a universal law transcending, even scorning, the petty accuracy of test-tube and balance, the law that in man "the measure of all things," mutability, variability, mobility, are the very marrow of his being.

—William Osler

in his Address to the
Army Medical School, Washington,
February 28, 1894.

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CHAPTER I

Introduction

THE PURPOSE of this communication is to present and interpret the results of a series of experimental investigations of the Wolff-Parkinson-White syndrome. The interest of this laboratory in this problem was aroused when, during certain investigations on ventricular aberrations occurring with auricular arrhythmias, it was noticed that some of these aberrations resembled in many respects those which are commonly associated with the WPW complex.[1] These similarities suggested that an experimental study of the WPW syndrome might throw new light on the problem of ventricular aberration in general, especially since existing theories concerning its mechanism seem to be inadequate.

An important conclusion of these investigations on the WPW syndrome was that this aberration is not dependent upon the presence of anomalous anatomical A-V connections for its production, and that the auricular impulses in WPW complexes are transmitted to the ventricle in an *abnormal* manner by way of the *normal conducting* system. The fundamental disorder appears to consist of a *failure of a part of the A-V node to delay the auricular impulse for the normal period of time before allowing its passage to the ventricles.* It is suggested that the term "accelerated conduction" be employed to designate this condition since it describes the altered physiology more exactly than other terms.

This physiologic disturbance, accelerated conduction of the excitation wave through one part of the A-V node instead of the normal uniform delay at the node, is the fundamental disorder responsible for a number of clinical conditions. The best known and most common of these is the WPW syndrome, but as will be shown, there are several others which result from the same basic abnormality.

A thorough understanding of all the manifestations of the WPW

syndrome is necessary before the experimental portion of this study can be read.

Definition

The WPW syndrome may be defined as a condition characterized by electrocardiographic and sometimes clinical abnormalities, consisting of (1) variable shortening of the P-R interval, usually to 0.12 second or less; (2) widening of the QRS complexes, usually to 0.10 second or more; (3) changes in the direction of the QRS complexes so that left or right axis deviation may be present; (4) changes in the ST segments and T waves; and (5) the frequent occurrence of paroxysmal supraventricular tachycardia and less commonly other supraventricular arrhythmias namely extrasystoles, flutter and fibrillation. The syndrome is observed in patients of all ages, although more commonly in those below the age of 30. The male sex appears predominantly affected. Usually, other evidence of heart disease is lacking, but exceptions to this rule have been observed.[2] Sudden death may occur from paroxysms of tachycardia even in the absence of organic heart disease,[3, 28] but the condition is ordinarily a benign one.

A large number of variations from these typical findings has been described. Only those which are pertinent will be discussed here. Among the unusual findings that have been reported is a P-R interval exceeding 0.12 second.[3a, 4] Rarely, the P-R interval in a given case may vary in duration.[5] Alternation of short and normal P-R intervals[6] and intermittent short P-R intervals have also been observed.[7]

While the common abnormality with reference to the QRS complexes is a widening to 0.10 second or more with a gradual initial slope and slurring at its onset only, Ohnell[3a] and Fox[8] have reported cases with short P-R intervals and QRS complexes of normal width. Slurring, however, was present in these QRS complexes. Lown, Ganong and Levine[9] have called attention to a group of patients subject to paroxysmal supraventricular tachycardia who, during sinus rhythm, had short P-R intervals and normal QRS complexes. Burch and Kimball[10] have described five types of ventricular aberration in the WPW syndrome. In one of these, the QRS component is not widened. Slurring may affect all or any part of the QRS, or may be absent altogether. Abnormalities in the ST segments and T waves are frequently observed.

An interesting variation from the usual form of WPW aberration is the "concertina effect."[3a] This consists of a progressive shortening of the P-R intervals with a corresponding widening of the QRS complexes in a series of WPW beats, giving the impression that the complexes are being "pulled out." The opposite phenomenon may then be observed, namely, a progressive lengthening of the P-R intervals and a corresponding narrowing of the QRS complexes, giving the appearance of "pushing in."

Another variation from the typical WPW aberration, and one of great importance, is the frequent occurrence of A-V nodal rhythm. With the heart continuing to beat at a normal rate, the A-V node may assume the role of pacemaker spontaneously or as a result of carotid sinus pressure.[11] In the many instances in which A-V nodal rhythm has been observed, the QRS complexes have usually been normal.

In the typical case, the widening of the QRS exactly compensates for the shortening of the P-R interval, resulting in a P-J interval of normal duration. Many instances have been found however, in which the P-J interval was shorter than the normal.[12]

When the P′ waves of the supraventricular arrhythmias associated with the WPW syndrome can be identified, they are generally inverted in leads 2 and 3. In the course of paroxysms of tachycardia, previously widened QRS complexes may or may not approach the normal. Wolff[13] has described two cases with very wide QRS complexes during this arrhythmia.

Historical

The first comprehensive description of the syndrome was published by Wolff, Parkinson and White[14] in 1930, with a review of 11 cases. Prior to this, there had appeared in the literature five scattered case reports with characteristic electrocardiographic findings. Of these, the earliest appears to have been that of Wilson[15] published in 1915. Six years later, a similar case was described by Wedd,[16] and in 1926, Bain and Hamilton[17] reported a third instance. Two more such cases were described in 1929.[18, 19]

Interest in both the theoretical and practical aspects of the syndrome was accelerated by the paper of Wolff, Parkinson and White, and in the 20 years since its appearance, a considerable body of literature comprising upwards of 150 papers concerning this subject has

accumulated. It is not within the scope of this work to present a detailed discussion of the historical development of the syndrome; excellent reviews may be found in the papers of Bishop,[20] of Lepeschkin,[21] and in the comprehensive monograph of Ohnell.[3a]

Theories Concerning The Mechanism of Production of WPW Complexes

The number and diversity of the hypotheses and suggestions which have been advanced in an effort to explain the mechanism of production of the WPW complex is remarkable. Ohnell tabulated and summarized the numerous theories in existence at the time of publication of his monograph,[3a] and was able to include 80 papers comprising about 40 different ideas concerning this subject. Furthermore, since that time (1944) several additional theories have been propounded, bringing the total number close to 50. Generally speaking, all these theories fall into one of the following two groups.

(A) Those which postulate the presence of a functioning accessory anatomical pathway by means of which the auricular impulse reaches the ventricle. This concept implies that the accessory bundle is a congenital anomaly, either the bundle of Kent or some other congenitally present connection between the auricles and ventricles. First to suggest this hypothesis were Holzmann and Scherf[22] and Wolferth and Wood.[23] According to the theory of these investigators, the auricular impulse passes over the aberrant anatomical bundle and reaches one ventricle early, thus setting off a premature ventricular response, and accounting for the short P-R interval. Shortly after this has occurred, the other ventricle is activated in the normal manner. This slightly asynchronous ventricular excitation is therefore responsible for the QRS aberration. This hypothesis has been widely accepted.

(B) A wide gamut of theories[3a] postulating various bizarre and complicated abnormalities of auriculoventricular or intraventricular conduction and having in common only the concept that the impulse utilizes the normal conduction pathway. None of these theories has been generally accepted.

Group A: The more popular theory of an accessory bundle has led to the use of the term "Bundle of Kent Syndrome" when referring to the WPW phenomenon.

There is a considerable body of evidence in support of the thesis that potentially functional, anomalous, anatomical conducting pathways joining auricle and ventricle are present in some mammalian hearts. Kerr and Sampson were among the earliest investigators to recall attention to the extensive microscopic studies of Kent[24] who published his first findings in 1893. Holzmann and Scherf[22] and Wolferth and Wood[23] suggested this work might afford a basis for WPW aberration. Kent demonstrated the presence of a rich muscular anastomosis between auricle and ventricle in young rats and rabbits. He also observed that the number of these connections decreases as the age of the animal increases, or as the animal scale is ascended. They are present in the monkey, although relatively few in number. More significantly, Kent was able to show the presence of an analogous structure in the human heart, a muscular bridge lying at the right cardiac border and joining the auricle and ventricle. To this he gave the name of "right lateral bundle," and implied, but did not actually state, that this connection was found in all the hearts which he examined. In later studies by Kent and his collaborators, the essential finding of accessory muscular A-V connections in animals was confirmed, but no further mention was made concerning investigations on the human heart. Similar muscular connections had been found as early as 1876 by Paladino,[25] although his researches were by no means as comprehensive as those of Kent.

A number of subsequent workers have also brought forth evidence pointing to the existence of accessory muscular A-V connections. Mall[26] reported their presence in both sides of the heart in embryos. Mönckeberg[27] described such a pathway in a "greatly deformed" heart studied post-mortem. More recently, Wood and his associates[28] were able to study the heart of a 13 year old boy who during life had typical ECG findings of the WPW syndrome and died suddenly. They found anatomical evidence of at least three accessory A-V muscle bundles situated in the same general region as those described by Kent. A left sided accessory connection was also found by Ohnell[3a] in the heart of a patient with the WPW syndrome. Others[29] have reported similar histologic findings. The careful studies of Robb and her associates[30] have demonstrated the existence in the human embryo of such connections and have confirmed and extended the work of Mahaim[31] who showed the presence of branches connecting the main