OKHAWA and his colleagues have provided histologic evidence that a lesion in the distal or branching portion of the His bundle, and thus actually within the ventricles, may produce complete atrioventricular (AV) block on the surface ECG associated with “AH” block on the His bundle electrogram (atrial impulses blocked proximal to the His deflection) and that this may be associated with an escape rhythm with narrow QRS complexes that are similar to normally conducted complexes (an “AV junctional escape rhythm”). They have also presented evidence that a lesion that blocks the two bundle branches near their origin may produce complete AV block on the surface ECG associated with an HV block on the His electrogram (an intraventricular block between the His depolarization and the ventricular depolarization), accompanied by an escape rhythm with wide aberrantly conducted QRS complexes (a “ventricular escape rhythm”). These observations are a further step to a better understanding of the origin of disorders of conduction and dysrhythmias and they remind us again that the surface ECG does not always provide a simple reflection of electrical events within the heart.

When the ECG became available, it provided what appeared to be a new and beautifully simple representation of the process of cardiac depolarization: an impulse originating in the sinoatrial node depolarized the atria, producing the P wave; the delay that occurred as the impulse traversed the atria and the AV node was represented by the PR interval; the depolarization of the ventricles, initiated by the AV node, was represented by a QRS complex that lasted not more than 0.10 second and had a relatively invariant configuration in normal hearts. Several clinical corollaries arose: a delay between the onset of the P wave and the onset of the QRS represented an AV block, the site of which was somewhere in the atrium or AV node; a prolonged QRS of unusual configuration represented an intraventricular block originating somewhere in the ventricle; a QRS complex lasting no longer than 0.10 second, not preceded by a P wave, and having a configuration like that of a normally conducted complex must originate in the AV node; and a prolonged, aberrantly conducted QRS complex not preceded by a P wave must originate in the ventricles.

These relatively simple assumptions, enshrined in elementary cardiology textbooks, served clinical medicine well during the first half century; but even as they were being formulated, there was information that showed them to be inaccurate. In 1910, when Epinger and Rothberger cut the bundle branches of the hearts of dogs and recorded the first ECGs of bundle branch block, they recorded instances in which dogs with both of the bundle branches severed also had a complete AV block. Two years later Mathewson described the electrocardiographic patterns of right and left bundle branch block and included ECGs from patients who had complete AV block accompanied by ventricular escape rhythms that were characterized by aberrant QRS complexes. In 1923, Wilson and Herman, and in 1931, Mahaim, reported further experimental and histologic studies showing that the ECG appearance of AV block with ventricular escape rhythms having a bundle branch block configuration could be created by lesions in the bundle branches. In 1936, Yater et al. described other cases in which a lesion of the branching portion of the His bundle was associated with a complete AV block and narrow, normal-looking QRS complexes. Such narrow ventricular escape beats were studied by various electrocardiographers in the next 30 years. In 1967, Rosenbaum and associates produced evidence that ectopic depolarizations with narrow QRS complexes resembling normally conducted QRS complexes might originate within the proximal fascicles of the bundle branches. By then, it was understood that a complete AV block might be created by a lesion that lay anywhere between the sinus node and the proximal fascicles of the bundle branches, and that escape rhythms and ectopic rhythms with QRS complexes not preceded by P waves also might originate anywhere from the AV node to the proximal fascicles of the bundle branches. Okhawa and his colleagues present evidence that support these findings.

Even as it became evident that AV blocks as well as apparently normally conducted escape rhythms and ectopic depolarizations might originate within the proximal parts of the ventricular conduction system, it also became evident that aberrantly conducted QRS complexes initiated by supraventricular impulses could not be distinguished from those arising from ventricular foci. In 1912, Sir Thomas Lewis had described auricular premature contractions with QRS complexes that were aberrantly conducted. He and Master later reported experimental studies indicating that this phenomenon could be attributed to incomplete recovery of the ventricular conduction system. Other causes for aberrantly conducted QRS
complexes associated with supraventricular rhythms were soon recognized or postulated, including the presence of concurrent bundle branch block, aberrant pathways of conduction within the atrium, preferential pathways within the AV junction, and aberrancy associated with QRS complexes originating in the lower, ventricular, portion of the AV node.

The problem of differentiating ectopic depolarizations and tachycardias of ventricular origin from those of supraventricular origin with aberrancy was recognized to be especially difficult. Careful studies of the characteristics of dysrhythmias as seen on surface ECGs, as carried out by Katz, Langendorf, Pick and Langendorf, and Mariott and Sandler, among others, yielded many clues that can be used to determine the probable site of origin of aberrantly conducted dysrhythmias; but this determination in all cases requires the careful review of an adequate sample of electrocardiographic complexes that are accurately recorded and reproduced. All of these investigators have concluded that, in many cases, the determination cannot be made on the basis of the surface ECG alone. The use of His bundle electrograms reinforces this conclusion.

Thus, cardiologists have known for many years that there is some indeterminacy associated with the electrocardiographic diagnosis of dysrhythmias and disorders of conduction from the surface ECG, but until the 1960s this appeared to be a matter of consequence for only a few patients who had serious heart disease. Since then, this matter has become of major concern for more people. Sudden death accounts for almost half of the deaths attributed to coronary heart disease each year, and the great majority of the sudden deaths are the result of the sudden development of a fatal arrhythmia. Many persons who have been resuscitated from what would otherwise have been fatal arrhythmias have been found, after their resuscitation, to have chronic dysrhythmias and disorders of conduction. A large proportion of the people whom we have examined before they experienced sudden fatal arrhythmias also have chronic dysrhythmias and disorders of conduction. The availability of long-term tape recordings of the ECGs of active people has made it clear that a very large proportion of men older than age 50 years (and probably women also) have abnormalities of cardiac rhythm and conduction. The large samples of cardiac complexes that are provided by these recordings have revealed that dysrhythmias may be highly variable in their frequency, both from hour to hour and from day to day; that they often appear to be both of ventricular and supraventricular origin; and that in many cases the variety and complexity of the dysrhythmias that are observed increase as the frequency of dysrhythmias increases or as the sample of cardiac complexes that is examined becomes larger. People with dysrhythmias often have abnormalities of their PR and QRS conduction that may also be transient and change in form. Thus, the diagnostic problem of evaluating cardiac dysrhythmias in terms of their origins and mechanisms has acquired a magnitude, complexity, and potential importance much greater than it appeared to have in the past.

The importance of the accurate diagnosis of dysrhythmias and disorders of conduction has been heightened by the evidence that some apparently have a prognostic significance that is much more ominous than others. The magnitude of the risk that is associated with individual phenomena of rhythm and conduction is difficult to determine because they often occur in association with each other and in association with other manifestations of heart disease. The assessment of risk is also difficult because most of the prospective studies have been based on special and unrepresentative samples of the population, such as patients who have experienced myocardial infarctions. Nevertheless, enough information has been generated to suggest a widely held hypothesis that supraventricular dysrhythmias and transient or permanent first-degree AV block, for example, are relatively benign, whereas ventricular dysrhythmias and intraventricular block, as well as higher grades of AV block, are much more ominous and carry a risk of the occurrence of fatal arrhythmias. Programs for the treatment of individual patients and programs for the development of antiarrhythmic drugs have been based on these hypotheses.

The findings of Okhawa and his colleagues add one more bit of evidence to a body of information that renders all of these hypotheses suspect if they are based upon evidence from the surface ECG alone. As one examines the large body of electrocardiographic data from individual patients that is made available by extended electrocardiographic recordings, one sees that PR (AV) conduction may be transiently prolonged; that those who have prolonged PR conduction may or may not also have prolonged QRS (intraventricular) conduction, which also may be transient and may change in configuration; that QRS complexes that are premature or delayed are sometimes associated with P waves and sometimes are not, and that those that meet certain criteria for ventricular premature complexes or ventricular escape rhythms may or may not be discernibly different from those that are called AV junctional in origin; and that QRS complexes that meet suggested criteria for left ventricular origin in lead V₅ sometimes do not satisfy the criteria in lead V₁ also. All of these factors suggest that there are changing pathophysiologic processes in the conducting tissue of many hearts that we do not fully understand which produce signals in the surface ECG that are not easily classified by the usual procedures.

Perhaps our classifications of disorders of conduction and of rhythm as seen on the surface ECG should be reconsidered and the nomenclature that goes with them revised. Thus, for example, it might be well to speak of PR or PQ conduction on the surface ECG rather than AV conduction, because the two terms clearly are not synonymous, and to speak of the duration of a PR interval or the presence of 2:1 PR block as empirically representing what is seen in the surface tracing without implication as to the origin of these.
phenomena. Similarly, it might be well to describe and classify QRS complexes not on the basis of their assumed origin, but on the basis of their relation to the P wave (preceding, fused, succeeding or absent), their duration, and their configuration in specified leads — for example, in V₆ and V₇.

Such a procedure might clarify our thinking about the limitations of the surface ECG. It would also enable us to collect an empirically coherent and systematically classifiable body of information about manifestation in the surface ECG that could be correlated with, and interpreted in the light of, information from His bundle electrograms and other sources of electrocardiographic data as well as from pathologic studies as this information becomes available. Such systematically descriptive electrocardiographic data might also be applicable to the increasingly large body of information that is becoming available from electrocardiographic recordings. It could be made the basis for classifications of electrocardiographic complexes by automatic data processing equipment as pattern recognition programs are improved. These data could thus become available for the types of systematic analyses of repetitive patterns of electrocardiographic data. All of this might give us a clearer idea of the relationships between different kinds of dysrhythmic manifestations and conduction disorders as seen on the surface ECG and the underlying pathophysiologic phenomena, and it might form the basis for a better understanding of the diagnostic and prognostic significance of these phenomena.

References

3. Wilson FN, Herman GR: An experimental study of incomplete bundle branch block and the refractory period of the heart of the dog. Heart 8: 229, 1921
13. Lewis T: Observations upon disorders of the heart's action. Heart 3: 279, 1912
19. Marriott AJL, Sandler JA: Criteria, old and new, for differentiating between ectopic ventricular beats and aberrant ventricular conduction in the presence of atrial fibrillation. Prog Cardiovasc Dis 9: 18, 1966
22. Hinkle LE Jr: The Antecedents of Sudden Death: Prospective Studies. Report prepared for the Cardiovascular Disease Branch, Division of Heart and Vascular Disease, National Heart, Lung, and Blood Institute, Bethesda. Copies available from the National Technical Information Service, 5285 Port Royal Road, Springfield, Virginia 22151
The indeterminate representation of disorders of conduction and dysrhythmias on the surface electrocardiogram: some practical consequences.

L E Hinkle, Jr

doi: 10.1161/01.CIR.64.2.232

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/64/2/232.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/