of the graphic records of different adventitious sounds are easily seen, but specific features characteristic of fine crackles or rhonchi, for example, cannot be readily identified. When adventitious sounds originate from multiple sites and produce a cascade of sounds, conventional recording techniques do not allow graphic representation of individual crackles or wheezes. This problem was overcome by Murphy et al. They used an expanded time axis (800 to 1,600 mm/sec) in their time-amplitude displays and were then able to identify specific features of different adventitious sounds. The following two results were of singular importance: (1) the similarity in tracings made from recordings of adventitious sounds provided by three separate groups; and (2) the ability of Murphy et al. to characterize adventitious sounds solely on the basis of their graphic appearance.

Quantitative methods and graphic records for measurements of respiratory sounds are all well and good, but wherein is the wisdom of relinquishing the tried and true term, rale? Why replace it with crackle? Like Lewis Carroll's Alice, we, too, can assign words whatever meaning we wish; but redefining rale in acoustic terms, as Forgacs notes has been done for crackle, will be difficult to achieve, in view of the current widespread use of rale and the equally widespread number of meanings. A new term with a well-defined meaning can be incorporated into our regular nomenclature with only modest difficulty. Consider the alternative of continuing to use the term, rale, with the attendant uncertainty over what particular definition the user had in mind. The word, crackle, will be seen with increasing frequency. It has been recommended for general use by a subcommittee on nomenclature to the membership of the American Thoracic Society.

Considerably more work is needed to establish satisfactory clinical, pathologic, and physiologic correlations with pulmonary sounds. Phonopneumography has a long road to travel before it achieves the importance that phonocardiography now enjoys, but the techniques and concepts are now at hand.

David W. Cugell, M.D., F.C.C.P. Chicago

*Basley Professor of Pulmonary Diseases, Northwestern University Medical School. Reprint requests: Dr. Cugell, 303 East Chicago Avenue, Chicago 60611.

REFERENCES


The Manifestation of Bundle Branch Block due to Lesions within the His Bundle

A Dilemma in Electrocardiographic Interpretations

Clinically, the electrocardiographic patterns of bundle branch block are generally attributed to a corresponding anatomic lesion or lesions in the bundle branches. Contrary to these classic views, it can now be stated that a discrete lesion or an alteration in refractoriness within the His bundle may also result in similar electrocardiographic patterns of partial or complete bundle branch block. These conclusions are based on my recent study which documents asynchrony in conduction or longitudinal dissociation within the His bundle for the first time in man.

During electrophysiologic studies in 27 patients with an electrocardiographic pattern of bundle-branch block or isolated axis deviation, the electrocardiographic abnormalities could be either produced or abolished at will by selective stimulation of different portions of the His bundle from the right side of the heart. During these studies, it was demonstrated that stimulation of the uppermost portion of the His bundle produced QRST complexes with left bundle branch block that were identical to those with sinus rhythm or atrial pacing with an interval from stimulus to QRS complex that was equal to the His-ventricle (H-V) interval. Stimulation of the His bundle at a slightly distal site abolished the left bundle branch block and normalized the duration of the QRS complex. The resultant interval from stimulus to QRS complex was greater than or equal to 40 msec, although slightly shorter (by 5 to 20 msec) than the control H-V interval. In some of the cases with left bundle branch block and left axis deviation, both the duration of the QRS
complex and the axis were normalized, whereas in others, left axis deviation persisted, despite a normalization of the duration of the QRS complex. This suggests multiple sites of lesions in patients with a similar electrocardiographic pattern.

My study also demonstrated that in cases with isolated left axis deviation and a narrow QRS complex, the shift in axis could be normalized by selective stimulation of the His bundle. Similarly, rate-dependent left bundle branch block could be abolished by stimulation of the His bundle at a site distal to the site related to alterations in the refractoriness. These findings indicate a discrete lesion within the His bundle as the underlying cause for left bundle branch block or isolated left axis deviation (or both) that were abolished by stimulation of the His bundle distal to the lesion, resulting in synchronized depolarization of all of the fibers of the His bundle destined for the bundle branches. The picture is completed by demonstrating not only an abolition of asynchronous conduction but also by the production of asynchrony in conduction within the His bundle, as all types of QRS complexes (left bundle branch block, narrow, and right bundle branch block) were also produced by selective stimulation of the His bundle in the latter study.

The anatomic substrate for findings of longitudinal dissociation within the His bundle has been reported by James and Sherf. Their studies show that the His bundle is partitioned into narrow cords by collagen. The various cords run in its long axis, have relatively few cross-connections, and are insulated from each other by the collagen. Until these recent observations in man, the counterpart to the histologic studies were lacking. An experimental study in isolated canine tissue has also demonstrated development of bundle branch block following transsection of about 50 percent of the cross-section of the His bundle. Findings in another canine study are also in agreement with these conclusions.

Whether longitudinal dissociation is a characteristic of a histologically normal His bundle or is manifested only in pathologic conditions cannot be decided with certainty at the present time. The observations reported in man were made in patients who probably had pathologic abnormalities in the His bundle, since (1) the H-V interval was prolonged in most of the patients, and (2) in some, despite normalization of the QRS complex with distal stimulation of the His bundle, the interval from the stimulus to the QRS complex was longer than the normal time of conduction to the ventricles.

These observations in man were initially made by me as early as 1969 (unpublished data). Due to the fact that they were so alien to the classic views, publication was delayed until the collected evidence was overwhelming and these observations had been documented in 27 patients; however, subsequent to the presentation of these data during the 28th annual scientific session of the American College of Cardiology in March 1977, several electrophysiologists have made inquiries regarding this subject and have mentioned isolated cases of a similar nature. From these inquiries and discussions, I am sure others will soon be publishing similar data confirming these findings.

These observations have several important clinical implications. First, a dilemma is posed in electrocardiographic interpretations, i.e., anatomic localization of the lesions on the basis of electrocardiographic patterns. The electrocardiographic patterns of bundle branch block or axis deviation can no more be considered synonymous with anatomic lesions in the bundle branches or their subdivisions. In addition to the previous objections, these data raise further questions about the validity of using the nomenclature of "fascicular blocks" or "hemiblocks."

Secondly, localization of the anatomic site of the lesion from the standard electrocardiogram may be misleading, not only for academic purposes but also from a clinical viewpoint. The prognosis of patients with an electrocardiographic pattern of right bundle branch block and left axis deviation may vary, depending upon the site of lesions and whether they are localized within the His bundle or in the right bundle branch and the anterior group of left bundle-branch fibers. It may be speculated that the lesions located within the His bundle may be more critical and at higher risk of progressing to atrioventricular block than the peripheral lesions; however, definitive answers should follow future studies.

Thirdly, prospective studies dealing with prognostic values of the H-V interval should be reevaluated, as the progression to complete heart block may not be dependent upon the H-V interval alone but also on the site of the lesion. The present controversies in the results reported from various laboratories on the subject of the prognostic value of the H-V interval might be explained on this basis.

As a fourth clinical implication, we are also confronted by the possible limitations and problems in correlating histologic findings with either an electrocardiographic pattern or even with simple recordings of His bundle electrogams.

Fifth, it may also be suggested that longitudinal dissociation within the His bundle may set a stage for reentrant tachycardias. The reentrant circuit may be completed either exclusively within the His bundle or within the His bundle in conjunction with
the bundle branches or the atrioventricular node. Tachycardias resulting from such a reentrant circuit may simulate atrial, junctional, or ventricular tachycardias.

Sixth, the demonstration of aberrant conduction due to altered refractoriness within the His bundle indicates the limitations in the determination of the refractory periods of the bundle branches during electrophysiologic studies in man.

Finally, previous studies have reported that in a significant number of patients with a right bundle branch block and left axis deviation, a prolonged H-V interval is associated with a delay within the His bundle, as indicated by prolonged or "split" His bundle potentials. In some of these cases, the occurrence of complete heart block was localized within the His bundle and was not due to a final breakdown of conduction in the posterior group of left bundle branch fibers and indicates the significance of lesions within the His bundle.

An alternative explanation for the frequent association of right bundle-branch block with left axis deviation may be suggested on the basis of these observations. If such a pattern was to result from a lesion within the His bundle, the fibers destined for both the right bundle branch and the anterior portion of the left ventricular wall are more likely to be affected, because of their proximity to each other within the His bundle (as opposed to the fibers destined for the posterior portion of the left ventricular wall).

Future studies should provide a proper perspective to the clinical spectrum of patterns of bundle branch block and arrhythmias due to discrete lesions or longitudinal dissociation within the His bundle.

Onkar S. Narula, M.D.*
North Chicago, Ill

*Division of Cardiology, Department of Medicine, Chicago Medical School, and the Veterans Administration Hospital, North Chicago, Ill.
Reprint requests: Dr. Narula, Department of Medicine, Chicago Medical School, North Chicago 60064.

REFERENCES
1 Narula OS: Longitudinal dissociation in the His bundle: Bundle branch block due to asynchronous conduction within the His bundle in man. Circulation 56:996, 1977

Not All Money Is Tainted

The place was the Americana Hotel in New York City and the time was a few months ago. The editor of the New England Journal of Medicine and I were participating in a national panel on the subject of "Authors and Editors." The audience consisted primarily of women and most of them were knowledgeable science writers. We anticipated searching questions on the respective responsibilities of investigators, referees and editorial boards, but the interrogators had a surprise in store for us. The dialog quickly centered on the subject of their major concern: the relationship between industry and scientific periodicals. Some individuals hesitantly suggested the possibility of undue influence by industry, whereas more aggressive members accused the scientific establishment of modifying editorial content in accordance with the dictates of commercialism. The candid view of this vocal group was that publication budgets were critically dependent upon advertising income and that this dependence permitted the intrusion of a "lobby" of industrial representatives who made their wishes known to editorial boards.

The specific issue which elicited most concern was the possibility that side effects of hormonal agents were under-reported because of the presumed reluctance of editors to accept papers which described untoward effects of newly marketed drugs. Thus, we were accused of condoning an editorial conspiracy of silence. Dr. Relman and I firmly refuted the implications that we were responsive to commercial influence. We had not experienced this type of pressure and could recount no instance when an attempt had been made to affect the editorial policy of the scientific peer review journals we represented.

The possibility of commercial influence on editorial content could not exist if advertising were not a major source of support for scientific periodicals. However, advertising is useful to readers and it is an entirely legitimate form of financial support; indeed without such assistance, the price of most periodicals would become forbiddingly high. Moreover, unwarranted influence is not a realized threat, possibly because it is in industry's own economic self-interest to have an unencumbered editorial community. I have noted that, "Manufacturers recognize that they benefit from honest criticism. Peer review provides guidance that often prevents fruitless study of compounds or devices that eventually prove to be either harmful or ineffective. It is the guarantee of impartiality which ultimately permits a fair hearing and a respectful response from the