SYMPOSIUM
Cardiac Arrhythmias
(Part 5)

Disturbances in Cardiac Conduction and their Management

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SUMMARY
A simple classification of atrioventricular block, criteria for diagnosis, and suggested therapy are discussed. The differences between Mobitz I and II second-degree A-V block are reviewed.

Good management of patients who have bundle-branch or other forms of intraventricular block depends on informed judgment which includes consideration of (1) the effect of intraventricular conduction disturbances on the pumping action of the heart, and (2) the likelihood that a specific intraventricular conduction disturbance will be complicated by complete heart block.

Evidence suggests that common forms of intraventricular disturbance, unlike ventricular fibrillation, do not alter significantly the pumping action of the heart. The role of intraventricular conduction disturbances in the genesis of ventricular fibrillation is assessed.

We concluded that only when right bundle-branch block is combined with block of the anterior or posterior fascicle of the left bundle branch is complete heart block sufficiently imminent to warrant special concern. If bilateral block is associated with symptoms of episodic severe brady-cardia, pacemaker therapy is indicated.

Additional Indexing Words:
Cardiac conduction disturbances
Fascicular block
Atrioventricular block
Bilateral bundle-branch block
Pacemaker therapy
Prognosis in intraventricular block

VENTRICULAR ECF

T HIS PRESENTATION will be divided into two parts: (1) atrioventricular (A-V) conduction disturbances and (2) bundle-branch block and intraventricular block.

Atrioventricular Conduction Disturbances and their Management
Occasional semantic problems have arisen over the use of the terms “A-V dissociation” and “A-V block.” “A-V dissociation, with or without ventricular captures,” is regarded as a physiologic disturbance of impulse formation. The resulting ECG pattern depends on the existence of at least two independent rhythms—one in the atria and the other in tissues at or below the A-V junction. The arrhythmia may be caused by slowing of the upper or atrial rhythm accompanied by an escaping junctional or subjunctional rhythm, or it may result from acceleration of the lower rhythm to a rate more rapid than the upper or atrial rhythm. In either case, retrograde A-V conduction must be absent, or a single functional rhythm controlling both atria and ventricles would result. This does not imply that A-V conduction is impaired. In fact, in this arrhythmia, A-V conduction is usually normal. Any lack of conduction in this arrhythmia is due to ordinary refractoriness in the junctional tissues or below, which is induced by penetration of excitation from two competing pacemakers. There is no reason why abnormal A-V conduction or “A-V block” cannot coexist with this physiologic phenomenon.

The presence of A-V block indicates abnormal A-V conduction. Definition of the various degrees of A-V block will be integral to selected comments on the clinical import of these arrhythmias, as well as to comments on a rational approach to management. More detailed comments on the indications for various types of pacemakers to be used in selected clinical situations are made elsewhere in this symposium.
First-Degree A-V Block

First-degree A-V block may be defined as a P-R interval of 0.21 sec or more, without any dropped beats or failures of the atrial impulse to traverse the junctional tissues to capture the ventricles. Clinically, it is encountered with inflammatory afflictions of the A-V junctional tissue, most notably acute rheumatic fever. More commonly it reflects the direct toxic or excessive vagotonic effect of certain drugs on the A-V junctional tissue, the most pronounced being digitalis excess. Therapy of first-degree A-V block in these circumstances is addressed to the underlying cause. The third common clinical presentation of first-degree A-V block is in acute myocardial infarction, usually of the inferior wall. The presence of first-degree A-V block merits observation for signs of progression to higher degrees of A-V block. Therapy is unnecessary but atropine or isoproterenol (Isuprel) may be effective.

Second-Degree A-V Block

Second-degree A-V block requires the presence of both conducted beats and dropped beats. One form of second-degree A-V block was initially described by Wenckebach from his analysis of jugular phlebograms. He described progressive a-c prolongation, with dropped beats, which corresponds to our present electrocardiographic definition of progressive P-R prolongation leading to dropped beats. He also noted that the P-R increments through the cycle tended to decrease. At a constant atrial rate, a progressive R-R shortening is often noted in a Wenckebach cycle. Subsequent to the widespread application of electrocardiography, Mobitz classified this form of second-degree A-V block as type I. However, present-day use of the Wenckebach eponym is widespread.

Mobitz I

While the advent of His bundle recordings has permitted accumulation of evidence that Mobitz I A-V block can be at the level of the peripheral conduction system, the block almost always is at the junctional level.

The electrocardiogram of Mobitz I A-V block may display any number of conducted beats before a dropped beat, but more than five successive conducted atrial impulses are uncommon. Commonly the ratio of P waves to QRS complexes may shift from 5:4 to 4:3 to 3:2 or back, in an unpredictable fashion. Occasionally, the ratio may change cyclically with the variations in vagal tone related to the respiratory cycle. The ratio may be 2:1, and, if stable, this creates a differential diagnostic problem to be discussed separately.

Unless there are concomitant, unrelated conduction disturbances in the bundle-branch system, the QRS complexes in Mobitz I A-V block are normal in duration and are unchanged from those that appear when sinus rhythm is present. When a Mobitz I A-V block proceeds to third-degree A-V block, the escape focus is usually a junctional one, with no change in the QRS complexes and a ventricular rate of about 50 beats/min.

Mobitz I A-V block is seen clinically in the same situations as first-degree A-V block, and usually can be regarded as an extension or aggravation of the underlying causes of first-degree A-V block. When encountered as a consequence of administration of digitalis in excess, the arrhythmia should be regarded as a sign of clinically significant intoxication, and the drug should be withheld. If the intoxication is severe and the degree of Mobitz I A-V block is increased to 2:1, the resulting bradycardia may require temporary transvenous pacing, especially if a long-acting form of digitalis has been administered. Mobitz I A-V block in acute myocardial infarction is transient and usually responds to atropine. Insertion of a pacing catheter is usually unnecessary unless there is progression to a higher degree of A-V block.

Mobitz II

Mobitz II second-degree A-V block, so classified in 1928, has justifiably generated a great deal of interest—justifiably in that the diagnosis of such a form of A-V block carries serious prognostic and therapeutic implications. The arrhythmia is defined as a form of heart block wherein dropped beats occur, but the P-R interval of all conducted beats is constant, as opposed to the Mobitz I form. While this arrhythmia occasionally presents as long runs of normally conducted beats with an occasional dropped beat, and sometimes as 3:1 or 4:1 A-V block, the most common presentation is that of 2:1 block.

In Mobitz II A-V block, the conduction defect lies below the A-V node. In some instances block has been found to be at the His bundle level, but in most cases the underlying conduction defect includes some degree of bilateral bundle-branch block. Mobitz II A-V block usually is seen in older hearts and in most instances is believed to result from idiopathic fibrosis of the cardiac skeleton.
We have noted a tendency on the part of students and house staff to regard all 2:1 A-V block as being of the Mobitz II type. This is not always the case. Delineation of the underlying mechanism of the 2:1 block usually can be accomplished with simple electrocardiographic technics and observations. If solution of the problem remains elusive, His bundle recordings may clarify the situation.

One of the striking features of Mobitz II A-V block, in its common 2:1 presentation, is its rate dependency. For instance, at a heart rate of 60 beats/min a patient may have 2:1 A-V conduction. The same patient, at a heart rate of 58 beats/min, may have 1:1 conduction. Thus, the conduction disturbance may be seen to come and go spontaneously, depending on the heart rate. Figure 1 demonstrates such rate dependency. Induced changes in heart rate may provoke comparable changes in A-V conduction. When 2:1 A-V block is present, a paradoxic response to carotid sinus massage may occur; that is, as the atrial rate slows, conduction converts from 2:1 to 1:1, with doubling of the ventricular rate. It is this paradoxical increase in ventricular rate in the Mobitz II form that may distinguish it from Mobitz I A-V block. Carotid massage in Mobitz I A-V block usually increases the degree of A-V block, slowing the ventricular rate. Carotid massage in such circumstances is not without hazard, since the involved carotid arteries are usually in elderly patients. Also, if the block is of the Mobitz I type, severe bradycardia can occur. It is safer to rely on monitored information, especially during sleep when spontaneous slowing of the atrial rate may allow 1:1 A-V conduction.

A history compatible with symptomatic bradycardia, in the presence of 1:1 A-V conduction and QRS complexes displaying features of bilateral bundle-branch block, suggests Mobitz II A-V block. The response to an increased atrial rate, with exercise or atrial pacing, should be observed. The development of the patient’s symptoms and 2:1 A-V block at an atrial rate likely to be achieved spontaneously in the patient’s daily life is an indication for a permanent demand pacemaker.

A Mobitz II A-V block can have a normal QRS if the conduction defect is in the His bundle or if a bilateral bundle-branch defect is precisely balanced. Moreover, a coincidental Mobitz I type block may develop in a patient with bilateral bundle-branch block type QRS complexes. It is in such situations that His recordings are of significant clinical value.

**Advanced Second-Degree A-V Block**

This final category of second-degree A-V block simulates complete heart block, but there are occasional conducted beats that are not arranged in orderly Mobitz I or II patterns. Advanced A-V block is prognostically indistinguishable from complete heart block and, therefore, is managed as such therapeutically.

**Third-Degree A-V Block**

Until recent times, a general account of heart block would be devoted largely to discussion of various aspects of complete or third-degree heart block. Since this condition is now so well documented, only brief comments follow. Third-degree heart block may be defined as a total lack of A-V conduction. The common form, representing total bilateral bundle-branch block, usually occurs in elderly hearts and is due to a fibrosing process of the cardiac skeleton. In the usual presentation, an idioventricular escape focus exists with a rate that ranges from 25 to 40 beats/min. When symptomatic, the prognosis, with and without medical therapy, is poor when symptomatic, it is generally felt that permanent cardiac pacing is indicated. Whether to institute permanent cardiac pacing for asymptomatic complete heart block remains controversial, but this is rare.

The less common types of complete heart block are expressions of block at a level above the His
bundle bifurcation. These types include congenital heart block, the unusual cases of Mobitz I A-V block which progress to complete block, the isolated His bundle lesions, and some forms of surgically induced complete heart block. In these situations, the resulting escape focus is in the His bundle, or above, and the resulting ventricular rate is often in the 50 beats/min range. If the rate is fast enough, if the rhythm is stable, and if there are no symptoms referable to the bradycardia, there is no need for cardiac pacing. This is true in congenital A-V block when the rate commonly approximates 60 beats/min and when the rate increases significantly with exercise. It should be pointed out, however, that in most cases of surgically induced complete heart block ventricular rates are slow and mortality without pacing is high. Most patients require permanent pacing. Fortunately, this entity is now rare.

**Bundle-Branch Block and Intraventricular Block**

What are the implications of new and revised knowledge of bundle-branch block and intraventricular block for the clinician who desires to apply that knowledge to the clinical management of his patients? Answer to this question will be formulated under two headings: (1) the effect of intraventricular conduction disturbances on the pumping action of the heart; and (2) the prognostic implication of intraventricular conduction disturbances.

As the clinician immediately will surmise, the answers will demand discerning appraisal of rather minor physiologic changes and prognostic variations, and the deductions derived will be more comfortably applied to populations of patients than to individuals. Nonetheless, well-grounded understanding of these physiologic and prognostic nuances is essential to proper management of patients who have such conduction disturbances. In the older age ranges, many of them do. Often, the clinician’s superior understanding benefits the patient not through medications and rituals of treatment but through sparing him needless anxiety and restrictions.

**What Is the Effect of the More Common Intraventricular Conduction Disturbances on the Pumping Action of the Heart?**

The new knowledge has not changed the old answer to this question. The common forms of abnormality in spread of ventricular excitation (that is, bundle-branch and divisional blocks) are not attended by clinically significant reduction in the pumping action of the heart. However, even though the old answer prevails, the new knowledge has provided some refinements in understanding of the interrelationship between ventricular excitation and ventricular function that merit brief comment.

Corday and Irving, in 1961, reported that in animal experiments when an ectopic ventricular focus was near the base of the heart, a decrease in cardiac output and a drop in blood pressure to shock levels invariably resulted.” An apical ectopic focus did not induce such effects. They believed “... the difference in the hemodynamic response in the two types of tachycardia can be explained by a difference in the contraction wave of the ventricle.” The locus and rate of driving stimulus in these experiments represented an extreme. Subsequent experience with ventricular pacing in patients afflicted with heart block has minimized the significance of the precise course of ventricular excitation to effective ventricular function. Moreover, in patients with A-V block and severe bradycardia, improvement in cardiac function has ensued when normal cardiac rate was restored by ventricular pacing, regardless of the site of the pacing electrodes.

These several observations support judgment that the hemodynamic consequences of aberrant ventricular excitation are minor so long as that excitation produces a beating—not a writhing—heart. How secure is such coordinate myocardial contraction against the threat of fibrillatory-inducing ventricular excitation? The experimental studies of Pruitt and Watt led them to conclude that:

"So long as the ventricular myocardium retained an approximation of physiologic and morphologic integrity, its excitation time could be little more than doubled by any form of damage to the high-speed conduction pathways of the subendocardium. Only in a specifically designed ventricular muscle strip on the one hand, and under conditions of severe regional ischemia of the ventricular wall on the other, was ventricular excitation time extended to a degree highly conducive to reentry of excitation and ventricular fibrillation. The singular difficulty of inducing this circumstance by simple morphologic damage has evident biologic significance. Upon it depends the preservation of coordinated ventricular activity. Once excitation is delivered to one responsive [ventricular] fiber, spread can proceed to the remainder of the myocardium over an almost infinite variety of pathways, and all this within an interval [little if any] greater than that required for ventricular excitation in left bundle-branch block.

Occlusive coronary disease can produce myocardial ischemia attended by abnormal depolarization and repolarization of the myocardial fibers; it can also produce myocardial necrosis and scarring with disruption of orderly spread of the excitation process. These
two, myocardial ischemia and [a profoundly] disorganized spread of the excitation process, were the two experimentally induced circumstances productive of QRS aberration of a degree likely to presage reentry and ventricular fibrillation.

From these studies, then, two deductions may be made. First, that the common types of intraventricular conduction disturbances such as bundle-branch and fascicular block do not seriously affect the pumping action of the heart, nor do they in themselves set the stage for ventricular fibrillation. Second, the induction of ventricular fibrillation depends on an interplay among structural, metabolic, and pharmacologic abnormalities, which affect the myocardial fibers themselves, and not on derangements specific and limited to the specialized conduction fibers of the bundle branches and the terminal fascicular network.

**What are the Prognostic Implications of Intraventricular Conduction Disturbances?**

We shall confine our comments on the prognostic implications of intraventricular conduction disturbances to those affecting components of the system below the level of the common A-V bundle and above the level of the ordinary myocardial fibers. The argument supporting such limitation was as follows: block of the A-V bundle produces heart block. A profound disturbance of conduction through the ordinary myocardial fibers is a prelude to ventricular tachyarrhythmias. In either instance, the prognosis is reasonably evident and sufficiently grave that indicated therapeutic measures should be applied promptly. These therapeutic measures are dealt with elsewhere in this symposium.

Simultaneous and complete block of right and left bundle branches produces complete third-degree A-V block, as considered in the first part of this review.

Components of the A-V conduction system included in this discussion of prognostic implications are, then, those that compose the subjunctional portion of the system, exclusive of the branching portion of the A-V bundle. These are the anterior fascicle of the left bundle branch (LBB), the posterior fascicle of the LBB, the right bundle branch (RBB), and the terminal fascicular (Purkinje) network.

**Prognostic Implications of Block of One or More Bundle Branches**

We have concluded in the preceding discussion that intraventricular conduction disturbances other than ventricular fibrillation have not been observed to produce remarkable alteration in the pumping action of the heart. Hence, in assessment of prognosis in such conduction disturbances, our comments shall concern other factors. Two of these factors are (1) that the conduction disturbance is caused by a specific and identifiable cardiac disease of such kind that the prognosis essentially is that of this underlying disease, and (2) that the conduction disturbance has an established relationship to total block of A-V conduction, in which event the prognosis derives from the relative immediacy or remoteness of the supervening heart block.

Accumulated experience seems to indicate that so long as block affects the right bundle alone, the left bundle alone, or the two subdivisions of the left bundle alone, prognosis is likely to depend on the first of the two considerations just defined, namely, the prognosis of the disease causing the block. Only when there is evidence of permanent block in the right bundle branch combined with block in one of the two divisions of the left branch does the imminence of complete heart block become sufficient to encourage effort to prognosticate its occurrence. This distinction regarding prognosis will be illustrated in the sections that follow.

**Right Bundle-Branch Block (RBBB).** The clinician is likely to feel rather secure about prognostication in block confined to the RBB. Either there is other and substantial evidence of cardiac disease imposing stress on the right ventricle or there is no evidence whatsoever of cardiac disease. Right bundle-branch block as an isolated phenomenon carries a benign import. Johnson, Averill, and Lamb observed that, “Complete right bundle branch block could not be correlated with an increase in clinical factors thought to be associated with an increased incidence of coronary artery disease.” These same investigators reported that among 67,375 asymptomatic individuals, the incidence of RBBB was 1.5 cases/1000 persons less than 40 years of age and 2.9 cases/1000 persons more than 40 years of age. In this same population, the incidence of LBBB was 0.09 case/1000 persons less than 40 years of age and 0.36 case/1000 persons more than 40 years of age. The morphologic basis for the vulnerability of the right branch to incidental block is almost certainly its comparatively long undivided course as a thin strand of tissue (fig. 2).

**Left Bundle-Branch Block (LBBB).** The electrocardiogram of LBBB results either from block of the undivided left branch or from simultaneous block of both its anterior and posterior fascicles.
This nonspecificity of origin in even an idealized construct of the left branch system is magnified by the considerable anatomic variation to which that system is subject. Moreover, the left branch system is vulnerable to any one of the many different pathologic processes which afflict the left ventricular subendocardium.

It is self-evident that prognostic simplicity cannot be based on an electrocardiogram so variously derived and affected. The time-honored dictum that prognosis in the left-branch block is best defined by the nature of coincident left ventricular disease goes unrefuted but retains only limited clinical value. In some patients, left-branch block is a prelude to complete heart block, but the incidence of this complication must be low as compared with the incidence of left-branch block itself. Left bundle-branch block occurs so rarely among young adults without other evidence of heart disease that its presence in a youthful patient is a compelling indication to proceed with careful search for substantiating evidence of such disease. Such search reasonably stops short of “invasive technics,” all else being negative. Annual reexamination of the patient is justified.

Left Anterior Fascicular Block (LAFB). In this condition the issue of prognostic import has been much debated. The relationship between LAFB and left-axis deviation (LAD) is not simple. Rosenbaum believes “... any axis shift beyond −45°, with a few exceptions, is due to LAFB” and has a significance similar to that currently ascribed to LBBB. The implication of axis positioning between 0 and −45° has provoked study and discussion for decades. Certain observations worthy of record are derived from these studies:

1. Blackburn, Vasquez, and Keys observed that with increasing age a progressive shift to the left of mean frontal-plane QRS axis developed in men. They concluded, “... that electrocardiographic age trends in healthy men provide neither a sensitive index of latent coronary heart disease in a population nor of population differences in coronary artery disease.”

2. Blackburn, Taylor, and Keys stated that “A number of claims have been made of the serious prognostic import of left-axis deviation [that is, −30° or beyond], but these are largely based on
clinical groups and have failed to consider associated variables. The evidence is only suggestive among these CHD-free cohorts, derived from total populations of working men, that extreme left axis is prognostically important."

3. Ostrander concluded that, "The age-adjusted mortality rate for persons with isolated left axis deviation was no greater than the rate for the total Tecumseh population." He does note that left-axis deviation may be a more important sign of latent coronary disease among younger persons (that is, less than 40 years of age).

4. The studies in which a high correlation was found between left-axis deviation and organic heart disease commonly dealt with the dead or the diseased.27-30

5. In these latter studies, if myocardial infarction and left-axis deviation coexisted, other electrocardiographic abnormalities more specific to infarction usually were present. If left-axis deviation was the sole electrocardiographic abnormality, coexisting myocardial infarct was uncommon.

In summarizing these five points, we conclude that left-axis deviation is a perilous sole criterion on which to base a diagnosis of myocardial infarction, and is, as an isolated clinical finding in older patients, of comparatively benign prognostic import.

Left Posterior Fascicular Block (LPFB). Such block has been produced in canine31 and in primate32 hearts and its electrocardiographic consequences have been identified in records made before and after production of the lesion. These consequences do not place the electrocardiogram clearly beyond the range of normal. A reliable diagnosis of PFB usually can be made only when single or sequential electrocardiograms on an individual patient display complexes recorded while the posterior fascicle was blocked and others recorded while it was conducting. Rarely is such evidence available. When it is, the prognosis becomes not that of stable posterior-division block but that of unstable function in the A-V conduction system. Prognosis under such circumstances is certainly more serious than that of stable anterior fascicular block. This more serious import is magnified by the anatomic peculiarities of the left posterior fascicle (see subsequent discussion of RBBB combined with LPFB) and probably is comparable to that of block involving more than a single branch of the subfunctional system.

RBBB Combined with either LAFB or LPFB. When block of the right bundle branch is combined with block of either the anterior or the posterior fasciculus of the left branch, prognostic implications specific to the conduction disturbance itself can be asserted. Accumulated data support judgment that such combined block bears a relationship to complete heart block sufficiently close to merit special surveillance of patients whose electrocardiograms support its presence.

Study of the relationship between combined block and complete heart block may be undertaken either when the conduction defect is at the combined stage or when it is at the stage of complete heart block. When RBBB is combined with left anterior fascicular block, substantial data can be obtained by either approach. This is so because the electrocardiographic identity of this combined lesion is sufficiently reliable that cases readily can be garnered from unselected series of records. Right bundle-branch block combined with left posterior fascicular block is not so distinctive electrocardiographically and probably occurs much less frequently than RBBB plus LAFB. Explanation for this less frequent occurrence has been analyzed by Rosenbaum and associates33 and by Scanlon and associates.34 Two factors are common to the comments of both these groups. First, a lesion of lesser size is required to block simultaneously the RBB and LAF than to block simultaneously the RBB and LPF. Second, the RBB and LAF are both supplied by the left anterior descending coronary artery whereas the LPF is supplied by the right coronary or circumflex branch of the left coronary artery, or sometimes both. "Because of the more diffuse nature of its blood supply compared to that of the superior radiation, the inferior radiation would be ischemic less often. Also, because of their different sources of blood supply, the right bundle and the inferior radiation would be less often diseased concomitantly."34

To these two considerations, a third may be added. Although the anatomic relationships of the common bundle, RBB, LAF, and LPF are subject to considerable variation from one heart to another, the fibers which form the LPF almost always leave the common bundle before that bundle gives rise to the LAF and the RBB. Therefore, a lesion which blocked LPF and the common bundle inevitably would block conduction through LAF and RBB. The clinical and electrocardiographic expression of a lesion so placed would be complete heart block. A lesion which blocked the common bundle "downstream" to exit of even the earliest
arising fibers of LPF would produce not complete heart block but RBBB combined with LAFB. Figure 2 provides diagrammatic expression of this point.

In the study of Lasser, Haft, and Friedberg, the incidence of RBBB combined with marked left-axis deviation (presumed RBBB plus LAFB) was approximately 1% in a series of consecutive hospital electrocardiographic records, nonrepetitive with respect to an individual patient. No comparable figure on incidence of RBBB combined with LAFB can be offered. Some inference of what that incidence might be can be derived from a study reported by Scanlon, Pryor, and Blount. They extracted from their electrocardiographic files for 1958–1968, inclusive, all electrocardiograms interpreted as showing RBBB. Among these they found 209 instances of “bilateral” or combined block. One hundred forty-seven were classed as RBBB plus superior intraventricular block (SIVB) and 24 as RBBB plus inferior intraventricular block (IIVB). The remaining 38 were classed as instances of perifascicular block. These figures suggest that RBBB plus SIVB occurs about six times as often as RBBB plus IIVB.

Among Lasser and associates, 55 patients who had RBBB with severe LAD, five (9%) had third-degree heart block and seven (13%) showed first-degree heart block. In Watt and Pruitt’s series of 65 patients with RBBB and severe LAD, five (8%) had third-degree heart block and three (5%) showed second-degree block. According to Scanlon and associates, during an average follow-up period of 18.9 months, complete heart block developed in 20 (13.6%) of 147 patients with RBBB and severe LAD and in five of 24 patients (21%) classed as having RBBB plus inferior intraventricular block.

Rosenbaum reported that, “Some form of A-V block was present in 25 (83.3%) of 30 cases of RBBB with LPH (left posterior hemiblock). In 19, complete or high-grade A-V block was observed initially or developed later. Adams-Stokes seizures were recorded in 18 (60%).” Since some of these patients had complete or high-grade block initially and some did not, the percentages are not strictly comparable either to results of the kind cited in the preceding paragraph, which began with combined block as the basis for inclusion in the series, or to results in the series now to be cited, which began with complete or high-grade A-V block as the criterion for inclusion. Such variations in approach led us to abandon efforts to tabulate data as reported in these several different studies.

Lasser, Haft, and Friedberg found that RBBB combined with abnormal LAD was the “... predominant conduction abnormality during ortho-grade (antegrade) conduction in patients who have experienced transient or permanent complete heart block (50% of a series of 44 patients).” Kulbertus and Collignon’s series was composed of 76 patients with transient or permanent complete heart block. In 31 cases, an electrocardiogram had been recorded before development of complete heart block or after recovery from transient block. Sixteen instances (51%) of combined block were identified among these 31 cases, 11 being RBBB plus superior intraventricular block, and five being RBBB plus inferior intraventricular block.

A series of crude approximations which ignore variability in approach and interpretation among different investigators runs as follows: (1) 1% of electrocardiograms recorded on hospitalized patients will show RBBB and marked LAD (presumably LAFB); (2) the incidence of RBBB plus LPFB is far less (0.1%); (3) among patients whose electrocardiograms show combined block, the subsequent occurrence of complete heart block will range from 10 to 20%, depending on the duration of follow-up; and (4) among patients displaying transient or permanent complete heart block on whom electrocardiograms have been recorded during periods of supraventricular rhythm, approximately 50% will display some form of combined block.

These approximations support initiation of precautionary measures in management of a patient found to have asymptomatic combined block and a decision for pacemaker therapy for a patient on whom other clinical data are supportive of episodic high-grade or complete heart block.

**Prognostic Implication of Block of the Terminal Fascicular (Purkinje) Network**

Criteria for precise and consistent distinction between block within the terminal fascicular (Purkinje) network on the one hand and myocardial fiber block on the other have not been established. Representative of experimental studies in search of such criteria are the reports of Pruitt, Burchell, and Essex, Wennemark and associates, Wennemark and Kossmann, and Watt and Pruitt. The delay in excitation of localized regions of the left ventricular wall after destruction of subendocardial myocardium has been well displayed in both the experimental and the clinical settings. Results of these studies do not separate
clearly the consequences of destroying conduction fibers from those of destroying myocardial fibers. Failure to achieve this distinction may but imply that functional differences between such types of fibers are of as little substance as are their morphologic differences.

Prognosis in terminal fascicular block cannot be distinguished, then, from prognosis in myocardial block. Commonly, one may surmise, terminal fascicular block and myocardial fiber block are coincident products of the same disease. Such combination in severe degree and diffusely distributed through the ventricles would lead rapidly to ventricular arrest or ventricular fibrillation. When confined to a relatively small segment of the myocardium, as in postinfarction block, the prognosis is essentially that of the disease causing the destructive myocardial lesion.

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