The Mechanism of Intermittent Bundle Branch Block: Relationship to Prolonged Recovery, Hypopolarization and Spontaneous Diastolic Depolarization*


Fourteen cases of intermittent bundle branch block (BBB) are reported, in which block was shown to occur both at rapid and slow rates, with an intermediate normal conduction range. Tachycardia-dependent BBB was related to prolonged recovery and was termed “phase-3 block;” bradycardia-dependent BBB was related to hypopolarization plus spontaneous diastolic depolarization (SDD), and was termed “phase-4 block.” Two critical rates do exist; one, separating phase-3 block from normal conduction; the other, separating normal conduction from phase-4 block. The range of normal conduction seems to be inversely related to the amount of injury. It may be extremely narrow when recovery is greatly prolonged, and/or when hypopolarization is severe, and/or when SDD is enhanced. This may simulate supernormality. In other patients, or in the same patient, the normal conduction range may widen up to several seconds, at the expense of both phase-3 and phase-4 block (accordion-like effect). Deeply inverted T waves correlated well with phase-3 and phase-4 block. Bayley’s theory of conventional myocardial injury applies remarkably well to the analysis of injury of the intraventricular conducting fascicles. The only difference is that SDD plays an important additional role in the conduction system.

Intermittent bundle branch block (BBB) is commonly attributed to a prolongation of recovery in one of the bundle branches. Under this assumption, the general mechanism determining normal or abnormal conduction is relatively simple. Above a particular critical rate, impulses reach the affected fascicle during the abnormally prolonged refractory period, causing slowed conduction or block. Below the critical rate, impulses reach the damaged region after recovery is completed and intraventricular conduction is normal. Most cases of intermittent BBB seem to depend on this mechanism, in such a way that the conduction disturbance becomes apparent only when the heart rate is sufficiently rapid.

However, “paradoxical” cases are known to occur where BBB appears only or also after long diastolic intervals or when the heart rate is slow. Apparently, paradoxical BBB is uncommon, and different mechanisms have been invoked, such as vagal effects, supernormality, concealed conduction, and hypoxia or stretching of the conducting tissue during the long diastolic period.
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Recently, it has been assumed that "bradycardia-dependent" BBB may be related to spontaneous diastolic depolarization (SDD). In this paper, it will be shown that paradoxical BBB is much more common than previously thought; that it seems to be related to hypopolarization of the involved fascicle in the presence of SDD; that it is almost invariably associated with BBB which also occurs at rapid rates; and that the underlying mechanisms are probably present in most cases of so-called intermittent BBB.

**Material and Methods**

Over the last years, a deliberate search for paradoxical BBB was made on a large series of patients showing intermittent BBB. Unilateral or bilateral carotid sinus stimulation was performed in every patient. Thus, 14 cases of paradoxical BBB were collected. Of these, left (L) BBB was observed in 12, right (R) BBB in one, and left anterior hemiblock (LAH) in one. Figure 1 indicates the range of diastolic intervals at which BBB or normal conduction was observed. In most cases, the study was repeated several times. To construct Figure 1, however, the most illustrative tracing for each particular case was chosen. All beats analyzed were of sinoatrial origin. For the late beats, a ventricular escape was ruled out for three reasons: (a) these were always preceded by a P wave with a reasonable and practically constant P-R interval; (b) the coupling of the late beats was highly variable; (c) the BBB pattern was always identical to that occurring in the same patient during early beats. True escape beats occurred only in cases 12 and 13; these will be reported separately.

The 14 patients showed definite evidence of heart involvement. Coronary artery disease, revealed by typical angina pectoris in addition to significant electrocardiographic changes, occurred in 12 cases; cardiomyopathy of unknown nature was observed in a young woman; and cardiomyopathy secondary to muscular dystrophy was found in a young man.

Anteroseptal ischemia, with deeply inverted T waves in leads V1 to V4 was observed in eight patients. In the discussion, this will be related to the electrophysiologic mechanisms underlying intermittent BBB.

**Results**

Figure 1 summarizes the main findings. Case 1 is described in greater detail later.

In all cases (except case 8), BBB was observed both at the shortest and the longest diastolic intervals, with an intermediate range of variable width from case to case at which intraventricular conduction was normal. Thus, two critical rates were present: one, above which BBB was observed; another one, below where the same BBB pattern was again observed. These two critical rates (or critical R-R intervals) separated three different conduction ranges. We shall refer to these as: (1) the "phase-3 block" range; (2) the "normal conduction" range; and (3) the "phase-4 block" range. The reasons for these names are discussed later.

In general, the critical R-R interval for phase-3

![Figure 1. Fourteen cases of intermittent bundle branch block, as related to cardiac cycle length.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20938/)
block was around 1.00 sec (Fig 1). If normal recovery in the bundle branches can be estimated to last no longer than 0.40 to 0.45 sec (according to rate), it may be said that recovery was prolonged in the affected bundle branches by an average of 0.50 to 0.60 sec, with a total duration of recovery or "Q-T interval of the bundle branches" of about 1.00 sec. On the other hand, the critical R-R interval for phase-4 block was much more variable. In general, however, it ranged between 1.22 and 1.66 sec, except for cases 1, 8 and 14, in which it was respectively 4.10, 3.20 and 5.60 sec. It will be seen later that this critical R-R interval for phase-4 block may be, under certain conditions, as long as 6.00 to 8.00 sec (Fig 7 and 8).

An "accordion-like effect" is descriptive of the fact that the two block ranges seem to "compress" the intermediate normal conduction range. (This accordion-like effect has been referred to in Argentina as the "bellows-like effect," or as the "Troilo effect" of intermittent BBB.) In some cases, the accordion seems to be nearly closing (cases 2, 3 and 12), whereas in some others, it seems to be extremely open (cases 1, 8 and 14). This accordion-like effect is even more remarkable when the same patient is studied at different stages of the underlying clinical process. Moreover, the closing or opening of the accordion seems to correlate surprisingly well with the polarity of the T waves in the corresponding conventional ECG. This will become clearer by analyzing the following case.

Case 1

A 57-year-old man was known to have angina pectoris for the last four years. On April 6, 1968, an ECG showed intermittent LBBB and anteroseptal ischemia. Figure 2 was prepared in such a way that normally conducted beats and LBBB beats are seen together in every lead. The normally conducted beats reveal the extensive anteroseptal ischemia. The same day, a long tracing was taken during which carotid sinus massage was performed several times (Fig 3). Figure 4 and the top diagram in Figure 1 illustrate the distribution of 650 sinoatrial beats from the long tracing from which Figure 3 was prepared. LBBB was observed in beats ending R-R intervals between 0.64 and 0.92 sec; normal conduction between 0.68 and 4.40 sec; and LBBB again between 4.10 and 6.16 sec. The overlapping was wide at both ends of this distribution. No beats showing incomplete LBBB were recorded. It should also be stressed that not a single escape occurred, even after R-R intervals as long as 6.16 sec.

One month later, on May 13, 1968, another tracing (not shown) revealed that the ischemia had improved, that normal intraventricular conduction prevailed, and that LBBB could neither be obtained when the heart rate was acceler-
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FIGURE 4. Case 1. Analysis of 650 sinoatrial beats, from long tracing from which Figure 3 was prepared. Bottom, cycle length in hundredths of seconds. Left: number of beats. Full lines: LBBB. Interrupted lines: normal conduction. LBBB occurs at both ends of distribution, with in-between normal conduction range. There is some overlapping between phase-3 LBBB and normal conduction (to left), and between normal conduction and phase-4 LBBB (to right), as in bars illustrated in Figure 1.

stated up to 100 beats per minute, nor after R-R intervals as long as 3.84 sec. Therefore, there was a definite improvement in the conduction disturbance when the anteroseptal ischemia was less. This was further confirmed two months later, when a long tracing (1,220 beats) was taken, from which Figures 5 to 8 were prepared. Figure 5 shows the full conventional ECG, to illustrate that the anteroseptal ischemia had greatly improved. Figure 6 shows that conduction remained normal at a heart rate of 100, induced by inhalation of amyl nitrate. Figure 7 reveals that conduction was normal after pauses as long as 5.90 sec, and that LBBB occurred only after a pause of 6.71 sec. Figure 8 depicts schematically, below the ECG, our hypothetical interpretation of the possible electrophysiologic mechanisms, which can be summarized as follows: (1) Recovery in the LBB was assumed to be abnormally prolonged. (2) The maximum resting potential was assumed to be slightly diminished, let us say from -90 to about -80 or -75 mv. We shall refer to this as "hypopolarization," meaning that the injured fibers of the LBB are unable to attain full polarization after the end of systole. (3) The slope of SDD during phase 4 was assumed to reach critical levels for abnormal conduction at about 6.71 to 8.04 sec of the preceding beat. Thus, the partial depolarization of fibers late in diastole resulted from the initial hypopolarization, plus the effect of SDD.

Initially, the anteroseptal ischemia (and eventually the injury in the LBB) was severe (Fig 2); subsequently, the clinical condition of the patient clearly improved and the ischemia (and possibly the injury in the LBB) became less extensive. This improvement was accompanied by a definite widening of the normal conduction range, at the expense of both phase-3 and phase-4 block; namely, both towards its tachycardic (left) and bradycardic (right) end. If this is now extrapolated to the general behavior illustrated in Figure 1, it may be said that the range for normal conduction may vary from case to case, depending mostly on the degree of injury.

FIGURE 5. Case 1 (three months later, as compared to Fig 2 and 3). Intraventricular conduction is normal, and anteroseptal ischemia has greatly improved.
and perhaps also on particular conditions related to the localization and extension of the damaged region within the affected fascicle. In general, it may be said that, the greater the injury, the shorter the normal conduction range; and the lesser the injury, the wider the range for normal conduction. It may even be envisioned that, if injury is extreme, both ranges for abnormal conduction will meet, and the normal conduction range will disappear entirely. Under such conditions, block will occur for any rate of stimulation, whether fast or slow, and BBB will be permanent rather than intermittent.

The patient was restudied two years later. Intermittent LBBB was still present and anteroseptal ischemia had recurred. An ECG (not shown) revealed LBBB at R-R intervals of 0.76 sec or less, and normal conduction up to 6.00 sec. No longer diastolic intervals were obtained. In the presence of a definite prolongation of recovery, phase-4 BBB was much more difficult to obtain.

**DISCUSSION**

BBB may apparently seem permanent (or may be inapparent), unless an appropriate change in rate occurs. Whether this change occurs spontaneously or is artificially provoked, is immaterial from the physiologic standpoint. Therefore, cases in which BBB is actually or potentially intermittent are the subject under discussion. The main point, however, is that in all of these cases, conduction in the corresponding bundle branch is only partially affected: namely, there is not complete interruption of conduction. Thus defined, intermittent BBB seems to be related to four main physiologic events: prolonged recovery, hypopolarization, SDD, and a shift of the threshold potential toward zero.

**Prolonged Recovery**

For many years, prolonged recovery has been considered practically the only physiologic derangement underlying delayed conduction and block. Prolonged recovery determines that impulses which reach the conduction system at a fairly normal rate of stimulation, arrive during the absolute or relative refractory period and are thus delayed or blocked in the corresponding fascicle. This prolongation of recovery may vary between small and relatively large. Accordingly, depending on the case, different critical rates will be necessary to unveil the corresponding BBB pattern.

In the cases presented in this paper, as well as in a much larger series of unpublished observations, the Q-T interval of the affected fascicle lasted commonly between 0.70 and 1.20 sec. The critical
mechanism of intermittent bundle branch block

Figure 8. Schematic representation of electrophysiologic mechanisms underlying phase-3 and phase-4 block. In upper strip (leads V1 and aVL simultaneously recorded), the third beat shows phase-4 LBBB after pause of 8.04 sec. Bottom strip summarizes electrophysiologic properties of injured area in left bundle branch, responsible for electrocardiographic changes. Recovery is assumed to be prolonged (action potentials of increased duration). Maximum resting membrane potential is slightly diminished (10 to 15 mv less than the normal −90). Spontaneous diastolic depolarization (slowly ascending line during phase-4) is assumed to reach critical levels for abnormal conduction quite late in diastole. See text for further description.

rates for phase-3 block were thus between 85 and 50 beats per minute. This prolongation of recovery implies prolongation in the duration of the action potential of the involved fibers, at the expense of phase 2, phase 3, or both.

When block is complete or stable for any rate of stimulation, it has been assumed that recovery is prolonged to infinity. This is definitely a misconception. Action potentials cannot last indefinitely and, when block is permanent, this most likely indicates either biologic interruption (death) of the fascicle, or hypopolarization to such a degree that the tissue becomes totally unresponsive (see below). In the same previously mentioned series of cases of intermittent BBB, recovery seldom lasted longer than 1.50 to 1.80 sec. Therefore, under pathologic conditions such as those determining the occurrence of BBB in the human being, action potentials lasting 1.80 sec must be close to the upper possible limit. From a practical standpoint, if intraventricular conduction is not normalized after diastolic intervals of 2.00 sec, it may be safely assumed that BBB is permanent or stable, at least at the time when the exploration was performed.

BBB related to prolonged recovery is usually understood as tachycardia-dependent. However, if recovery is greatly prolonged, BBB may occur with rates which are far from being rapid in the conventional sense. It was then thought preferable to designate this as “phase-3 block,” under the understanding that phase 2 may be also involved. This facilitates referring to two important, closely related, but physiologically independent types of block: phase-3 versus phase-4 block.

Hypopolarization

Perhaps the most important determinant of conduction velocity is the rate of rise of the action potential (dV/dT), which depends primarily on the level of membrane potential at the time of stimulation. In general, conduction will be more and more deteriorated as the membrane potential is less, regardless of the cause or mechanism of lowering of the membrane potential.

The membrane potential is low during repolarization, whether normal or abnormally prolonged. This explains phase-3 block, as well as the aberrant ventricular conduction of premature supraventricular beats, which can thus be considered a physiologic variant of phase-3 block. On the other hand, the membrane potential may be low, in Purkinje fibers, after long pauses, due to SDD. However, there is still another important cause of a diminished membrane potential, which is related to the existence of injury.

Many chemical, mechanical, metabolic or inflammatory processes may injure the conducting tissue. The ability of the cellular membrane to
sustain a normal density of electrical charges is affected in a particular, but not well understood, manner. Under such conditions, the resting membrane potential is reduced, and impaired conduction and block may readily occur. A lowering of the membrane potential to about $-70$ mV (instead of the normal $-90$) is needed to cause a significant conduction disturbance.\textsuperscript{18} Further lowering will cause greater deterioration in conduction, up to complete block or to total unresponsiveness of the involved fibers. Indeed, below a certain level of membrane potential, the cell may be considered electrophysiologically dead, even if other morphologic and physiologic features are preserved. Such a situation tends to be unstable, leading either to irreversible death, or to some improvement, in such a way that the involved tissue may again become responsive, either normally or abnormally.

While prolonged recovery has been considered the main cause of block under clinical conditions,\textsuperscript{22} hypopolarization has been considered the main cause of block under experimental conditions,\textsuperscript{24} and it is remarkable that two such important mechanisms could have been so “dissociated.” Here, an attempt will be made to reconcile both views. The main reason why hypopolarization block has not so openly entered the electrocardiographic and clinical field is because it usually tends to be a rather acute and unstable phenomenon. On the other hand, block due to prolonged recovery is commonly neglected by electrophysiologists because of its being a chronic type of process, which is more readily provoked by human disease than by present experimental designs.

As mentioned before, up to a membrane potential of around $-70$ mV, conduction is fairly well preserved, and this has been related, first, to the fact that the stimulating effect is closer to the threshold potential and, second, to an increase in the space constant in relation to an increase in membrane resistance.\textsuperscript{18} If the membrane potential is further reduced, there will be a small range for incomplete BBB to occur, and beyond this, complete BBB must occur. Apparently, the conduction disturbance resulting from hypopolarization, let us say between $-70$ and $-50$ mV, should result in the same degree of BBB for any rate of stimulation. However, there are several reasons why this is not always the case. First, because the degree of hypopolarization may itself be rate dependent (see later); second, because hypopolarization block will be commonly associated with phase-3 block; and third, because Purkinje fibers, whether normal or hypopolarized, tend to show SDD and phase-4 block. Under the latter two circumstances, hypopolarization block will be associated with either phase-3 or phase-4 block, both of which are rate dependent. When hypopolarization is extreme, reaching the level of total unresponsiveness, block will be sustained and truly independent from any change in rate.

**Spontaneous Diastolic Depolarization. Shift of the Threshold Potential Toward Zero**

Although predictable from Weidmann’s work,\textsuperscript{23} Singer et al\textsuperscript{18} really incorporated SDD as an essential mechanism of abnormal conduction. They demonstrated that during phase-4 depolarization, a similar impairment of conduction occurs, in addition to that which develops during phase-3 repolarization at equivalent levels of reduced membrane potential. This introduces the possibility of conduction disturbances at slow rates of stimulation. However, some qualifications about the way this mechanism operates may now be necessary.

First of all, SDD of normal Purkinje fibers is not likely to be accompanied by conduction disturbances. Indeed, as Singer et al emphasized, an important impairment of conduction never occurred, unless SDD was accompanied by what they termed “generalized diastolic depolarization,” which is probably equivalent to what we call “hypopolarization.” This is so because, normally, the threshold potential of Purkinje fibers is around $-70$ mV and, as previously mentioned, up to this reduction in membrane potential, conduction is not likely to be severely affected. In some way, this may be said to be teleologically fortunate. If not, SDD, which is the mechanism that leads fibers to fire or discharge when needed, would become ineffective, because the conduction disturbance resulting from SDD beyond $-70$ mV would preclude the escape impulse to be propagated to the rest of the heart. This should be kept in mind when discussing later the reasons why escape beats are so uncommon in the presence of phase-4 BBB.

In addition, a shift of the threshold potential toward zero has been shown to be an indispensable requirement for SDD to give rise to a conduction defect.\textsuperscript{19} Unfortunately, little is known about the clinical circumstances which may shift the threshold potential in that direction. It seems that, at least under acute experimental conditions, such shift may occur in the presence of a normal resting potential.\textsuperscript{18} Whether the same will hold true under chronic or more stable conditions, is unknown. On the other hand, hypopolarization seems to be commonly associated with a shift of the threshold potential toward zero.

A small degree of hypopolarization, let us say a level of membrane potential between $-80$ and $-75$
mv, seems to be the most basic physiologic derangement underlying phase-4 block. Obviously, such a degree of hypopolarization will not cause block by itself. On the other hand, a much greater degree of hypopolarization will cause block for any rate of stimulation; and intermediate levels of membrane potential may perhaps cause incomplete BBB. However, if such a low degree of hypopolarization is accompanied by a shift of the threshold potential toward zero, the additional effect of SDD, whether enhanced or not, will cause a significant impairment of conduction whenever the rate is sufficiently slow. Since both hypopolarization and SDD must be present for phase-4 block to occur, it is immaterial which of the two factors is more important. However, it seems likely that hypopolarization may be the initiating mechanism in most cases. Although this type of block will almost invariably be bradycardia-dependent, we prefer to call the entire process “phase-4 block,” for previously mentioned reasons.

In all our cases, phase-4 block was associated with prolonged recovery and phase-3 block. This seems to imply a contradiction, because hypopolarization is usually associated with a shortening of the action potential duration. Two possible explanations may be considered. First, perhaps in the presence of only slight hypopolarization, the action potential duration may be increased. Some information given by Singer et al.\(^\text{18}\) may support this view: “In deteriorated fibers, after the onset of generalized diastolic depolarization, the entire voltage-time course of diastolic potential is altered and the maximum value (of resting potential) may be reached quite late in the diastolic interval.” A second possibility is that the area of fibers showing hypopolarization and a shortened action potential may be surrounded by another area of cells in which there is a prevalence of prolonged recovery with a normal resting potential. If such were the case, one population of cells would be responsible for phase-4 block, while another adjacent group of cells would be responsible for phase-3 block. This latter possibility is more in accordance with known facts of the effects of myocardial injury in general, to be discussed later.

The critical R-R interval for phase-4 block showed an extremely wide range of variation. In the circumstances in which this critical interval was extremely long (diastolic intervals of several seconds, as in cases 1, 8 and 14), the conclusion is inescapable that SDD was not significantly enhanced. Conversely, in the cases in which the critical R-R interval was much shorter, in the order of 1.10 to 1.40 sec, it is possible that SDD was truly enhanced. However, since such cases seem to correspond to situations in which injury was greater, it is also possible that a greater degree of hypopolarization could have produced the same end result in the presence of a still normal slope of SDD. Moreover, it is also conceivable that, in the presence of a critical amount of hypopolarization, even a subnormal slope of SDD could determine the occurrence of block, provided the threshold potential is significantly shifted toward zero.

The latter view is favored by the remarkable feature that, in only two of the 14 cases were escape beats recorded during carotid sinus stimulation. Actually, the absence of escape beats allowed the extremely long pauses which so well unveiled phase-4 block. The main reason for the absence of escape beats is that the threshold potential is shifted toward zero. However, it seems unlikely that this alone could have precluded the occurrence of escape beats in the presence of truly and significantly enhanced SDD. Therefore, two other possibilities may be considered. One, that SDD may reach some particular level, and then remain stable (or even decline). The second possibility is that escape beats perhaps do occur, but are not conducted beyond the area of injury, because of the blocking effect of SDD on the neighboring fibers. In this case, the most injured area would be depolarized and the process of SDD would start anew from the “concealed escape.” It should be mentioned that when intermittent BBB related to SDD was reproduced experimentally on the canine heart (unpublished observations), true escape beats were much more common.

**The Overlapping of Normal Conduction with Both Phase-3 and Phase-4 block**

In phase-3 block, it is known that the critical rate will be different whether the heart rate is slowly increased or slowly decreased. When the heart rate is slowly increased or slowly decreased, the adaptation of the duration of recovery in the affected fascicle is such that the critical rate for phase-3 block will be higher when the rate increases and lower when it decreases (Fig 9A). In addition, when the critical rate is reached and the first beat showing BBB appears, retrograde activation of the affected fascicle (coming from the contralateral ventricle) tends to maintain the same pattern of ventricular activation even if the heart rate is subsequently slowed down, at least within certain limits. In other words, the critical rate will again be higher when the heart rate is increased (to reach the first BBB beat), and lower when it is decreased (to reach the first unblocked beat). This latter
process has recently again been described under the name of “linking.” It is obvious that the “linking” and the “sensitivity to gradual change in rate” effects are additive, regarding the establishment of different critical rates for phase-3 block.

Perhaps a more pertinent cause of overlapping for phase-3 block (at the left end of the curve) is the sensitivity of the duration of recovery to sudden changes in rate. If the heart rate decelerates suddenly after a relatively stable rapid rate (which implies a relatively shorter recovery time), it is understandable that normalization of conduction may occur after a relatively shorter R-R interval (or at a relatively higher rate); whereas, when the heart rate is suddenly accelerated after a preceding period of slow rate (which implies a relatively longer recovery time), BBB will tend to occur after R-R intervals relatively longer (or at a slower rate (Fig 9B)). In our cases, indeed, most of the normally conducted beats were obtained when the heart rate was decelerated rather suddenly by vagal stimulation; and most of the beats showing phase-3 BBB were obtained when the heart rate was accelerated (when the effects of a previous vagal stimulation were subsiding, or by the effects of atropine sulphate, amyl nitrate, or exercise), or when the heart rate was spontaneously high and rather constant. Although the two mechanisms depicted in Figure 9 A and B, tend to balance each other, it is still understandable that the overlapping at the left end of the curve could have been so wide and unpredictable. Actually, this overlapping suggests that the duration of recovery in the affected fascicle was highly sensitive to changes in rate and supports previous observations that, when recovery is abnormally prolonged, it becomes much more sensitive to sudden changes in rate.

The overlapping for phase-4 block (at the right end of the curve) may be, at least in part, secondary to the alteration in the duration of recovery during changes in rate. If for a particular beat recovery is extremely prolonged, SDD will start later, as compared to another beat with a much shorter recovery period. Therefore, phase-4 block will occur relatively earlier if a long R-R interval follows suddenly after a rapid and stable rate, and relatively later if the long R-R interval is preceded by several other long R-R intervals (Fig 9C). However, a more direct mechanism may also be implied. SDD depends, among other factors, on the rate of stimulation, in such a way that the slope is steeper at slower rates and flatter at rapid rates.

Therefore, if a long pause occurs suddenly after a relatively rapid and stable rate, phase-4 block will occur relatively later than if the long pause occurs after several other long R-R intervals (Fig 9D). It may be seen that the two mechanisms just described tend to counteract each other. The prevailing effect will depend on what is more sensitive to changes in rate: the slope of SDD, or the duration of recovery.

The possibility of concealed escapes as another cause for overlapping at the right end of the curve should also be considered. Finally, another poten-
tial, although rather unexplored factor, could be a rate-related change in the degree of hypopolarization, since at least under experimental conditions, a given amount of hypopolarization can be diminished by an increase in the rate of stimulation.

The above listing and summarized discussion of the several factors which may cause temporal overlapping between normal conduction and BBB, clearly indicates that there is no such a thing as a fixed critical rate (or critical diastolic interval), neither for phase-3 nor for phase-4 block. The critical rate, both for phase-3 and phase-4 block, actually covers a range of variable width from case to case, according to the several mechanisms which may be involved. When a particular case is considered, it may become extremely difficult to determine how much of the overlapping is due to one factor and how much is due to the other factors. This will be specifically analyzed in another publication.

Hypopolarization and Prolonged Recovery in Clinical Electrocardiography

There are reports that myocardial injury gives rise to three different physiologic derangements, hence, to three different types of electrocardiographic signals.26-29 The first and most severe is death of the tissue. Abnormal Q waves may be the result. Marked hypopolarization is the second. The potential difference thus established between hypopolarized and adjacent normally or better polarized areas gives rise to the well known injury currents. S-T segment elevation, as observed in the acute stage of myocardial infarction, is the most classical expression of hypopolarization. The third is prolonged recovery. This results in inverted, symmetrical, "coronary" T waves, because the entire recovery process points toward the "ischemic" area. It is also classical to assume that an area of severe injury is composed of three parts26: a central core of dead tissue, a surrounding area of hypopolarization, and a third and more peripheral area of prolonged recovery.

Although this theory of myocardial injury has been disputed, it seems still to be the one which accounts best for most of the electrocardiographic consequences of myocardial injury, at least within the clinical setting. It is therefore reasonable that the same theory should be applied to the analysis of injury of the conducting tissue. For instance, if there is prolonged recovery in the common myocardium of the upper part of the septal region, revealed by the presence of deeply inverted T waves from V₁ to V₄, it makes sense to assume that prolonged recovery may also exist in the main LBB, which is probably included in the ischemic process. Similarly, if there is hypopolarization in the same region, revealed by a slightly elevated S-T segment (or perhaps by the symmetry of the inverted T waves), hypopolarization may also be anticipated to occur in the main LBB. The only difference, but quite a significant one, is that SDD will be an additional physiologic mechanism present in the conducting tissue but not in the working myocardium. If all this is true, the same theory will be adequate to explain not only the QRS, S-T and T wave changes of conventional myocardial injury, but also the way conduction will be impaired in the corresponding bundle branch. The theory will thus be more universal and, consequently, much closer to its truth and its practical implications.

The material presented in this paper indicates that the attempted correlation proved to be remarkably good. In several cases, the presence and depth of the inverted T waves in leads V₁ to V₄ correlated well with the evidence of prolonged recovery in the LBB, or with the presence of phase-4 block. Indeed, this seems to be the first time that such correlation between conventional myocardial injury and injury within the conducting fascicles is established on a physiologic basis.

A Proposed General Mechanism of Intermittent Bundle Branch Block

Figure 10A illustrates a model of injury within a fascicle of the intraventricular conduction system, following the lines postulated by Bayley26 for the common myocardium. Obviously, if dead tissue involves the entire cross-section of the fascicle for an adequate length, complete and irreversible block must occur. This situation is probably the one which underlies most, if not all, cases of permanent and irreversible BBB. Necrosis dominates the picture, and the manifestations of hypopolarization and prolonged recovery become irrelevant.

In the model illustrated in Figure 10B, hypopolarization and prolonged recovery are the two main components. If hypopolarization is extreme, the tissue will be unresponsive. Complete BBB will occur and will be irreversible, unless the hypopolarization recedes, which is not unlikely under acute conditions. This is probably the basis for transient BBB during the course of an acute myocardial infarction. Hypopolarization dominates the picture and block must occur for any rate of stimulation. Prolonged recovery is still irrelevant.

The model in Figure 10C is the one which best explains the observations presented in this paper. Hypopolarization is slight, with a maximum resting potential between -80 and -70 mv. Prolonged
recovery is important, with a maximum duration of the action potential of the involved fibers around 1.00 sec. Under such conditions, phase-3 BBB will occur at rapid rates, phase-4 BBB at slow rates due to the additional effect of SDD, and a normal conduction range will be present at intermediate rates. The width of the normal conduction range will vary from case to case and even in the same case from day to day, according to the degree and evolutionary stage of the injury.

It is possible that prolonged recovery may exist alone, without any hypopolarization, as in the model illustrated in Figure 10D. If such is the case, BBB will only occur at fast rates: phase-3 BBB without phase-4 BBB will occur. However, it is difficult to be sure about this possibility, because most cases of this kind are seldom explored for sufficiently long diastolic intervals. Since the critical R-R interval for phase-4 block may range from 1.50 to 8.00 sec, it may be extremely difficult to demonstrate (or to exclude) the phase-4 component of intermittent BBB.

Since both prolonged recovery and hypopolarization are rather unstable processes, changes may be expected in the critical rate for both phase-3 and phase-4 block, according to the clinical course of events. This was confirmed in all the cases, when exploration was repeated after a long time. Moreover, a possible evolving sequence of injury of the intraventricular conducting fascicles was predicted and favorably tested on the canine heart (unpublished observations).

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Pseudomineral

Amber, succinite of mineralogists, so much used for personal ornament, is not, strictly speaking, a mineral at all, being of vegetable origin, and consisting of the more or less considerably altered resin of extinct trees. It resembles minerals in its occurrence in the beds of earth's crust. Amber consists essentially of carbon, hydrogen and oxygen. Chemical formula: C_10H_18O. Perfectly clear and transparent specimens contain 3-4 percent succinic acid; cloudy specimens contain more and in the frothy amber there may be as much as 8 percent. It is amorphous and shows no indication of crystalline structure. When rubbed on cloth, amber becomes strongly charged with negative electricity and it is, indeed, from the ancient name for amber, electron, that the word electricity is derived. Amber ranges from perfect transparency to complete opacity. The turbidity is due to the enclosure of a number of bubbles. Nearly one-half of the total production of amber is devoted to the manufacture of cigar- and cigarette-holders and for pipes. The commonest ornamental articles are beads, perforated so that they can be strung together and worn as necklaces and bracelets, or used as rosaries.

Bauer M: Precious Stones (translated from German by Spencer LJ). Rutland, Vermont, C E Tuttle, 1969

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