Phase-3 and Phase-4 Intermittent Left Anterior Hemiblock

Report of First Case in the Literature*

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A 33-year-old man developed left anterior hemiblock (LAH), which was shown to occur both at rapid and slow rates, with an intermediate normal conduction range. LAH occurred at R-R intervals of 1.10 second or less; normal conduction between 1.10 and 1.60 second; and LAH again from 1.60 onwards. The tachycardia-dependent LAH was related to a prolongation of recovery in the anterior division of the left bundle branch and was termed “phase-3 block;” the bradycardia-dependent LAH was related to hypopolarization plus spontaneous diastolic depolarization and was termed “phase-4 block.” Progressively decreasing degrees of LAH were observed at the transition between phase-3 block and normal conduction; and progressively increasing degrees of LAH occurred at the much wider transition between normal conduction and phase-4 block.

Paroxysmal or bradycardia-dependent bundle branch block (BBB) refers to cases in which BBB occurs only after long diastolic intervals or with a slow heart rate.1,2,17 Recently, however, it was demonstrated that intermittent BBB tends to occur both at rapid and slow rates, with an intermediate normal conduction range.18,19 The tachycardia-dependent BBB was related to a prolongation of recovery and was termed “phase-3 block;” the bradycardia-dependent BBB was related to hypopolarization plus spontaneous diastolic depolarization (SDD), and was termed “phase-4 block.” It was also demonstrated that phase-4 block is much more common than previously thought and that the underlying mechanisms are present in most cases of so-called intermittent BBB.

So far, phase-4 block has been shown to occur in clinical cases of BBB,2,18 or in peripheral Purkinje fibers in a bath preparation.20 In this paper, a case of intermittent left anterior hemiblock (LAH) is reported, in which the above mechanisms were operative at the level of the anterior division of the left bundle branch (LBB). This seems to be the first case in which both phase-3 and phase-4 block are shown to occur together in the anterior division of the LBB. In addition, different degrees of LAH related both to phase-3 and phase-4 block are documented.

CASE REPORT

A 33-year-old man with an advanced form of muscular dystrophy was known to have intermittent LAH. Actually, ECGs of this patient were previously used to demonstrate the existence of intermittent LAH21 (case 1 of that publication). From 1967 to 1969, LAH was only seen during either spontaneous or induced high rates. Figure 1 shows intermittent, tachycardia-dependent, or phase-3 LAH; and Figure 2 was prepared in such a way that, in every lead, the first beat shows normal conduction, and the second, LAH. The diagnosis of LAH was made on the basis of previously reported criteria.21-23

On July 2, 1969, a long tracing was recorded during which carotid sinus massage was performed several times. The analysis of 1,250 sinoatrial beats of this tracing revealed that LAH was present in all beats terminating R-R intervals of 0.60 to 1.10 sec; normal conduction between 1.10 and 1.60 sec, and LAH again between 1.60 and 3.60 sec (Fig 3). Figure 4 shows several strips representative of the long tracing just analyzed. Phase-3 LAH is seen after the relatively shorter R-R interval of 0.80 sec; progressively decreasing degrees of phase-3 LAH, after the R-R intervals of 1.06 and 1.10 sec; normal conduction, when the R-R intervals increase to 1.26 and 1.46 sec; and progressively increasing degrees of phase-4 LAH, when the R-R intervals are of 1.60 sec or longer, up to 3.22 sec. A similar high degree of LAH occurs both after the shortest and longest R-R intervals. Neither

For editorial comment, see page 655

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Ficure 1. Intermittent, tachycardia-dependent, or phase-3 LAH. LAH occurs only after the relatively shorter R-R intervals. R-R intervals in seconds. The two strips are continuous.

Thus, two critical rates were documented: one, above which LAH was observed; another one, below which the same LAH pattern was again present. These two critical rates, or critical R-R intervals, separated three different conduction ranges: a phase-3 block range, a normal conduction range, and a phase-4 block range. It should be stressed, however, that progressively decreasing degrees of LAH were observed at the transition between phase-3 block and normal conduction; and that progressively increasing degrees of LAH occurred as a much wider transition between normal conduction and phase-4 block (arrows in Fig. 3).

An accordion-like effect (Fig. 3) is descriptive of the fact that the two block ranges seemed to compress the normal conduction range. Indeed, nine months later the patient was restudied and, at that time, it was found that much longer pauses were needed to elicit phase-4 LAH (Fig 5). This indicated that the normal conduction range had increased, and that the accordion had opened itself, mostly at the expense of phase-4 block.

Figure 6 illustrates schematically, below the electrocardiographic curve, our interpretation of the possible electrophysiologic mechanisms. The three following aspects should be stressed:

1. Recovery in a group of cells of the anterior division of the LBB was assumed to be abnormally prolonged, to about 1.10 sec (bottom diagram). If recovery in a fascicle of this kind can be estimated to last no longer than 0.40 to 0.45 sec (according to rate), it may be said that the prolongation of recovery was in the order of 0.50 to 0.60 sec. Since incomplete degrees of LAH were recorded at the end of the phase-3 block range, it may also be assumed that both the absolute and relative refractory periods were prolonged, although the former much more than the latter.

2. The maximum resting potential was assumed to be slightly diminished, let us say from -90 to about -80 or -75 millivolts in another group of injured cells in the anterior division of the LBB (top diagram). We refer to this as "hypopolarization,"18-19 meaning that these abnormal fibers were 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 1.60 sec of the preceding beat (top diagram). At that moment and thereafter, those fibers were partially but significantly depolarized, and this resulted from the initial hypopolarization, plus the effect of SDD.

For several months, the patient exhibited the same electrocardiographic features, until he developed intermittent LBBB. This probably meant extension of the lesions either toward the posterior division of the LBB ("divisional" LBBB),22,23 or toward the main LBB. LBBB was only seen when the heart rate was rapid, and normal conduction when the rate was slowed down. Neither LBBB nor LAH were seen after extremely prolonged diastolic intervals. It was assumed that the phase-4 component of the intermittent LAH had disappeared or became much less, and that no phase-4 component was present in the additionally involved fascicle.

**Discussion**

**Mechanism of the Electrocardiographic Changes**

One of the most important determinants of conduction velocity in Purkinje fibers is the maximum rate of depolarization (phase 0 of the action potential), which depends primarily on the level of membrane potential at the time of stimulation. In general, conduction will be slower and slower, up to the point of total block, as the membrane potential
INTERMITTENT LEFT ANTERIOR HEMIBLOCK

Figure 3. LAH is related to cardiac cycle length (from the analysis of 1,250 sinoatrial beats). NC = Normal conduction. P3-LAH = Phase-3 left anterior hemiblock. P4-LAH = Phase-4 left anterior hemiblock. R-R intervals in seconds. LAH occurs both at the shortest and longest R-R intervals, with an intermediate range during which intraventricular conduction is normal (accordion-like effect). The arrows indicate the ranges for incomplete LAH, and the direction in which its degree progressively increases or decreases. Note the much wider range for incomplete LAH during phase-4 block.

is less, regardless of the cause of lowering of the membrane potential.

The membrane potential is low during repolarization, whether normal or abnormally prolonged. This explains the phase-3 LAH of our case, up to R-R intervals of 1.10 sec. Indeed, the occurrence of LAH up to this range indicated that recovery was markedly prolonged in the anterior division of the LBB.

In Purkinje fibers, the membrane potential may also be low during phase-4 (diastole) when there is hypopolarization, SDD, or both. Hypopolarization is caused by many chemical, mechanical, metabolic or inflammatory processes, which may injure the conducting tissue. In this type of injury, the ability of the cellular membrane to sustain a normal density of electric charges is affected. Thus, the resting membrane potential is reduced, and impaired conduction and block may occur. A lowering of the membrane potential to about —70 millivolts is needed to cause a significant conduction disturbance. Further lowering will cause greater deterioration of conduction, up to complete block and complete unresponsiveness of the involved fibers. In addition, Singer and associates demonstrated that, during phase-4 SDD, a similar impairment of conduction occurs to that occurring during phase-3 repolarization (at equivalent levels of reduced membrane potential). This introduced the possibility of conduction disturbances at slow rates. However, two points should be made clear. First, SDD of normal Purkinje fibers is not likely to cause conduction disturbances; and this is so because, normally, the threshold potential of Purkinje fibers is around —70 millivolts, and as mentioned before, up to this reduction in membrane potential, conduction is not greatly affected. Second, a shift of the threshold potential toward zero has been shown to be a prerequisite for SDD to give rise to a conduction defect.

Accordingly, a small degree of hypopolarization was considered to be the basic physiologic derangement underlying the phase-4 LAH. Obviously, such a degree of hypopolarization cannot cause block by itself. However, if this is accompanied by a shift of the threshold potential toward zero, the additional effect of SDD may cause an important impairment of conduction whenever the rate is sufficiently slow.

The fact that phase-4 LAH was accompanied by phase-3 LAH seems to imply a contradiction, because hypopolarization is generally associated with a shortening and not with a prolongation of the action potential duration. This was explained in the following way. An area of fibers exhibiting hypopolarization and shortened action potentials may be surrounded by or intermingled with another area of cells in which there is predominantly prolonged recovery and normal polarization (Fig 6). If such were the case, one population of cells would be responsible for phase-4 block, while another group

Figure 4. Several strips from the long tracing on which Fig. 3 was based. R-R intervals in hundredths of seconds. As the R-R interval increases, there is first phase-3 LAH (R-R interval: 0.80 sec); then, progressively decreasing degrees of LAH (R-R intervals: 1.06 and 1.10 sec); then, normal conduction (R-R intervals: 1.26 and 1.46 sec); and finally, progressively increasing degrees of LAH (R-R intervals: 1.60, 1.86, 2.12 and 3.22 sec).
of cells would be responsible for phase-3 block. This explanation is in accordance with Bayley’s old theory of conventional myocardial injury.24

Incomplete Degrees of Left Anterior Hemiblock

The fact that incomplete forms of LAH were observed both during phase-3 and phase-4 block is interesting. As stated, the incomplete forms of LAH during phase-3 block indicated that both the absolute and relative refractory period were prolonged in the anterior division of the LBB. When a large series of cases of intermittent BBB was studied, it was found that, in some cases, recovery was prolonged and extremely abrupt, so that incomplete forms of BBB did not occur; whereas in other cases, recovery was prolonged and much more gradual, so that many different degrees of incomplete BBB could be observed (unpublished observations). Thus, during phase-3 block, both the presence or absence of different degrees of block can be anticipated.

In phase-4 block, since the slope of SDD is rather slow, the existence of different degrees of block should be apparently the rule. Indeed, progressively increasing degrees of phase-4 LAH were documented in the case reported in this paper, as theoretically predicted. However, it should be mentioned that the same did not hold true in seven out of nine cases of intermittent BBB previously reported.18,19 This problem will be extensively analyzed in a future publication.

Absence of Escape Beats

The absence of escape beats during the prolonged diastolic intervals provoked by vagal stimulation is remarkable. If SDD is invoked, at least escapes from the injured area should be anticipated. However, in the present case, as well as in the previously reported cases,18,19 the absence of escapes actually allowed the extremely long pauses which so well unveiled the phase-4 component of intermittent BBB. The main reason for the absence of escapes is that the threshold potential is shifted toward zero as a sine qua non condition for phase-4...
block to occur; and this, by itself, may prevent the firing of cells in which SDD is present. However, two other possibilities may be considered. One is that SDD may reach some particular level, and then remain stable without further depolarization. The second possibility is that escapes perhaps do occur, but are not conducted beyond the area of injury (concealed escapes), caused by the blocking effect of SDD on the neighboring fibers.

Types of Tissue Involved

In all the cases so far published, phase-4 block was related to the presence of either right or left BBB. On the other hand, phase-4 block has been demonstrated to occur in peripheral strands of Purkinje fibers. The present case indicates that the same physiologic derangement may occur halfway in between the main bundle branches and the peripheral Purkinje fibers, in the main divisions of the LBB. This suggests that phase-4 block is a general mechanism of abnormal conduction, which may probably occur in any part of the conduction system, from the NH region (where SDD is high) onwards.

All possible varieties of block, including prolonged conduction time, 2:1 and 3:1 block, Wenckebach periods, Mobitz II block, concealed conduction, etc., have been shown to occur clinically in the divisions of the LBB, in the same way as they can occur in the main bundle branches. The fact that phase-4 block can also occur in the divisions of the LBB as well as in the main bundle branches, is another evidence that the divisions of the LBB can be considered not only anatomically, but also physiologically, as discrete and independent fascicles.

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