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## LETTERS TO THE EDITOR

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Letters to the Editor will be published, if suitable, and as space permits. They should not exceed 1,000 words (double spaced) in length, and may be subject to editing or abridgment.

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### Lown-Ganong-Levine Syndrome

To the Editor:

The article by William J. Mandel, Ronald Danzig, and H. Hayakawa (*CIRCULATION* 44: 696, 1971) offers three possible models to explain their results. In three patients they documented normal atrial-to-His (A-H) intervals, normal right bundle-branch-to-ventricle (RBB-V) intervals, but uniformly abbreviated His-to-ventricle (H-V) intervals. Because their patients had low normal A-H intervals which increased with atrial pacing they felt that complete A-V nodal bypass by the posterior internodal tract (PIT) was not the anatomic feature of their patients' syndrome, thus eliminating their first model. However, these same findings led them to believe that at least partial bypass of the A-V node was present in their subjects. Inasmuch as the QRS complexes of their patients were not distorted, accelerated conduction via possible His-Mahaim fibers was logically considered unlikely, thus eliminating their second possible model. They then suggested that accelerated conduction distal to the A-V node but apparently proximal to the recording site of their right bundle-branch electrode provided the preferred model for the Lown-Ganong-Levine syndrome as manifested by their patients.

I would like to suggest a fourth possible model which can explain the data they have collected. It is somewhat simpler to conceptualize, for it does not need to impute accelerated conduction. It further is readily amenable to clinical testing. The two diagrams provided in figure 1 adapted from the article by Mandel et al. show the posterior internodal tract joining the His-Purkinje system just distal to the site where the His potentials were recorded. Under these circumstances the proximal portion of the His bundle, and particularly the area subjacent to the His electrode, may be depolarized in a retrograde fashion. Synchronously the distal His-Purkinje system is depolarized antegrade. The resulting A-H interval would be at the lower normal limit. The H-V interval, however, would be truncated by an interval equal to twice the retrograde conduction time to the His recording electrode. For example, using the data obtained from case 1 in Mandel's report, where the A-H interval equals 82 msec and the H-V

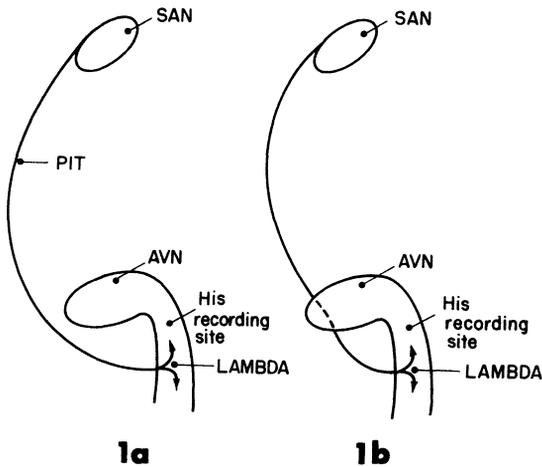
interval equals 28 msec, if the conduction time from the sinoatrial node to point lambda equaled 75 msec then the retrograde conduction time from lambda to point H would equal 7 msec. Since antegrade and retrograde conduction from point lambda could be occurring simultaneously, then the actual lambda-to-V time would be  $7 + 28 = 35$  msec, and the actual H-to-V time would be  $2 \times 7 + 28 = 42$  msec, which is within the normal H-V time interval range. Assuming a conduction velocity of approximately 1 m/sec, the distance between the His recording electrode and the junctional point of the accessory pathway would be approximately 7 mm.

The variation in A-H time observed in two of the patients studied by Mandel during rapid atrial pacing may be explained by the slightly modified conduction illustrated in figure 1b. Here the impulse may arrive at the A-V node by the normal tracts, emerge from it prematurely, bypass the site selected for His bundle recording, and reenter the His-Purkinje conduction system as in figure 1a. This would explain, as Mandel et al. suggest, the moderate prolongation in A-H interval during rapid atrial pacing, and would also explain the failure of such maneuvers to modify the H-V interval.

Since Mandel et al. did confirm by ventricular pacing the capability of V-A condition in their patients, this model is particularly attractive. It further suggests the reentry route by which at least two of their patients sustained supraventricular tachycardias. Finally, this model can be readily tested clinically. Pacing with the His catheter as positioned should produce an H-V interval considerably longer than observed during sinoatrial rhythms if retrograde His depolarization is involved in the latter. Further, if either model 1a or 1b is correct, careful repositioning of the His recording catheter should add substantiating evidence. Moving the catheter proximally should result in a paradoxical shortening of the H-V interval moving it distally as far as point lambda would paradoxically prolong the H-V interval.

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**Figure 1**

*Schematic diagram of an alternate possible mechanism in the Lown-Ganong-Levine syndrome. The impulse from the sinoatrial node (SAN) arrives either via the posterior internodal tract (PIT) or the normal tract to the atrioventricular node (AVN) at a His-Purkinje junction (LAMBDA) distal to the recording site of the His electrode. Retrograde depolarization of the proximal, and antegrade depolarization of the distal His-Purkinje system may therefore occur, producing an unusually shortened H-V interval.*

*The authors reply:*

To the Editor:

Dr. Douglas presents a thoughtful analysis of how a bypass fiber entering the His bundle distal to the recording electrode can result in apparent shortening of the H-V interval due to simultaneous antegrade and retrograde conduction. Depending upon the relative proximity of the bypass fiber to the proximal His bundle, the distal His bundle, and antegrade versus retrograde conduction times, it would also be possible to explain simultaneous occurrence of His and ventricular electrograms, or His potential following the V potential.

In order to explain the variation in A-H time observed in two of the patients during rapid atrial pacing, Dr. Douglas essentially postulates a bypass fiber between the upper A-V node and mid-His bundle. Although theoretically possible, such an anatomic variant has as yet, to our knowledge, not been demonstrated.

The possibility of accelerated A-V conduction cannot be ruled out. Fibers of greater diameter or hyperpolarized fibers could account for such a phenomenon.

Based upon the recent work of Drs. Moore and Spear<sup>1</sup> demonstrating absence of delta waves in X, Y, and Z ECGs with preexcitation of the base of septum along the course of the His bundle, we

would have to consider a combination of James and Mahaim fibers as a more likely explanation of the low normal A-H and short H-V times.

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**Reference**

1. MOORE EN, SPEAR JF: Electrophysiological studies on simulated preexcitation. (Abstr) *Circulation* 44 (suppl II): II-74, 1971

**Pacemaker Failure following External Defibrillation**

To the Editor:

The "Brief Communication" by Dr. Giedwojn (CIRCULATION 44: 293, 1971) requires additional comment.

Knowledge of "normal" function of a pacemaker is essential in order to realize that the abnormalities depicted in the electrocardiograms might easily represent normal pacemaker behavior. In strip B, for example, where the author states that the "pacemaker rate was faster than the set rate," one can easily conceive of the phenomenon of partial recycling of the pacemaker by a relatively weak intracardiac signal. The rest of that strip revealed persistent ventricular fibrillation which undoubtedly activated the pacemaker-sensing circuit and suppressed any pacemaker activity. Similarly, in strip C, with the rate apparently slower, there may have been intracardiac activity not detected on the surface electrogram. (The author also suggested this possibility.) To further corroborate normal pacemaker function, strips D and E revealed the return to the normal fixed-rate interval.

Obviously, one must be aware of the possibility of damaging a pacemaker with external countershock, but these days most pacemakers are well protected from this eventuality. The author should have suggested a better emergency action, namely the application of an external magnet to the skin overlying the pacemaker to convert it to its fixed-rate mode. In the case presented, however, failure to interrupt ventricular fibrillation by external countershock was a more significant observation than the erroneous conclusion that the pacemaker had been damaged. There are several excellent recent papers on unusual modes of function of noncompetitive pacemakers.<sup>1</sup>

## Lown-Ganong-Levine Syndrome JOHN E. DOUGLAS

*Circulation*. 1972;45:1143-1144  
doi: 10.1161/01.CIR.45.5.1143

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://circ.ahajournals.org/content/45/5/1143.citation>

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