

# Lown-Ganong-Levine Syndrome

## A Study Using His Bundle Electrograms

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### SUMMARY

His bundle recordings were obtained in three patients with histories of recurrent supraventricular tachycardias and electrocardiograms demonstrating a short P-R interval with a normal QRS (the Lown-Ganong-Levine syndrome). The His bundle electrograms obtained during sinus rhythm demonstrated a normal A-H (atrium-to-His bundle) time in one patient and a low normal A-H time in two patients. The H-V (His bundle-to-ventricle) time was short in all three patients. Atrial pacing in two patients produced an attenuated degree of prolongation of the A-H time without a change in conduction distal to the proximal His bundle. The probable mechanisms of accelerated conduction in these three patients include: (1) partial A-V nodal bypass via (a) the posterior internodal tract or (b) functional bypass fibers within the A-V node; and (2) accelerated conduction within the A-V conduction system distal to the A-V node.

### Additional Indexing Words:

Accelerated conduction	Arrhythmias	A-V node	Paraspecific fibers
Posterior internodal tract	Right bundle branch		

THE COMPLEX of paroxysmal tachycardia associated with electrocardiographic evidence of a short P-R interval and normal QRS complex has received increasing attention since Lown, Ganong, and Levine presented a review of their case material in 1952.<sup>1</sup> These authors felt that the genesis of this syndrome was not due to anomalous A-V conduction but suggested the importance of possible endocrine and autonomic nervous

system factors. Recent development of a technique for the recording of electrical activity from the specialized conducting system in the human heart<sup>2</sup> has aided considerably in the study of abnormalities of the A-V conduction system.<sup>2-5</sup> In an effort to elucidate more completely the electrophysiologic features of the Lown-Ganong-Levine syndrome, three patients who presented with this syndrome were studied.

### Method

A-V conduction was studied during cardiac catheterization in the postabsorptive state. Patients were premedicated with secobarbital, 100 mg by mouth, 1 hour prior to catheterization. The procedure was explained to the patients in detail and informed consent was obtained. Using local anesthesia, a tripolar 6Fr electrode catheter was introduced into the right femoral vein by means of the Seldinger technique. The catheter was then advanced, using fluoroscopic control, until the tip was across the septal leaflet of the tricuspid valve. A median branch of the basilic vein was isolated in the left antecubital fossa, and through a venotomy a 6Fr bipolar electrode catheter was

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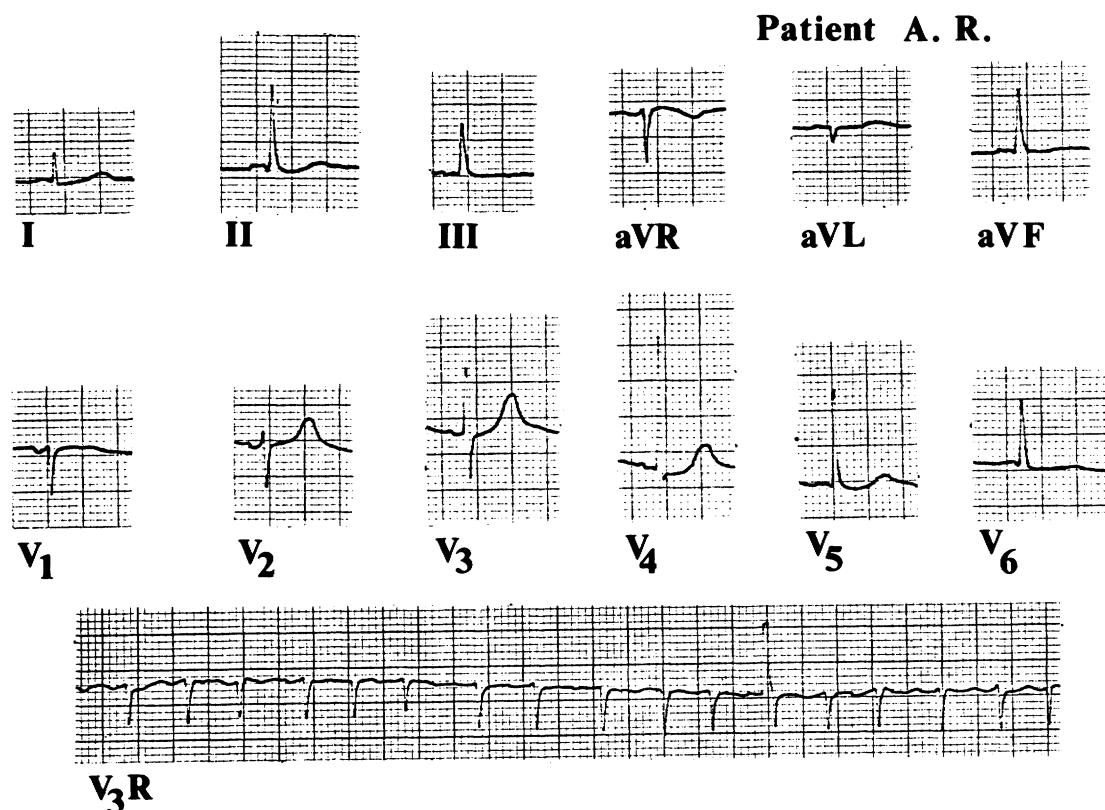
inserted. This catheter was positioned under fluoroscopic control at the junction of the superior vena cava and right atrium, and was used either for atrial pacing or for recording of a bipolar atrial electrogram. A standard lead II electrocardiogram was also displayed with the electrograms, and all tracings were recorded on an Electronics for Medicine recorder (DR-8) at variable paper speeds. The filter settings used for the intracardiac electrograms were 40 and 500 Hz. A-H time (normal 50–120 msec) was measured as the interval from the first high-frequency component of the *a* wave to the His bundle potential. H-V time (normal 35–50 msec) was measured from the onset of the His bundle deflection to the earliest ventricular deflection. Right bundle-branch electrograms were obtained by slightly advancing the His bundle recording catheter into the ventricle until a right bundle potential was obtained.

The patients were paced at varying rates using a Medtronic paired-pulse generator (model 5734) with stimulus characteristics (rectangular pulses of 1½–2 times diastolic threshold and 2-msec duration) set to ensure reliable atrial pacing. The

electrode catheters were connected to a specially constructed isolation transformer-distribution box assembly to prevent leakage current from traversing the low-resistance catheter pathways. Careful attention was paid to ensure proper grounding of all electrical equipment in the laboratory.

### Case Histories

**Case 1.** A. R. is a 62-year-old woman with a 10-year history of intermittent palpitations. The episodes occurred suddenly lasting “several minutes” and disappeared without warning. They were not associated with chest pain, dizziness, or dyspnea. There was no family history of arrhythmias. The patient had smoked one package of cigarettes per day for more than 20 years. Two years prior to this admission, the patient noted the onset of asthmatic attacks which required intermittent treatment with prednisone, Tedral, and aminophylline. The patient had been off all medications for 48 hours prior to the study. Physical examination revealed a blood pressure of 160/90 with a regular pulse rate of 80 beats/min. There was a slight increase in anterior-posterior diameter of the chest with



**Figure 1**

*Patient A. R.'s 12-lead electrocardiogram taken on admission.*

occasional scattered end-inspiratory rales. The remainder of the physical examination was within normal limits. Electrocardiograms demonstrated regular sinus rhythm with a short P-R interval with normal QRS duration (fig. 1), as well as atrial fibrillation. No documentation of the actual onset of atrial fibrillation was obtained in this patient. Serial electrocardiograms had demonstrated P-R-interval variation from 0.10 to 0.12 sec associated with changes in P-wave configuration.

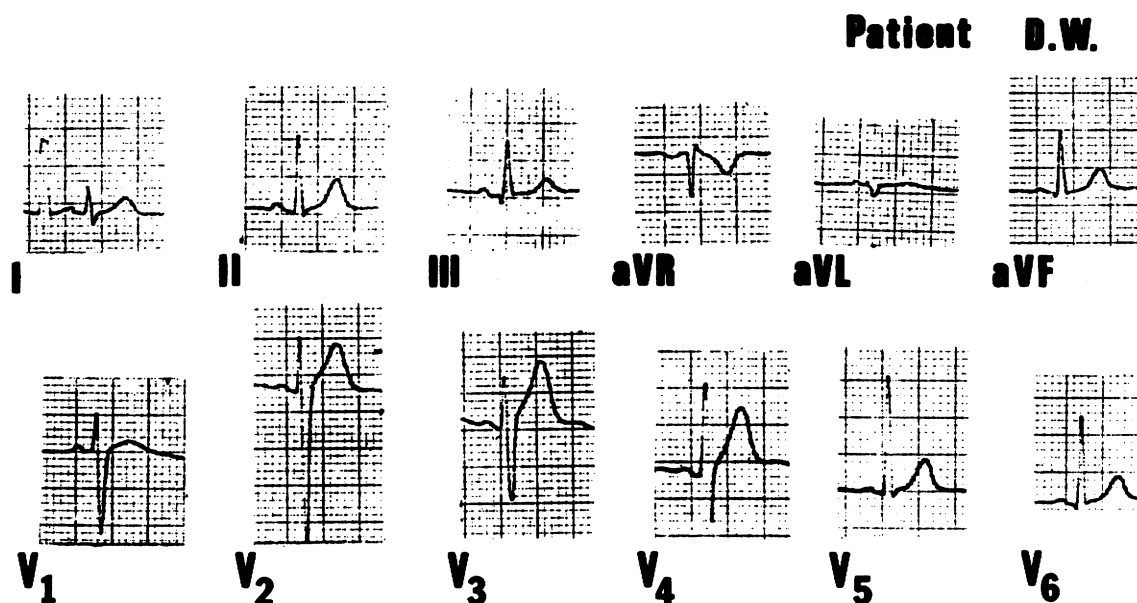
**Case 2.** D. W. is a 21-year-old man with a 3-year history of palpitations. Episodes were characterized by sudden onset unrelated to activity. These episodes would last 5–45 min and then stop suddenly, and they were not associated with pain, dyspnea, or dizziness. There was no family history of arrhythmias. The patient did not smoke and only occasionally drank coffee, and he had not received any antiarrhythmic medication prior to his hospitalization. Physical examination was within normal limits with a blood pressure of 118/78 and a regular pulse rate of 72 beats/min. Studies of thyroid function were within normal limits. An electrocardiogram demonstrated a short P-R interval with normal QRS duration (fig. 2). Numerous electrocardiograms obtained demonstrated P-R intervals varying from 0.12 to 0.14 sec.

**Case 3.** H. A. is a 51-year-old man with at least a 5-year history of palpitations. Two years prior to his present admission he was hospitalized for a myocardial infarction, from which he

recovered without sequelae. There was no family history of arrhythmias, nor did the patient smoke or drink coffee to excess. The arrhythmias, usually atrial fibrillation, would occur spontaneously and last for minutes to several hours. Physical examination revealed a blood pressure of 130/84 with a regular pulse rate of 62 beats/min. The remainder of the physical examination was within normal limits. An electrocardiogram obtained while he was in sinus rhythm demonstrated a short P-R interval with no QRS abnormality (fig. 3). In former years, the patient had received a variety of antiarrhythmic agents, including procainamide, quinidine, propranolol, and various combinations, without success. All antiarrhythmic drugs were discontinued 4 days prior to study. Thyroid-function studies were within normal limits. Prior electrocardiograms demonstrated P-R intervals varying from 0.12 to 0.14 sec.

### Results

**Case 1.** The His-bundle recording during sinus rhythm at a rate of 77/min is shown in panel A of figure 4. A normal A-H time of 82 msec is noted with an abnormally abbreviated (28) H-V time. The His spike is seen to precede the onset of QRS in standard lead II. The patient was then paced from the right atrium at rates of 110, 130, 140, and 156 beats/min (table 1). A representative tracing during pacing at 156 beats/min is shown in



**Figure 2**

*Patient D. W.'s 12-lead electrocardiogram taken on admission.*

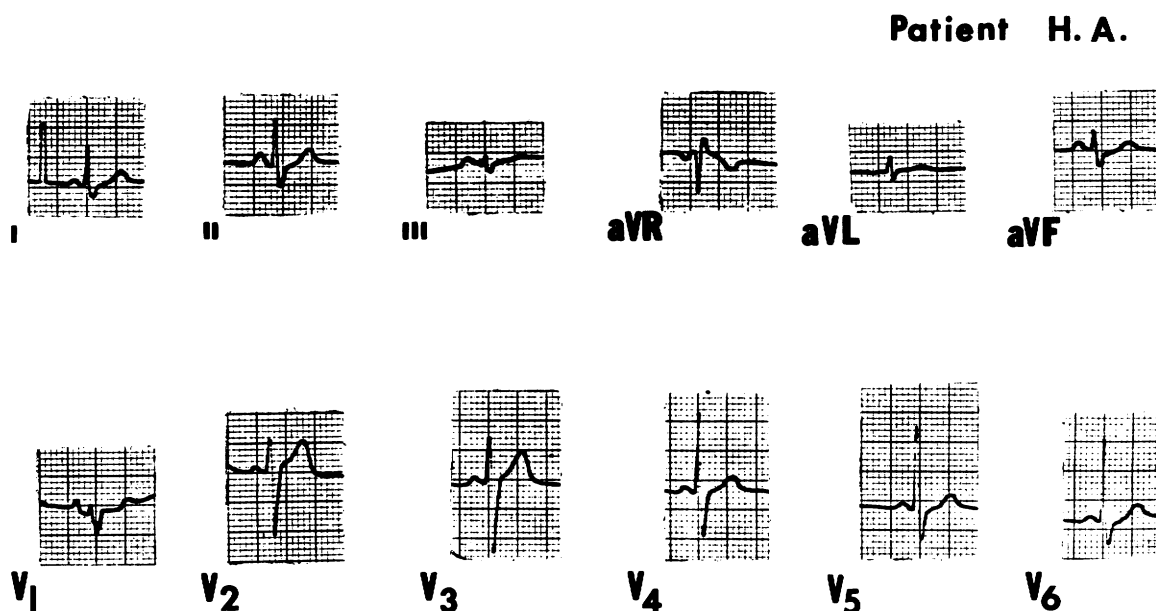
**Table 1***Effect of Atrial Rate on A-V Conduction*

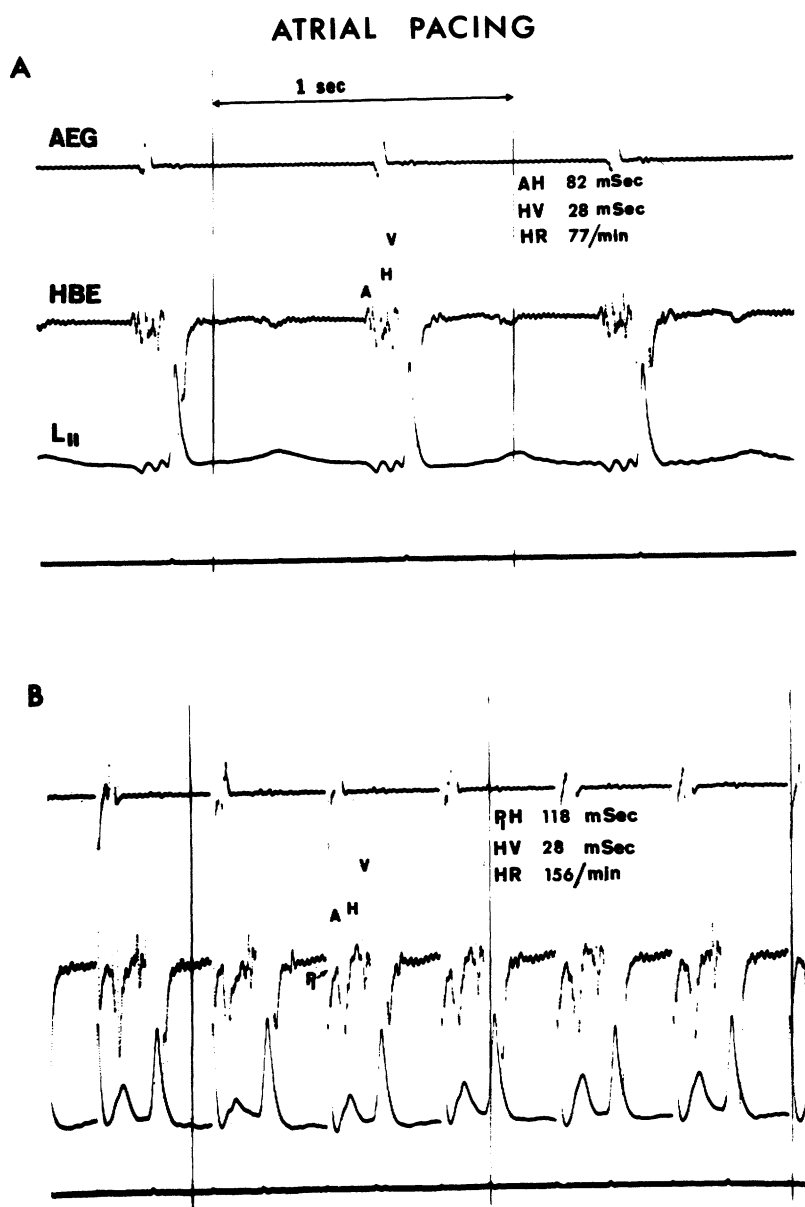
Patient	Atrial rate (beats/min)	A-H time (msec)	H-V time (msec)
A. R.	77	76	28
	110	100	28
	130	115	28
	140	120	28
	156	118	28
D. W.	100	64	27
	120	103	27
H. A.	60	102	24

panel B of figure 4. The P-H time was prolonged from the control of 82 msec to a maximum of 120 msec during pacing at 140 beats/min. There is change in P-wave morphology with high right atrial pacing indicating possible ectopic origin of atrial impulse formation during sinus rhythm. However, A-H times during spontaneous sinus rhythm were within normal limits, suggesting lack of A-V-nodal bypass. Panel A of figure 5 shows an example of His-bundle electrogram trace during sinus rhythm. Panel B of the same figure shows a similar recording demonstrating a right bundle-branch (RBB) potential

obtained during sinus rhythm. The His-RBB time was 15 msec, and the RBB-V time was 13 msec.

**Case 2.** Recordings obtained during sinus rhythm are shown in figure 6. The basic sinus rate is 105 beats/min, and the A-H time was low normal (64 msec). The H-V time of 26 msec is substantially shorter than normal, with the His spike preceding the onset of QRS in standard lead II. Right bundle-branch potentials were also recorded in this patient with RBB-V time of 15 msec. At this rate, there is no evidence of abnormality of QRS. Right atrial pacing was initially performed at a rate of 100/min, and no significant changes in A-H or H-V intervals were seen. The atrial-pacing rate was then increased to 120 beats/min with a 49% increase in the A-H time (103 msec) and no increase in the H-V time (27 msec) (fig. 7, table 1). Further increases in rate were associated with the prompt development of a supraventricular tachycardia. Figure 8 shows the spontaneous onset and termination of one of these episodes in this patient. The onset of the tachycardia was initiated by a premature atrial beat with an inverted P wave and an R-P interval of 332 msec. The tachycardia lasted

**Figure 3***Patient H. A.'s 12-lead electrocardiogram taken on admission.*



**Figure 4**

His bundle electrogram recording obtained during sinus rhythm (A) and after pacing at 156/min (B) in patient A. R. The upper trace in each panel is a bipolar atrial electrogram recorded from the right atrium-superior vena caval junction. The middle trace is the His bundle electrogram (HBE) with A, H, and V signifying low right atrial, His bundle, and ventricular depolarization, respectively. The lower trace is an L<sub>II</sub> (lead II) electrocardiogram. Atrial-His conduction times (AH), His ventricular times (HV), and heart rate are shown in the upper right of each panel. P<sub>i</sub> signifies pacemaker artifact. The vertical lines signify 1-sec time marks.

8 beats with slight R-R oscillation being evident. Termination of the paroxysm was followed by depression of sinoatrial node

(SAN) function for 977 msec, followed by return of sinus rhythm. The patient was then paced with the right ventricle to determine

## HBE vs. RBB

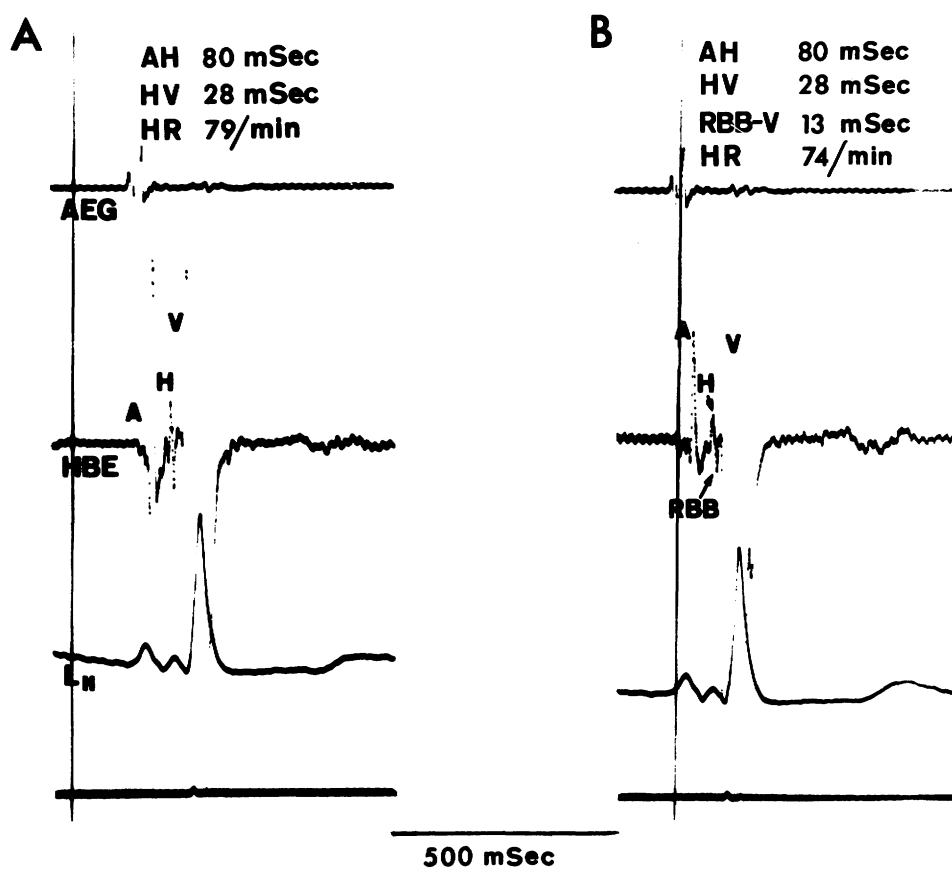


Figure 5

His bundle and right bundle-branch potentials obtained in patient A. R. during sinus rhythm. (A) His bundle recording at a heart rate of 79/min. (B) Record obtained a few moments later with the catheter advanced a few millimeters into the right ventricular cavity. The right bundle-branch potential is now recorded with an RBB-V time of 13 msec.

the state of retrograde conduction. V-A conduction was intact at rates up to 135 beats/min with a maximum conduction time of 195 msec.

*Case 3.* The recording obtained during sinus rhythm at a rate of 60/min is shown in figure 9. A normal A-H time of 102 msec is noted with an abnormally abbreviated (24 msec) H-V time (see table 1). The His spike is seen to precede the onset of QRS in both standard leads I and aV<sub>F</sub>. Right bundle-branch potentials were recorded in this patient demonstrating a RBB-V time of 14 msec. All attempts at pacing the atrium at rates in excess of his sinus rate immediately

resulted in runs of atrial fibrillation. An example of a spontaneous episode is shown in figure 10. The fourth QRS is followed by a premature atrial contraction conducted to the ventricle with aberration. This beat initiates a short run of atrial fibrillation lasting 11 beats. Spontaneous termination was followed by depression of SAN function lasting 1023 msec, followed by return of sinus rhythm. Ventricular pacing was then performed to evaluate retrograde conduction. Pacing at rates of up to 120 beats/min were associated with 1:1 V-A conduction, with a maximum retrograde conduction time of 142 msec. No further increases

PATIENT DW

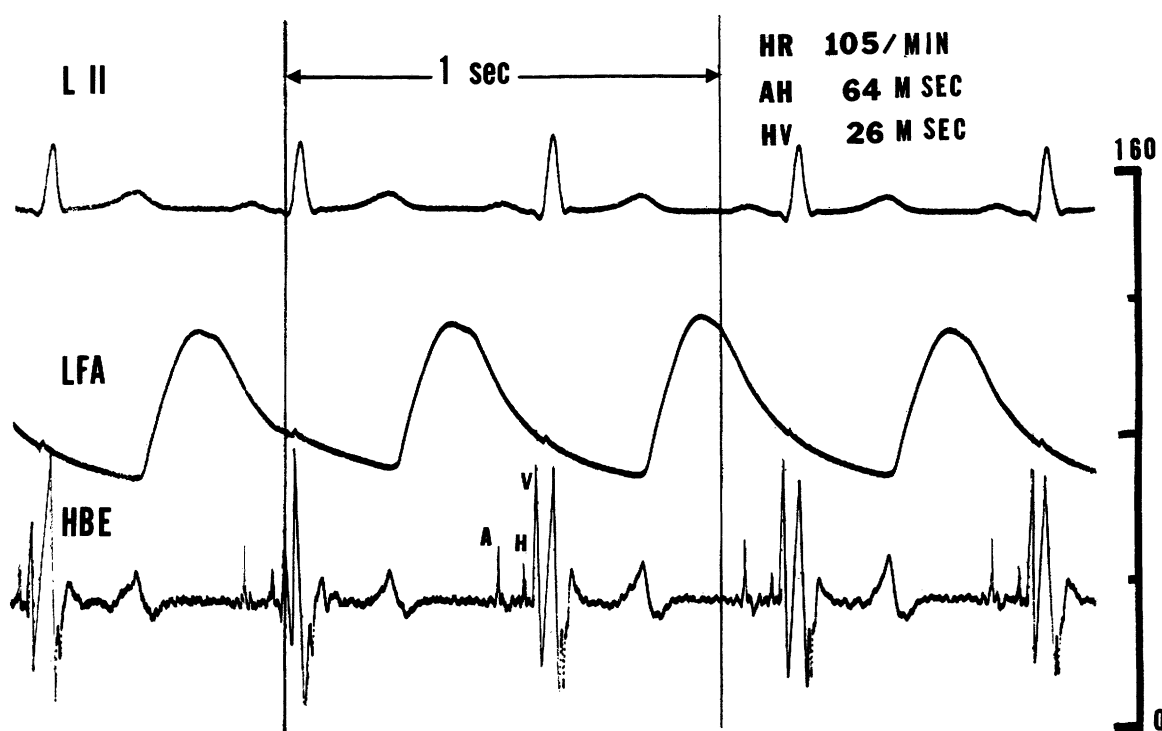


Figure 6

His bundle electrogram recordings in patient D. W. obtained during sinus rhythm. The upper trace is lead II electrocardiogram, and the middle strip is the left femoral intraarterial blood pressure. The lower trace is the His bundle electrogram with A, H, and V signifying low right atrial, His bundle, and ventricular depolarization, respectively. Heart rate (HR), atrial-His conduction times (AH), and His-ventricular times (HV) are shown in the upper right of the figure and a 1-sec time calibration is shown at the top of the figure. Abbreviations used in subsequent figures are similar to these.

in pacing rate were attempted, because of the potential induction of atrial arrhythmias.

### Discussion

The three patient studies are presented here in an attempt to define more clearly the electrophysiologic characteristics of the Lown-Ganong-Levine (LGL) syndrome. Electrograms from the area of the proximal His bundle in all three demonstrate the presence of a short H-V conduction time distal to the His bundle. Recording of His and right bundle-branch electrograms in each patient eliminated the possibility that the shortened H-V time, in fact, represents the right bundle branch-ventricular (RBB-V) conduction time. The RBB-V conduction times obtained in

these patients are consistent with recent published reports.<sup>6,7</sup> The etiology of this acceleration of conduction may be either related to a partial bypass of the A-V conduction system or an actual acceleration of conduction through the normal pathways.

The Wolff-Parkinson-White syndrome (WPW) has recently been studied in detail using a technique similar to the one used in the present study.<sup>8,9</sup> These authors found that conduction time proximal and distal to the His bundle was within normal limits. The His potential, however, was preceded by approximately 10 msec, by the inscription of the delta wave of the surface electrocardiographic lead. This finding strongly suggested that ventricular preexcitation was due to anatomic bypass

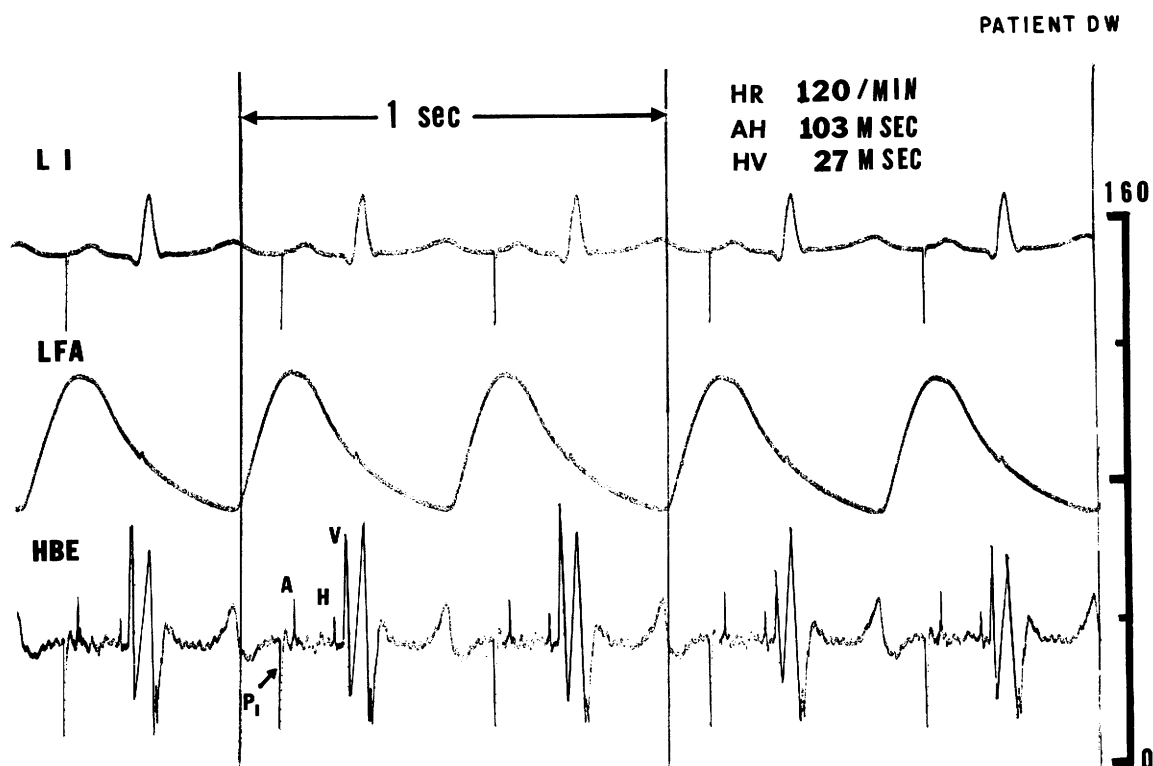


Figure 7

*His bundle electrogram in patient D. W. during right atrial pacing at 120/min. P<sub>1</sub> signifies stimulus artifact. Note the prolongation in AH time without any change in the HV time.*

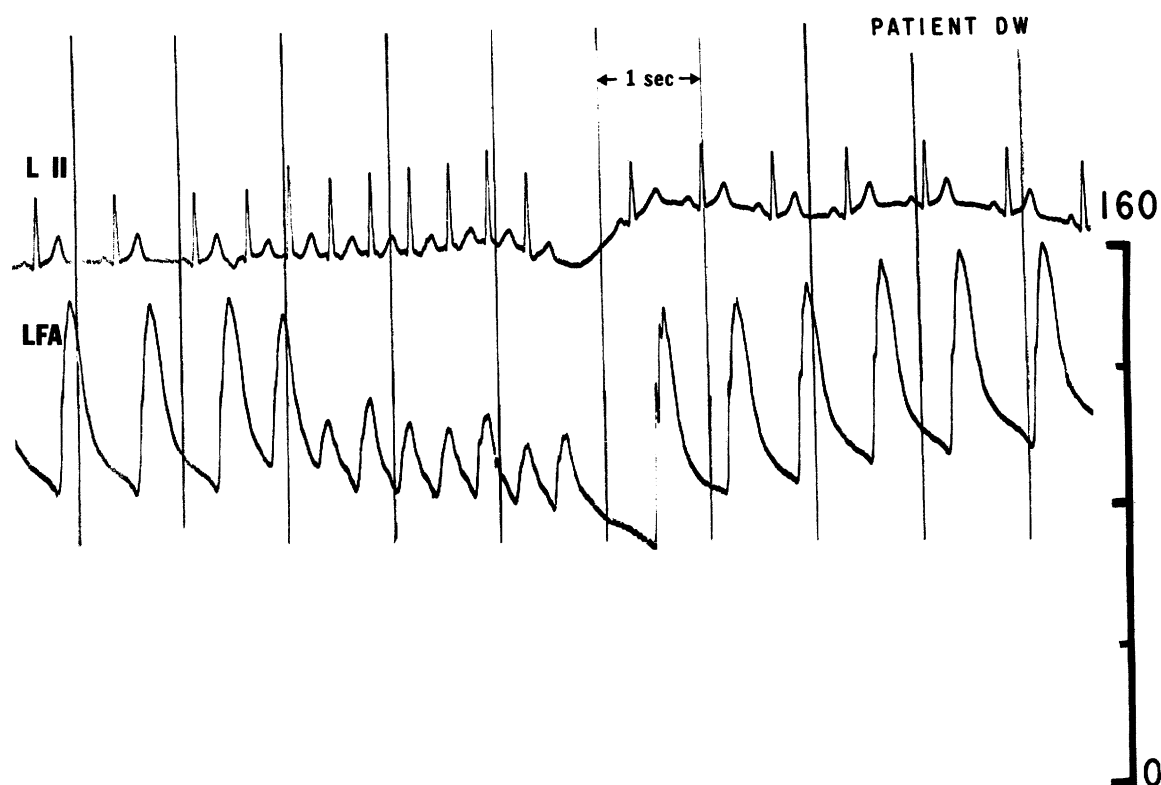
of the A-V node. The presence of A-V-nodal bypass tracts has been anatomically confirmed by numerous studies.<sup>10-13</sup> Recent reviews of the electrophysiologic-anatomic correlates of the WPW syndrome have suggested that the possible major anatomic variants include: right or left bundle of Kent, Mahaim fibers, posterior internodal tract, or a combination of these.<sup>14-16</sup>

The LGL syndrome has been felt by some to represent a variant of the WPW syndrome.<sup>17-19</sup> Three major possibilities exist to explain the development of short P-R with a normal QRS duration (fig. 11). The first possibility is the presence of paraspecific Mahaim fibers, which act as a short circuit to the ventricular septum distal to the A-V node.<sup>20</sup> Durrer<sup>21</sup> and Castillo and Castellanos<sup>22</sup> have suggested that the LGL syndrome may in fact represent conduction via these paraspecific fibers. Criteria established by James<sup>23</sup> and Ferrer<sup>14</sup> for the electrocardio-

graphic diagnosis of Mahaim-fiber conduction include: (a) normal P-R interval with an isoelectric P-R segment and (b) an initial abnormality of QRS. Since Mahaim fibers classically enter the ventricular septum in an area that might be expected to result in an abnormal initial sequence of activation, the presence of normal initial QRS forces in the three patients studied would suggest that these fibers are not of etiologic significance. However, if small, distally located paraspecific fibers were present, it is possible that no initial QRS morphologic changes would be noted. One would then also have to postulate that conduction in the bypass fiber was more rapid than in the His bundle to explain the findings in the three patients reported here.

A second possibility may be partial A-V-nodal bypass via the posterior internodal tract. This is a specialized atrial tract, recently described in detail by James, which traverses the posterior right atrium from the sinoatrial





**Figure 8**

*Electrocardiogram and femoral arterial pressure at onset and termination of the arrhythmia in patient D. W. The third QRS is followed by a premature inverted P wave, which is followed by a run of tachycardia at a rate of 160/min lasting eight beats. A substantial drop in intraarterial blood pressure accompanies the tachycardia.*

node to the A-V node, and may act as a partial or complete bypass of the A-V node.<sup>24</sup> A short P-R interval with a normal QRS would occur if this tract were to bypass the A-V node and enter the proximal His bundle. The presence of a normal QRS with an increase in P-R and A-H intervals with atrial pacing would suggest that complete A-V-nodal bypass via the posterior internodal tract is not the anatomic feature of this syndrome. Delay in A-H interval during atrial pacing could, however, be noted if there is partial bypass of the A-V node or may be due to delayed conduction within the bypass fibers.

A third possibility suggested by Prinzmetal and coworkers and by Moe et al. is that the A-V node or distal conduction system, or both, may contain fibers that have the capability of accelerated conduction.<sup>25, 26</sup> Although not de-

finitively documented in the animal laboratory, the possibility may exist that accelerated conduction within and/or distal to the A-V node accounts for the observed P-R shortening, and thus no QRS abnormality is noted.

Cases 1 and 3 demonstrate: (a) a normal to low normal A-H time, (b) a short H-V time, (c) an attenuated response to atrial pacing, and (d) normal QRS activation. The relatively short A-H times seen in patient 3 may be due to either partial or total anatomic A-V-nodal bypass or to acceleration of conduction through functional bypass fibers within the A-V node. The posterior internodal tract may reenter the A-V conduction system at the A-V node or bundle of His regions accounting for either partial or total anatomic A-V-nodal bypass with accelerated A-V conduction. The attenuated response to atrial

## PATIENT HA

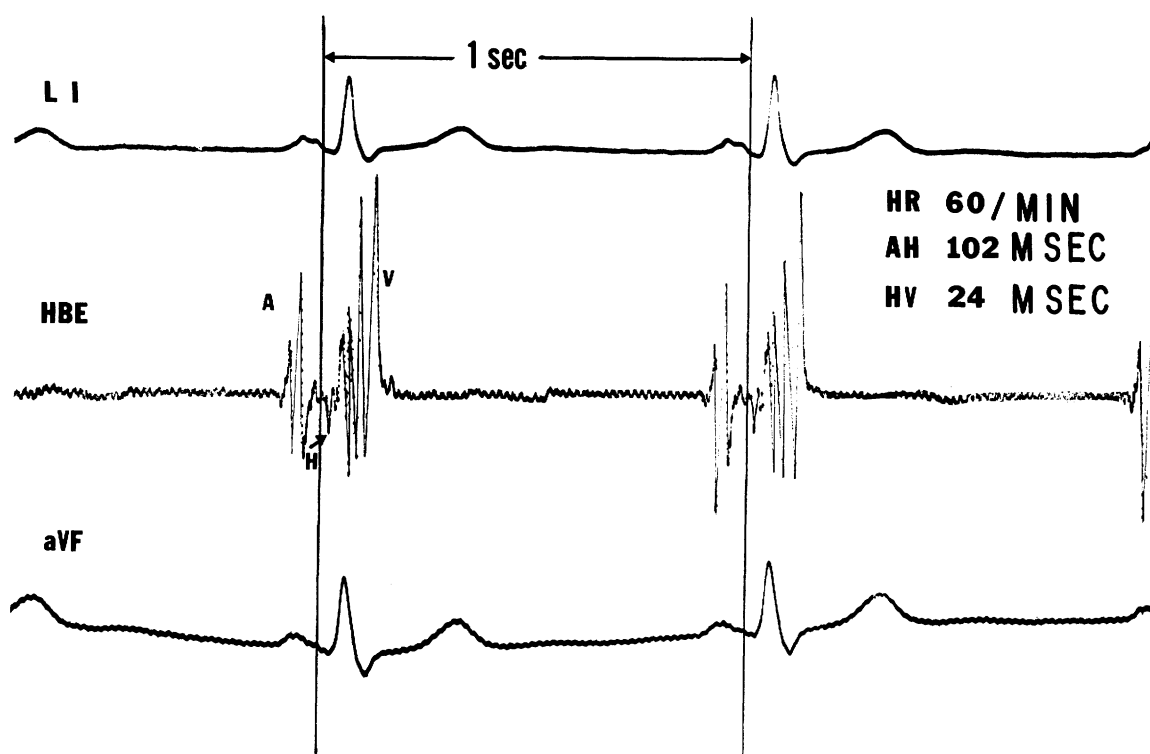


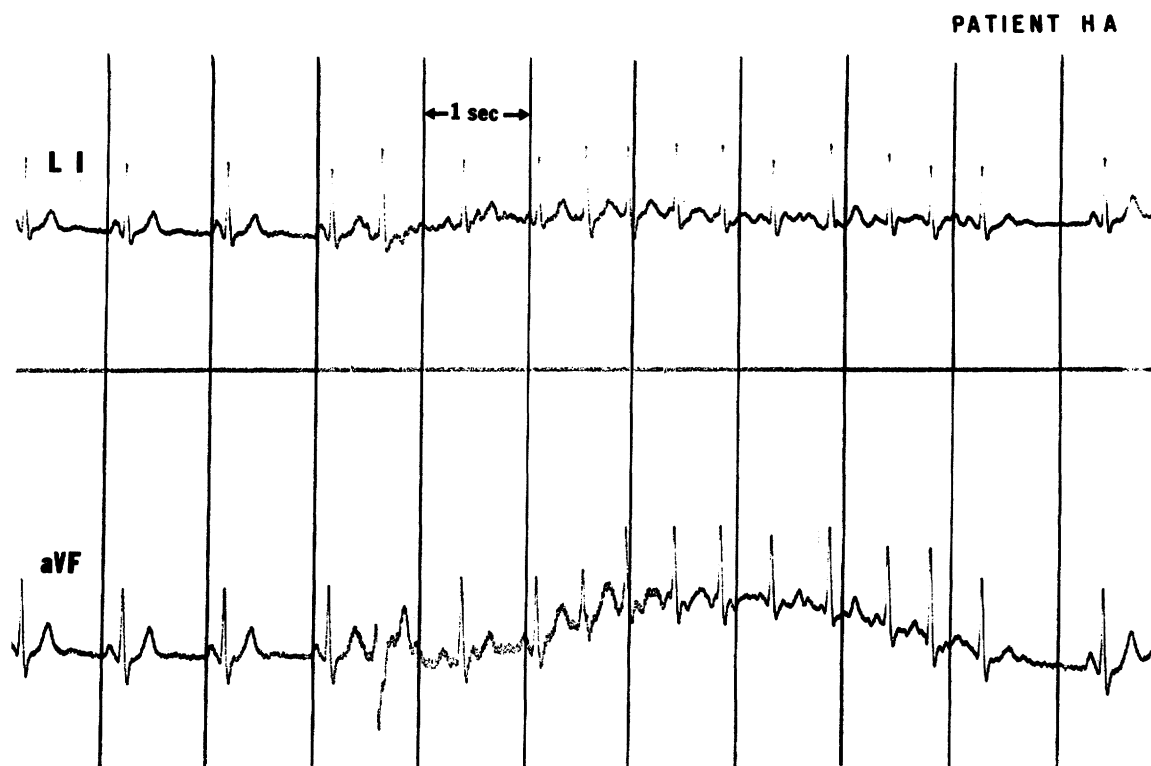
Figure 9

*His bundle electrogram during sinus rhythm in patient H. A. The upper trace is a lead I electrocardiogram, and the lower trace is an aVF electrocardiogram. The middle trace is the His bundle electrogram.*

pacing seen in both patients 1 and 3 would suggest partial A-V-nodal bypass. A more prolonged A-H time in response to atrial pacing would be expected in the absence of A-V-nodal bypass. Total A-V-nodal bypass would be expected to result in a constant P-R interval in response to atrial pacing at increasing rates, unless there was delay within the bypass fibers. Atrial pacing at increasing rates in WPW patients during accelerated conduction did not result in changes in P-R interval, suggesting absence of delay in bypass fibers.<sup>9, 27</sup> The normal QRS would tend to rule out Mahaim-fiber conduction. Thus, accelerated A-V conduction in both patients 1 and 3 can best be explained by partial anatomic or functional bypass of the A-V node and acceleration of conduction distal to the A-V node.

In case 2, the A-H time was normal (102 msec), but the H-V time was again short (24 msec). The response to atrial pacing could not be determined because of the prompt induction of atrial fibrillation with all attempts at pacing the atrium. The same possibilities must, however, be considered as in the other cases. However, the normal A-H time would suggest that conduction through the A-V node may be normal, and acceleration distal to the bundle of His would adequately explain the findings in this patient.

Arrhythmias developed in the three patients studied and are a sine qua non of the LGL syndrome. In patients H. A. and A. R., atrial fibrillation was the arrhythmia documented. In patient H. A., the onset of this arrhythmia occurred after a premature atrial systole. This sequence of atrial fibrillation induced by a



**Figure 10**

*Electrocardiogram during onset and termination of the arrhythmia in patient H. A. The upper and lower traces are L I and aVF electrocardiograms, respectively. The fourth QRS is followed by a premature atrial contraction which is conducted with aberrancy. This beat initiates an episode of atrial fibrillation lasting 11 beats.*

premature atrial systole has been well documented by Killip and Gault and by Bennett and Pentecost.<sup>28, 29</sup> The electrophysiologic significance of this concept may be either the immediate development of atrial fibrillation secondary to the premature atrial systole falling within the atrial "vulnerable period" or the presence of a return extrasystole initiating a run of atrial fibrillation by exciting atrial tissue in "vulnerable period." Patient D.W. developed runs of regular supraventricular tachycardia which were initiated and terminated by premature supraventricular beats which may have originated in the area of the coronary sinus. Based on evidence recently reported by Bigger and Goldreyer,<sup>30, 31</sup> the etiology of this patient's arrhythmia may have been reentry. Unfortunately His bundle recordings were not obtained during the onset of this arrhythmia to define its pathogenesis

more clearly. Further support for a reentry mechanism as the etiology of this arrhythmia is the fact that the arrhythmia could be converted by atrial pacing which presumably interrupts the reentry pathway.

#### Addendum

Since submission of this manuscript, a paper has been published on a similar topic (CASTELLANOS A JR, CASTILLO CA, AGHA AS, TESSLER M: His bundle electrograms in patient with short P-R intervals, narrow QRS complexes, and paroxysmal tachycardias. *Circulation* 43: 667, 1971).

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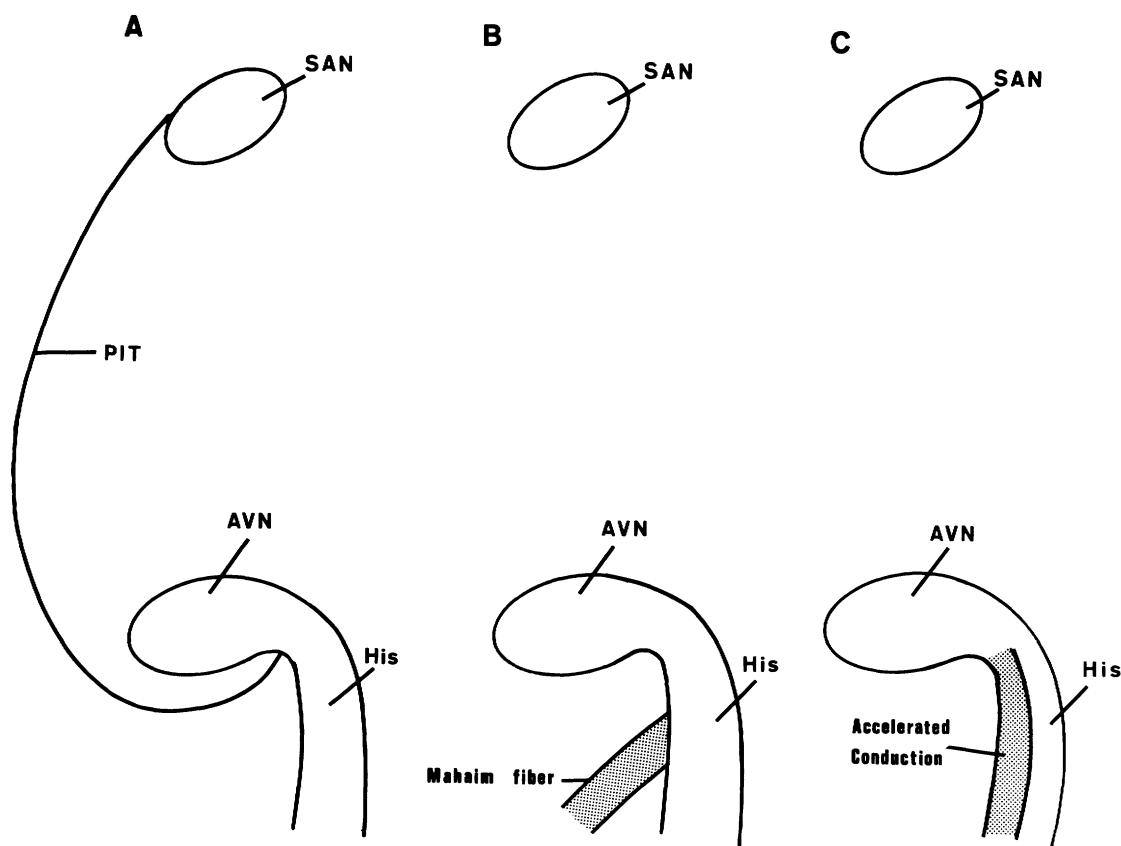


Figure 11

*Schematic diagram of possible mechanisms in the Lown-Ganong-Levine syndrome. (A) Connection from the sinoatrial node (SAN) to the A-V node-proximal His bundle junction by the posterior internodal tract (PIT). (B) Example of a paraspecific or Mahaim fiber. (C) Possible site of accelerated conduction in the conduction system distal to the A-V node (AVN).*

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