Lack of Sensing by Demand Pacemakers due to Intraventricular Conduction Defects

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SUMMARY
A previously unrecognized form of failure of normally functioning noncompetitive R-inhibited or R-triggered pacemakers to sense is described in 30 patients. The failure was produced by the delayed arrival of ventricular depolarization due to intraventricular conduction disturbances at the site of pacemaker sensing electrodes. These patients with late activation of the right ventricle exhibited right bundle branch block (RBBB) during conducted sinus beats or had ectopic beats arising from the left ventricle. In both these situations, several late occurring QRS complexes were not sensed by the pacemaker electrodes in the right ventricular apex (RVA) and thereby the R-inhibited pacemaker spike discharged into the initial portion of the QRS complex. Activation of the RVA was delayed up to 65 m sec in sinus beats with RBBB and even longer in premature beats arising from the left ventricle. The depolarization front of such beats reached the myocardium around the right ventricular pacemaker electrodes late in the QRS and thereby were not sensed up to 65 m sec after the QRS onset as seen on the surface electrocardiogram (ECG). It was found in this study that if the inscription of such QRS complexes began up to 65 m sec before the next due pacemaker impulse, these complexes were not sensed, allowing discharge of normally functioning R-inhibited pacemakers within the QRS which mimicked pacemaker malfunction. Failure to sense due to this phenomenon was observed in patients with noncompetitive pacemakers, both R-inhibited and R-triggered; temporary and permanent; with unipolar as well as bipolar electrodes. Similarly, patients with left ventricular epicardial electrodes and left bundle branch block in sinus beats also exhibited this phenomenon. Complete evaluation proved that pacemaker function was normal in the entire patient group. Recognition that failure to sense was the result of intraventricular conduction disorder prevented the untimely replacement of the pulse generator. This phenomenon of nonsensing by noncompetitive pacemakers should be considered in the presence of wide QRS complexes before the pacemaker is concluded to be malfunctioning.

Additional Indexing Words:
- Bipolar electrodes
- Fusion beats
- Noncompetitive pacemaker
- R-inhibited pacemaker
- Endocardial electrodes
- Heart block
- Pacemaker function
- R-triggered pacemaker
- Epicardial electrodes
- Left bundle branch block
- Premature ventricular beats
- Right bundle branch block

The sensing circuitry of demand pacemakers is designed to sense appropriate intrinsic signals of at least 2 to 3 mV amplitude. Oversensing or unexpected sensing of a signal is an uncommon finding and has been reported in association with prominent T waves,1 concealed ventricular extrasystoles,2 and also with pacemaker after-potential.3 Undersensing or lack of sensing by a normal demand pacemaker is a relatively common finding4 and is encountered when the signal a) falls in the pacemaker refractory period, b) is of inadequate voltage, or c) does not have an optimal spatial relation to the pacemaker electrodes. Undersensing may also occur if d) the sensing electrodes are positioned in an abnormal location or e) the demand function of the pacemaker system is rendered temporarily inoperative due to electromagnetic interference. The purpose of the present report is to describe a previously unrecognized form of lack of sensing by normal pacemakers which is consequent to intraventricular conduction disturbances. This phenomenon may occur in normal sinus rhythm with bundle branch block or in the presence of ectopic QRS complexes arising in the ventricle that does not contain the pacemaker electrodes. Undersensing due to abnormal intraventricular conduction has been
documented in our laboratories in a variety of patients with both the R-inhibited and R-triggered types of noncompetitive pacemakers.

Right bundle branch block (RBBB) combined with left anterior hemiblock (LAH) is a common type of bilateral bundle branch block. Since there is a high frequency of complete heart block in RBBB with LAH, patients with this conduction disturbance commonly require permanent pacemakers. Therefore, in the present report, the mechanism of undersensing associated with intraventricular conduction disorders usually involved delayed depolarization of the right ventricle in patients with transvenous electrodes. An introductory explanation is provided diagrammatically in figure 1. A sinus impulse in the presence of RBBB is known to arrive at the right ventricular apex 50–60 msec after the onset of the QRS complex. Therefore, in patients with RBBB and right ventricular R-inhibited pacemakers, the sinus impulse reaches the site of the apical electrode in the right ventricle well after the onset of the QRS complex (fig. 1A). When activation of the apex is sufficiently late (beat number 5), the pacemaker cycle is completed with its resultant spike delivered within the QRS, thereby suggesting loss of sensing due to pacemaker malfunction. This undersensing process may also take place following ectopic left ventricular beats (fig. 1B) with delayed spread of impulses to the electrode site.

Methods and Results

The phenomenon of failure to sense due to late arrival of impulse at the pacemaker electrode recording site was observed in 30 of 110 patients requiring permanent or temporary pacing. This was most frequently observed in patients with RBBB during normal sinus rhythm (NSR) and was brought about by spontaneous slowing of the sinus rate (fig. 2), and in these patients, could also be unmasked easily by induced slowing of sinus rate by carotid sinus massage. Pertinent clinical information in 13 of these 30 patients is described in table 1 and six patients who represent special aspects of the spectrum of this phenomenon are presented in detail. Ten of these patients had right ventricular (RV) endocardial, two RV epicardial, and one left ventricular (LV) epicardial noncompetitive pacemakers. Nine pacemakers were unipolar and four bipolar; 11 were R-inhibited and two R-triggered; and ten were permanent and three temporary. The pacemakers involved were manufactured by three different companies as indicated in table 1. In all but one patient, QRS complexes not sensed were characterized by RBBB type QRS pattern in lead V1 and appeared late with wide coupling inter-

Figure 1

Diagrammatic representation of the mechanism of undersensing caused by abnormal intraventricular conduction due to late arrival of impulse at the electrode site in the right ventricular apex (RVA). The examples given are representative of patients with right ventricular endocardial R-inhibited demand pacemakers. Panel A depicts the situation in sinus beats with right bundle branch block (RBBB). Complex 3, a sinus beat with RBBB configuration, is sensed by the pacemaker, thereby suppressing the next pacemaker discharge which is recycled. The time of sensing of complex 3 can be determined by measuring one full pacemaker cycle length (as between 1 and 2) back from complex 4 in the absence of pacemaker hysteresis. The arrow indicates the moment complex 3 is sensed, which is clearly after the QRS onset. The time from onset of QRS 3 to the arrow is the delay interval of RVA activation in RBBB. After the paced beat 4, a conducted beat 5, similar to complex 3, occurs later than complex 3 relative to the preceding paced QRS. The pacemaker automatic interval is completed before the conducted impulse 5 has time to reach the sensing electrode and thereby the pacemaker spike is generated within complex 5. Likewise, in panel B, an ectopic left ventricular impulse arrives late at the RVA sensing electrode site and therefore is sensed considerably after the QRS onset in the surface lead (arrow, QRS complex 8). Another ectopic QRS of similar configuration (beat 10) occurring with a longer coupling interval allows completion of the pacemaker cycle with its electrical discharge appearing within the surface-recorded QRS before the intrinsic impulse reaches the sensing electrode. RBB = right bundle branch; LBB = left bundle branch; E = transvenous bipolar electrode.

Figure 2

Continuous V1 rhythm strip (paper speed 50 mm/sec) from a patient with normal sinus rhythm and RBBB who received a permanent transvenous, unipolar, R-inhibited (Medtronic 3945) pulse generator with RVA electrode. Sinus beats 1 and 2 with RBBB were normally sensed and inhibited the demand pacemaker. Similar complexes 3 through 8, with slightly longer R-R cycles than the pacemaker cycle length, contained pacemaker spikes 30 to 65 msec after the onset of these QRS complexes. Complex 9, an ectopic ventricular beat, was normally sensed and QRS 10 and 11 are pacemaker-induced.
Table 1

Conduction Disturbances and Pacemaker Characteristics in Representative Patients Demonstrating Failure to Sense due to Intraventricular Conduction Defects

<table>
<thead>
<tr>
<th>Patient</th>
<th>Rhythm</th>
<th>QRS configuration</th>
<th>Sensing electrode(s) and location</th>
<th>Pacemaker</th>
<th>Maximum QRS-Spike interval (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. W.C.</td>
<td>NSR</td>
<td>RBBB</td>
<td>Uni; Endo; RVA</td>
<td>Medtronic 5945 P</td>
<td>60</td>
</tr>
<tr>
<td>2. O.A.</td>
<td>NSR</td>
<td>RBBB + LAH</td>
<td>Bi; Endo; RVA</td>
<td>Medtronic 5942 P</td>
<td>60</td>
</tr>
<tr>
<td>3. T.M.</td>
<td>SSS; EVB</td>
<td>RBBB pattern</td>
<td>Uni; Endo; RVA</td>
<td>Omni-Stanicor P</td>
<td>80</td>
</tr>
<tr>
<td>4. A.C.</td>
<td>CHB; EVB</td>
<td>RBBB pattern</td>
<td>Uni; Epi; RVA</td>
<td>Medtronic 5880A T</td>
<td>70</td>
</tr>
<tr>
<td>5. J.W.</td>
<td>NSR</td>
<td>LBBB</td>
<td>Uni; Epi; LV</td>
<td>Medtronic 5961 P</td>
<td>55</td>
</tr>
<tr>
<td>6. T.E.</td>
<td>SSS; EVB</td>
<td>RBBB pattern</td>
<td>Uni; Endo; RVA</td>
<td>Cordis EctoCor P</td>
<td>80</td>
</tr>
<tr>
<td>7. R.K.</td>
<td>2°HB; EVB</td>
<td>RBBB pattern</td>
<td>Uni; Endo; RVA</td>
<td>Cordis StaneCor P</td>
<td>100</td>
</tr>
<tr>
<td>8. M.G.</td>
<td>NSR</td>
<td>RBBB + LAH</td>
<td>Bi; Endo; RVA</td>
<td>American Optical T</td>
<td>65</td>
</tr>
<tr>
<td>9. J.B.</td>
<td>NSR</td>
<td>RBBB + LAH</td>
<td>Bi; Endo; RVA</td>
<td>Medtronic 5942 P</td>
<td>60</td>
</tr>
<tr>
<td>10. M.P.</td>
<td>2°HB; NSR</td>
<td>RBBB + LAH</td>
<td>Uni; Endo; RVA</td>
<td>Cordis EctoCor P</td>
<td>55</td>
</tr>
<tr>
<td>11. J.M.</td>
<td>NSR</td>
<td>RBBB + LPH</td>
<td>Bi; Endo; RVA</td>
<td>Medtronic 5942 P</td>
<td>45</td>
</tr>
<tr>
<td>12. Z.T.</td>
<td>CHB; EVB</td>
<td>RBBB pattern</td>
<td>Uni; Endo; RVA</td>
<td>Omni-Stanicor P</td>
<td>90</td>
</tr>
<tr>
<td>13. R.P.</td>
<td>NSR; EVB</td>
<td>RBBB pattern</td>
<td>Uni; Epi; RVA</td>
<td>Medtronic 5880A T</td>
<td>90</td>
</tr>
</tbody>
</table>

Abbreviations: RBBB = right bundle branch block; LBBB = left bundle branch block; NSR = normal sinus rhythm; EVB = ectopic ventricular beats; SSS = sick sinus node syndrome; 2°HB = second degree heart block; CHB = complete heart block; LAH = left anterior hemiblock; LPH = left posterior hemiblock; Uni = unipolar electrode; Bi = bipolar electrode; Endo = epicardial electrode; Epi = epicardial electrode; RVA = right ventricular apex; P = permanent pacemaker; T = temporary pacemaker.

vals; there was a delay of up to 65 msec from QRS onset to the next pacemaker spike within the QRS in instances of conducted beats with RBBB and up to 100 msec in ectopic LV beats. The remaining patient had left bundle branch block (LBBB) and LV epicardial electrodes.

In each patient, all parameters of pacemaker function were thoroughly evaluated7 and documented to be normal: analysis of tracings revealed no change in fixed rate obtained by application of magnet over the pulse generator; oscilloscopic examination revealed no change in pacemaker artifact amplitude, width, or configuration; radiologic and fluoroscopic examination showed normal electrode location with stable position, as corroborated by appropriate and unaltered paced QRS configuration;8 and the possibility of myocardial perforation was excluded by absence of pericardial rub, intercostal muscle twitch, or change in paced QRS pattern. All patients showed satisfactory intracardiac voltage at the time of pacemaker implantation. No patient had acute myocardial infarction. In patients with RV electrodes, all conducted beats with RBBB and ectopic QRS complexes with RBBB configuration appearing earlier than those not sensed, were detected with delay in their sensing. Otherwise, conducted complexes and spontaneous idioventricular systoles without RBBB patterns were properly sensed without delay by RV electrodes. Normal sensing was also documented by the method of chest wall stimulation.9

R-inhibited Pacemakers with RV Electrodes

Patient 1 (NSR with RBBB and Unipolar Pacemaker)

A 65-year-old male with old inferior myocardial infarction and RBBB received a permanent R-inhibited transvenous pacemaker (Medtronic 5945) because of Stokes-Adams attacks. The unipolar electrode catheter was positioned transvenously in the right ventricular apex. Three days after pacemaker implantation, lead V1 rhythm strip (fig. 2) revealed transient periods of normal sinus rhythm (NSR) with RBBB during which time the pacemaker impulses were discharging from 30 to 65 msec after onset of certain QRS complexes (beats 3 through 8). Sinus beat 2
with slightly shorter preceding cycle length and LV ectopic beat 9 were properly sensed. The pacing mechanism was intact as manifested by paced beats 10 and 11. The patient was asymptomatic and repeated evaluations by Holter ambulatory monitoring and long rhythm strips demonstrated pacemaker discharges to occur only in the first 65 msec of some conducted RBBB-QRS complexes. No such pacemaker discharges were seen to occur in the latter part of the QRS or during the ST-T segments. Extensive investigation as outlined above revealed no other evidence to implicate pacemaker malfunction. Intracardiac voltage obtained at the time of pacemaker implantation was 8.7 mV and thus could not be implicated as a cause of the intermittent nonsensing. Serial follow-up of the patient for the next three months revealed no disorders in pacemaker function. In view of the pacemaker discharges occurring only in the first 65 msec of the QRS complexes with RBBB, normal sensing occurring in the latter portion of such complexes, and exclusion of other causes of undersensing, it was concluded that failure to sense in this patient was due to RBBB-induced late arrival of the depolarization wavefront at the site of the RV apical sensing electrode as described in figure 1A.

**Patient 2 (NSR with RBBB and Bipolar Pacemaker)**

A 69-year-old male with RBBB plus LAH received a bipolar permanent R-inhibited pacemaker (Medtronic 5942) with RV endocardial electrodes because of intermittent complete heart block. On follow-up evaluation, the patient was asymptomatic and the sensing and pacing functions of the pacemaker were normal. During periods of NSR with RBBB, the pacemaker spike was seen to fall well within some QRS complexes: numbers 5, 7, and 12 in figure 3. QRS complexes with similar configuration inscribed earlier, numbers 6 and 11, or inscribed considerably earlier following the preceding complex, as in beats 4 and 10, were appropriately sensed. It was observed that QRS complexes 5, 7, and 12 possessed a common characteristic in that their inscription began 60 msec or less before the next pacemaker discharge. The most likely explanation for this phenomenon is again, as illustrated in figure 1A, lack of sensing due to RBBB-produced late arrival of the impulse at the right ventricular apical electrode site. The arrow beneath beat 4 shows the time that this complex was sensed. The interval from onset of sensed QRS to the arrow was longer than that of the onset of unsensed QRS to pacemaker spike, with later occurrence of the unsensed QRS as compared to sensed QRS from the immediately preceding QRS complex. These features are characteristic of the mechanism of the failure to sense.

**Patient 3 (Ectopic LV Beats)**

A 64-year-old male received a permanent transvenous unipolar R-inhibited (Cordis Omni-Stanicor) pacemaker with RV apical electrode placement for persistent sinus bradycardia. Shortly after implantation the patient continued to have ectopic ventricular beats with RBBB pattern in V1 indicative of late activation of the right ventricle (fig. 4). QRS complexes 3 and 8 were normally sensed, whereas beats 10, 13, 17, 19, and 21 of similar configuration showed pacemaker spikes from 25 to 80 msec after QRS onset. Again, there was no other clinical evidence to suggest pacemaker malfunction. Closer analysis of the ectopic beats revealed that normally sensed complexes 3 and 8 had substantially shorter

![Figure 3](image-url)  
*Figure 3*  
Rhythm strips of lead V2 from a patient with a R-inhibited bipolar pacemaker. There was proper sensing of early conducted RBBB beats 4, 6, 10, and 11. However, late occurring RBBB-QRS complexes 5, 7, and 12 were not sensed as indicated by the appearance of the pacemaker spike within the QRS.
coupling intervals of 740 and 720 msec, respectively, compared to 800, 780, 800, and 800 msec of unsensed complexes 10, 13, 17, and 21, respectively. The beats in which the pacemaker failed to sense can readily be explained on the basis of late arrival of the depolarization front at the site of the RV apical electrode from an ectopic LV beat as illustrated in figure 1B. The shorter coupling interval of QRS 3 and 8 allowed the ectopic impulse to reach the RV apex before pacemaker cycle completion, thereby inhibiting the impulse of the R-inhibited pacemaker. The longer coupling intervals of QRS 10, 13, 17, 19, and 21 further delayed arrival of the ectopic impulse at the RV apex, by which time the pacemaker cycle was completed with pacemaker discharge into the QRS.

**Patient 4 (LV Rhythm)**

A 46-year-old male, following prosthetic replacement of a stenotic bicuspid aortic valve, developed complete heart block with escape ventricular rhythm characterized by RBBB-QRS pattern. Intraoperatively, a RV epicardial unipolar electrode was implanted and attached to a temporary pacemaker (Medtronic 5880A). The pacemaker rate was identical to that of the patient’s escape rhythm and there were frequent periods of transition alternating from paced rhythm to idioventricular rhythm (fig. 5). During some periods of idioventricular rhythm, the pacemaker spike was seen to fall within the QRS complex. Figure 5 shows a continuous recording of lead V₁ of which complexes 1 through 4 were pacemaker induced. During idioventricular rhythm (bottom strip), beats 5, 6, and 7 show the pacemaker spike within the QRS complex 28, 30, and 55 msec, respectively, after QRS onset. QRS 8 was normally sensed; hence, the pacemaker discharge was inhibited. This patient demonstrated failure to sense in still another setting, that of idioventricular rhythm causing delayed activation of the myocardium at the site of the pacemaker electrode, as explained in figure 1B.

In addition, this recording depicts fusion beats resulting from combined activation of the ventricles by the ectopic LV focus and the RV pacemaker electrode. Beat 1 was purely pacemaker-induced whereas beat 8 is entirely of spontaneous ectopic origin. Beats 3 through 6 are fusion beats in which the right ventricle was stimulated by the pacemaker: the right ventricle was activated by the pacemaker because the LV ectopic impulse had not had sufficient time to depolarize this site. In unsensed beats, this fusion phenomenon is likely to occur with pacemaker discharge occurring very shortly after the QRS onset as in beats 5 and 6, in contrast to beat 7 which is not a fusion beat. In beat 7, the ectopic LV impulse has depolarized the RV muscle around the pacemaker electrode simultaneous with the pacemaker discharge.

It is important to differentiate failure to sense due to intraventricular conduction disorders described in the previous four patients from failure to sense consequent to pacemaker malfunction. An example of failure to sense due to pacemaker malfunction is shown in a 67-year-old male with RBBB plus LAH who received a permanent transvenous R-inhibited pacemaker (Cordis, Omni-Stanico) because of syncopal episodes (fig. 6). Evaluation after implantation revealed an occasional pacemaker spike occurring in the QRS (beats 3 and 5, top strip) which superficially suggested failure to sense because of the presence of RBBB. However, long periods of observation revealed some pacemaker discharges to occur within the ST-T segments, QRS 14 and 18 (bottom strip). The majority of conducted beats, however, were normally sensed. Otherwise, the pacing function was intact and there was no evidence of cardiac perforation by the electrode. Intracardiac QRS voltage at the time of pacemaker implantation was satisfactory (5.4 mV). Serial follow-up over a four month period revealed no additional abnormalities and there have been no
episodes of ventricular tachycardia induced by erratic sensing.

R-Inhibited Pacemakers with LV Electrodes

**Patient 5 (NSR with LBBB)**

Just as ectopic LV beats and conducted beats with RBBB configuration can produce failure of the RV apical sensing electrodes to sense, likewise conducted beats with LBBB patterns can be sensed late in patients with LV epicardial electrodes. In a patient with a LV pacemaker, the pacemaker will discharge into the QRS if the pacemaker cycle is completed before the impulse reaches the LV sensing electrode. This phenomenon was documented in a 63-year-old male with idiopathic LBBB and syncopal episodes due to periods of intermittent complete heart block who received a unipolar R-inhibited pacemaker (Medtronic 5961) with an epicardial LV electrode. Follow-up examinations revealed normal pacing and sensing functions. However, there were occasional periods during which the pacemaker spikes fell within several of the sinus beats with LBBB pattern. Figure 7, taken at paper speed 50 mm/sec, showed in sequence:

**Figure 6**

Two lead V1, rhythm strips from a patient with conducted RBBB beats and a R-inhibited unipolar pacemaker (Cordis Omni-Stanisor) with RV unipolar endocardial electrode. Complexes 3 and 5 revealed pacemaker spikes within the QRS which appeared to be caused by RBBB. However, the bottom strip shows pacemaker spikes well beyond QRS complexes 14 and 18, thereby indicating that erratic sensing was probably the result of malfunction of the pacemaker system itself.

pacemaker-induced beats 1 and 2, a fusion beat 3, and sinus conducted QRS beats 4, 5, and 6 with LBBB. A pacemaker spike was seen 40 msec after the onset of QRS 4. The occurrence of this pacemaker discharge in QRS 4 is explained by LBBB which causes the conducted impulse to arrive at the LV electrode too late to prevent pacemaker discharge.

**R-Triggered Pacemakers**

R-triggered pacemakers afford a unique opportunity for studying the time of arrival of ventricular depolarization at the site of the sensing electrode. Figure 8 was obtained from a 71-year-old patient with RBBB who received an R-triggered pacemaker (Cordis, Omni-Ectocor) because of syncopal episodes. The top strip began with two paced beats followed by conducted beats with RBBB. These conducted impulses were normally sensed as indicated by the discharge of the R-triggered pacemaker 50 msec after QRS onset. In this case, the pacemaker discharge served as a marker for the moment QRS sensing was achieved and was seen better at fast paper speed of 50 mm/sec (bottom strip). This observation lends further support to the fact that in the presence of RBBB, RV pacemaker electrodes sense ventricular depolarization late after QRS onset.

**Patient 6 (Ectopic LV Beats)**

R-triggered pacemakers with RV electrodes sense ectopic LV beats considerably after QRS onset for the same reason as R-inhibited pacemakers (figure 1B), namely late arrival of the ectopic impulse at the electrode site. This delayed sensing is demonstrated in figure 9, beats 3 and 6 (top strip). Lack of sensing by an R-triggered pacemaker can be assumed if the

**Figure 7**

Lead I (50 mm/sec) from a patient with conducted LBBB beats and an LV epicardial electrode with a unipolar R-inhibited pacemaker (Medtronic 5961). QRS 1 and 2 were pacemaker induced; QRS 3, a fusion beat; and beats 4, 5, and 6, sinus beats with LBBB. The pacemaker spike seen 40 msec after QRS onset in complex 4 indicated no pacemaker sensing due to LBBB.

**Figure 8**

Recordings of rhythm strips demonstrating an R-triggered pacemaker in a patient with sinus conducted beats with RBBB. In the top strip sequentially are shown two pacemaker-induced beats, 8 consecutive conducted sinus beats followed by one conducted ectopic atrial beat. In the bottom strip (50 mm/sec), three pacemaker-induced beats are followed by three sensed sinus beats. The pacemaker spike in the conducted beats was delayed until 50 msec after QRS onset as shown. These recordings illustrate the delayed sensing encountered in RBBB as objectively demonstrated by the R-triggered discharge. R-S = QRS to pacemaker spike interval.
pacemaker spike falls within the QRS while its discharge interval from the immediately previous pacemaker discharge is identical to the spontaneous rate. In LV ectopic beats 3, 6, and 13, the pacemaker discharge occurs within the QRS indicating delayed sensing. That these beats have been sensed is proven by the pacemaker discharge within these beats having been ‘‘pulled in’’ closer to the discharge of the immediately preceding pacemaker-induced beats 2, 5, and 12, respectively. In contrast, in beat 12, the basic pacemaker discharge interval (840 msec) was unchanged indicating a failure to sense.

Discussion

Inability of a ventricular demand pacemaker to sense a conducted sinus QRS or an ectopic ventricular beat is considered an early sign of pacemaker malfunction, necessitating replacement of the pulse generator. However, a normally functioning pacemaker may exhibit lack of sensing under certain conditions such as inadequate voltage of inherent ventricular depolarization, improper position of sensing electrodes, the signal appearing in the pacemaker refractory period, and temporary reversion of the pacemaker to a fixed rate mode of operation due to electromagnetic interference. None of these causes were operative in the lack of sensing described in patients presented herein. All patients comprising this report exhibited the common denominator of wide QRS complexes in all instances of unsensed beats, either due to bundle branch block with NSR or due to ectopic ventricular systoles.

It has been documented in this report that non-competitive pacemakers with ventricular sensing electrodes detect depolarizations late if there is ipsilateral bundle branch block during sinus rhythm (figs. 1A, 3, and 8) or in instances of ectopic depolarization arising from the contralateral ventricle (figs. 1B and 9). If, in such complexes of RBBB configuration, the impulse reaches the RV apex after the completion of the pacemaker automatic cycle, it will allow the pacemaker discharge to occur within the QRS complex and thereby mimic loss of sensing due to pacemaker malfunction. This phenomenon was observed commonly in patients with permanent pacemakers (table 1), as well as in temporary units, and should be considered before loss of sensing is concluded to be a manifestation of pacemaker malfunction.

In the presentation of our preliminary communication on the lack of noncompetitive pacemaker sensing due to intraventricular conduction defects with RV electrodes, it was predicted that this phenomenon should also occur in the presence of conducted LBBB beats or in instances of ectopic RV beats in patients with LV epicardial electrodes. Indeed, we have been able to document this phenomenon in a patient with LBBB and a LV epicardial electrode (fig. 6). Further, a second such example was encountered in a patient with conducted LBBB beats whose transvenous endocardial electrode in the coronary sinus was seen lying over the posterior wall of the left ventricle resulting in pacing and sensing from the left ventricle.

In addition to lack of pacemaker sensing due to intraventricular conduction defects described for the first time in this report and the other causes of undersensing mentioned above, another cause of undersensing by demand pacemakers is delayed rate of rise or dv/dt of inherent ventricular depolarizations. A noncompetitive pacemaker requires an adequate dv/dt along with sufficient total amplitude for proper sensing of ventricular complexes. In normal circumstances, the dv/dt and total amplitude of P and T waves are inadequate to interfere with the pacemaker sensing mechanism. The QRS, on the other hand, nearly always possesses adequate dv/dt and it is decrease in total QRS voltage that is usually responsible for QRS undersensing when the inherent ventricular depolarization is at fault. However, in the present group of patients the intracardiac electrocardiogram recorded from the sensing electrode of the pacemaker at the time of implantation was more than adequate in magnitude (4.5 to 11 mV). However, decrease in dv/dt of a ventricular complex may occur in the presence of severely diseased myocardium, drug toxicity due to quinidine or potassium, and in preterminal states. It is considered highly unlikely that decreased dv/dt in the present patients with bun-

![Figure 9](https://circ.ahajournals.org/)

**Figure 9**

Interrupted V, rhythm strips from a patient with an R-triggered pacemaker. The normal pacemaker cycle is 840 msec as between beats 1 and 2. Ectopic LV beats 3 and 6 in the top strips trigger the pacemaker discharge prematurely indicating adequate but delayed sensing. In contrast, LV ectopic beat 12 of similar configuration (bottom strip) did not shorten the normal pacemaker spike-to-spike interval (S-S), thereby indicating that this QRS was not sensed by the pacemaker due to intraventricular conduction delay.
dle branch block contributes to the phenomenon of undersensing due to intraventricular conduction disorders, since the majority of QRS complexes with bundle branch block were sensed, and it is impossible that the few complexes of similar configuration were not sensed because of diminished dv/dt. Likewise, ectopic ventricular beats arising from the ipsilateral ventricle were sensed while only occasional complexes from the contralateral ventricle were not sensed. Again it would be unlikely that the dv/dt of ectopic complexes of similar configuration would differ from beat-to-beat or would differ substantially from that arising in the ipsilateral ventricle. Furthermore, the complexes in which sensing failed to occur were always wide and the absence of sensing only occurred in the initial portion of the QRS. These observations are consistent with the presented concept and evidence of late arrival of the impulse at the RV apex in the presence of RBBB.

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