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Electrocardiographic Follow-Up of Biventricular Pacemakers

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Multisite pacing for the treatment of heart failure has added a new dimension to the electrocardiographic evaluation of device function. During left ventricular (LV) pacing from the appropriate site in the coronary venous system, a correctly positioned lead V1 registers a right bundle branch block pattern with few exceptions. During biventricular stimulation associated with right ventricular (RV) apical pacing, the QRS is often positive in lead V1. The frontal plane QRS axis is usually in the right superior quadrant and occasionally in the left superior quadrant. Barring incorrect placement of lead V1 (too high on the chest), lack of LV capture, LV lead displacement or marked latency (exit block or delay from the stimulation site), ventricular fusion with the spontaneous QRS complex, a negative QRS complex in lead V1 during biventricular pacing involving the RV apex probably reflects different activation of an heterogeneous biventricular substrate (ischemia, scar, His-Purkinje participation in view of the varying patterns of LV activation in spontaneous left bundle branch block) and does not necessarily indicate a poor (electrical or mechanical) contribution from LV stimulation. In this situation, it is imperative to rule out the presence of coronary venous pacing via the middle cardiac vein or even unintended placement of two leads in the RV. During biventricular pacing with the RV lead in the outflow tract, the paced QRS in lead V1 is often negative and the frontal plane paced QRS axis is often directed to the right inferior quadrant (right axis deviation).

In patients with sinus rhythm and a relatively short PR interval, ventricular fusion with competing native conduction during biventricular pacing may cause misinterpretation of the ECG because narrowing of the paced QRS complex simulates appropriate biventricular capture. This represents a common pitfall in device follow-up. Elimination of ventricular fusion by shortening the AV delay, is often associated with clinical improvement. Anodal stimulation may complicate threshold testing and should not be misinterpreted as pacemaker malfunction.

One must be cognizant of the various disturbances that can disrupt 1:1 atrial tracking and cause loss of ventricular resynchronization. (1) Upper rate response. The upper rate response of biventricular pacemakers differs from the traditional Wenckebach upper rate response of conventional antibradycardia pacemakers because heart failure patients generally do not have sinus bradycardia or AV junctional conduction delay. The programmed upper rate should be sufficiently fast to avoid loss of resynchronization in situations associated with sinus tachycardia. (2) Below the programmed upper rate. This may be caused by a variety of events (especially ventricular premature complexes and favored by the presence of first-degree AV block) that alter the timing of sensed and paced events. In such cases, atrial events become trapped into the postventricular atrial refractory period at atrial rates below the programmed upper rate in the presence of spontaneous AV conduction. Algorithms are available to restore resynchronization by automatic temporary abbreviation of the postventricular atrial refractory period.

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The advent of multisite pacing for the treatment of congestive heart failure (CHF) has added a new dimension to the electrocardiographic evaluation of pacemaker function.¹⁻¹³ For many years,

the electrocardiogram (ECG) taken during pacemaker follow-up has often consisted of only single-lead recordings displayed by "high-tech" ECG/marker systems of pacemaker programmers.

The "low-tech" paced 12-lead ECG was relegated to a minor role and often neglected.¹⁴ However, biventricular (BV) pacing has generated a well-deserved renaissance of the 12-lead paced ECG that has become an indispensable tool in the evaluation of cardiac resynchronization.

NORMAL QRS PATTERNS DURING RIGHT VENTRICULAR PACING

Pacing from the right ventricle (RV) regardless of site almost always produces a left bundle branch block (LBBB) pattern in the precordial leads (defined as the absence of a positive complex in lead V1 recorded in the 4th or 5th intercostal space).^{15,16} Pacing from the RV apex produces negative paced QRS complexes in the inferior leads (II, III, and aVF) simply because the activation begins in the inferior part of the heart and travels superiorly away from the inferior leads. The mean paced QRS frontal plane axis is superior either in the left or less commonly in the right superior quadrant. Displacement of the electrode from the RV apex toward the RV outflow tract (OT) shifts the frontal plane paced QRS axis to the left inferior quadrant, a site considered normal for spontaneous QRS complexes. The inferior leads become positive. The axis then shifts to the right inferior quadrant as the stimulation site moves more superiorly toward the pulmonary valve.

With the backdrop of dominant R waves in the inferior leads, RVOT pacing may generate qR, QR, or Qr complexes in leads I and aVL. Occasionally with slight displacement of the pacing lead from RV apex to the RV outflow tract, leads I and aVL may register a qR complex in conjunction with the typical negative complexes of RV apical stimulation in the inferior leads. This qR pattern must not be interpreted as a sign of myocardial infarction.¹⁷ RV pacing from any site never produces qR complexes in V5 and V6 in the absence of myocardial infarction or ventricular fusion with a spontaneous QRS complex. A qR or Qr (but not QS) complex in the precordial or inferior leads is always abnormal in the absence of ventricular fusion. In contrast, a q-wave is common in the lateral leads (I, aVL, V5, and V6) during uncomplicated biventricular pacing (using the RV apex), and should not be interpreted as representing myocardial infarction or RVOT displacement of an RV apical lead. Finally, lead I (but not aVL) rarely displays a qR complex in uncomplicated RV apical pacing.

Table 1. Causes of a Dominant R wave during Conventional Ventricular Pacing

Ventricular Fusion
<ul style="list-style-type: none">• Pacing in the myocardial relative refractory period• LV pacing from the coronary venous system• LV endocardial or epicardial pacing• Lead perforation of RV or ventricular septum with left ventricular stimulation• Uncomplicated RV pacing

DOMINANT R WAVE OF THE PACED QRS COMPLEX DURING CONVENTIONAL PACING

A dominant R wave in V1 during ventricular pacing has been called a right bundle branch block (RBBB) pattern of depolarization, but this terminology is potentially misleading because this pattern is often not related to RV activation delay^{14,16,18–20} (Table 1). A dominant R wave of a paced ventricular beat in the right precordial leads occurs in approximately 8–10% of patients with uncomplicated RV apical pacing. The position of precordial leads V1 and V2 should be checked because a dominant R wave can be sometimes recorded at the level of the third intercostal space during uncomplicated RV apical pacing. The pacing lead is almost certainly in the RV (apex or distal septal site) if V1 and V2 are negative when recorded one space lower (5th intercostal space).^{15,18} A dominant R wave may not be eliminated at the level of the 5th interspace if RV pacing originates from the midseptal region.²¹ Furthermore, the "RBBB" pattern from pacing RV sites results in a precordial vector change from positive to negative by lead V3 in the precordial sequence. Therefore, a tall R wave in V3 and V4 signifies that a pacemaker lead is not in the RV after excluding ventricular fusion from spontaneous AV conduction.²¹ The ECG pattern with a truly posterior RV lead has not been systematically investigated as a potential cause of a tall R wave in V1 during pacing.

Significance of a Small r Wave in Lead V1 During Uncomplicated RV Pacing

A small early (r) wave (sometimes wide) may occasionally occur in lead V1 during uncomplicated RV pacing. There is no evidence that this r-wave

Figure 1. Twelve-lead ECG showing LV pacing from the coronary venous system. There is typical RBBB pattern and right axis deviation. Note the dominant R wave from V1 to V6 consistent with basal LV pacing. LV pacing shown in all the figures was performed from the coronary venous system.

represents a conduction abnormality at the RV exit site. Furthermore, an initial r wave during biventricular pacing does not predict initial left ventricular (LV) activation.¹

LEFT VENTRICULAR ENDOCARDIAL PACING

Unintended passage of a pacing lead into the LV occurs via the subclavian artery or an atrial septal defect (or foramen ovale). The access sites to the LV can be easily identified by the typical widespread RBBB pattern in the precordial leads during pacing, standard chest radiographs, and echocardiography.^{14,22-26} As a rule with a RBBB configuration (tall R wave in V3 and beyond), the frontal plane axis cannot differentiate precisely an endocardial LV site from one in the coronary venous system.

ECG PATTERNS RECORDED DURING LV PACING FROM THE CORONARY VENOUS SYSTEM

An RBBB pattern in correctly positioned lead V1 occurs with few exceptions^{1,2,9,11,15,26,27} (Figs. 1

and 2). With apical sites, leads V4–V6 are typically negative. With basal locations, leads V4–V6 are usually positive as with the concordant positive R waves during overt pre-excitation in left-sided accessory pathway conduction in the Wolff-Parkinson-White syndrome.¹

Middle Cardiac Vein

Pacing usually produces an RBBB pattern but a LBBB configuration may also occur. Rarely the pattern alternates from RBBB to LBBB.¹⁵ The LBBB configuration may represent preferential entry into the RV from the septum perhaps on the basis of local pathology such as a myocardial infarction scar. The activation from the pacing site travels away from the inferior surface of the LV and generates negative QRS complexes in leads II, III, and aVF with the frontal plane axis usually in the left superior quadrant.

Great Cardiac Vein

Little data are available from this site.²⁷ The ECG usually shows a RBBB pattern in lead V1 with axis

A

B

Figure 2. (A) Twelve-lead ECG with leads V1 and V2 recorded during LV pacing at the level of the second intercostal space in a thin patient with an elongated chest. There is no dominant R wave in lead V1. The ECG during biventricular pacing also failed to show a dominant R wave in V1 at the level of the second intercostals space. (B) The dominant R-wave in V1 becomes evident only when lead V1 is recorded in the 4th intercostal space. The R wave in V1 recorded in the 4th intercostal space during biventricular pacing also became dominant.

deviation to the right inferior quadrant as with LV pacing from the lateral or posterior coronary veins especially if one of the lateral branches of the great cardiac vein (anterior interventricular vein) is used for pacing.

Lateral and Posterior Veins

LV pacing from the traditional site for resynchronization produces a RBBB in most cases. The frontal plane axis often points to the right inferior

quadrant (right axis deviation) and less commonly to the right superior quadrant. In an occasional patient with uncomplicated LV pacing with a typical RBBB pattern in lead V1, the axis may point to the left inferior or left superior quadrant. The reasons for these unusual axis locations are unclear. Lead V1 rarely shows a negative QRS complex during uncomplicated LV pacing. This may be due to incorrect ECG lead placement (lead V1 too high), middle cardiac vein location, or an undefined mechanism requiring elucidation.

ECG PATTERNS AND FOLLOW-UP OF BIVENTRICULAR PACEMAKERS^{11,28-30}

A baseline 12-lead ECG should be recorded at the time of implantation during assessment of the independent capture thresholds of the RV and LV to identify the specific morphology of the paced QRS complexes in a multiplicity of leads.¹⁴ This requires having the patient connected to a multichannel 12-lead ECG during the implantation procedure. A total of four 12-lead ECGs are required. (1) Intrinsic rhythm and QRS complex prior to any pacing. (2) Paced QRS associated with RV pacing. (3) Paced QRS associated with LV pacing, and (4) Paced QRS associated with biventricular pacing (Fig. 3A). The four tracings should be examined to identify the lead configuration that best demonstrates a discernible and obvious difference between the four pacing states (inhibited, RV only, LV only, and biventricular). This ECG lead should then be used as the surface monitoring lead for subsequent evaluations. Loss of capture in one ventricle will cause a change in the morphology of ventricular paced beats in the 12-lead ECG similar to that of either single-chamber RV pacing or single-chamber LV pacing. A shift in the frontal plane axis may be useful to corroborate loss of capture in one of the ventricles.^{1,10,11} If both the native QRS and the biventricular paced complex are relatively narrow, then a widening of the paced QRS complex will identify loss of capture in one chamber with effectual capture in the other.

First-Generation Devices with a Common Output

In questionable cases involving first-generation devices with a common ventricular output, loss of capture requires monitoring the ECG with

telemetered markers and the intracardiac ventricular electrogram (EGM).⁹⁻¹¹ When there is intact capture in both the RV and LV, the evoked response on the ventricular electrogram will show a monophasic complex in contrast to the two distinct depolarizations during spontaneous AV conduction if the native QRS is wide (LBBB or left intraventricular conduction delay) (Fig. 4). With loss of capture in one of the ventricles, ventricular pacing will persist in the contralateral ventricle. The impulse will then be conducted via the native pathways to the other ventricle in a manner identical to traditional single ventricular pacing systems. During threshold testing by gradually reducing the output, LV capture is commonly lost before RV capture. In such a case, if there is a delayed left intraventricular conduction delay, the ventricular electrogram will change from a monophasic complex to two discrete complexes similar but not identical to the two components registered in the ventricular electrogram of spontaneously conducted (LBBB) QRS complexes (Figs. 4 and 5). Two discrete electrographic deflections will not occur in the spontaneous ventricular electrogram of patients with underlying RBBB or lesser degrees of LBBB. Rarely, when the pacing thresholds of the RV and LV are identical, testing should be started at a high output. The first transition will be to a spontaneous QRS complex if there is no anodal stimulation (discussed later). The presence of biventricular pacing must then be corroborated by the expected depolarization pattern in the 12-lead ECG. Fortunately, the threshold testing procedure has become simpler in contemporary devices with programmability of separate output for the RV and LV.

Paced QRS Duration and Status of Mechanical Ventricular Resynchronization

The paced QRS during biventricular pacing is often narrower than that of monochamber pacing. Barring fusion beats, a narrower QRS implies depolarization from the RV and LV. Thus, measurement of QRS duration during follow-up is helpful in the analysis of appropriate biventricular capture and fusion with the spontaneous QRS.^{1,2,11} If the biventricular ECG is virtually similar to that recorded with RV or LV pacing alone and no cause is found, one should not automatically conclude that one of the leads does not contribute to biventricular depolarization without a detailed evaluation of the

Figure 3. (A) Typical 12-lead ECG of biventricular pacing with the RV lead at the apex. Note the dominant R wave in lead V1 and the frontal plane axis in the right superior quadrant. (B) Asynchronous biventricular pacing (VOO) in a different patient with the RV lead at the apex. The arrows point to QRS complexes without a preceding P wave. These complexes therefore do not represent ventricular fusion with the spontaneous conducted QRS complex. There is a typical q wave in lead I and lead V6 shows a QR complex during pure biventricular pacing. (C) Biventricular DDD pacing in the same patient as in panel (B). With appropriate programming of the AV delay, the configuration of the paced QRS complexes becomes identical to that in panel (B) thereby ruling out fusion with the spontaneous conducted QRS complex. A dominant R wave in V1 is not always the rule during biventricular pacing with the RV lead at the apex. The frontal plane axis points to the left superior quadrant rather than the right superior quadrant often associated with this arrangement (reproduced from Garrigue, Barold, and Clémenty¹¹ with permission).

Figure 3. Continued.

Figure 4. Simultaneous recording of the ECG and telemetered ventricular electrogram in a patient with left bundle branch block and a biventricular pacemaker with a common ventricular sensing channel. The ventricular electrogram displays a monophasic pattern during biventricular capture. With loss of LV pacing, the ventricular electrogram shows a late deflection, which represents delayed LV activation through ordinary myocardium. AS = atrial-sensed event; VP = ventricular paced event (reproduced from Garrigue, Barold, and Clémenty¹¹ with permission).

Figure 5. Simultaneous recording of the ECG and telemetered ventricular electrogram during spontaneous rhythm in a patient with left bundle branch block and a biventricular pacemaker with a common ventricular sensing channel. The ventricular electrogram shows two discrete components corresponding to RV and delayed LV activation. The pattern is different from the one recorded in Figure 4 during RV pacing with loss of LV capture. AS = atrial-sensed event; VS = ventricular-sensed event (reproduced from Garrigue, Barold, and Clémenty¹¹ with permission).

pacing system. So far, evaluation of the overall ECG patterns of biventricular pacing has focused on simultaneous RV and LV stimulation. The electrocardiographic consequences of temporally different RV and LV activation with programmable V-V timing in the most recent biventricular devices have not yet been studied.^{31,32}

Chronic studies have shown that the degree of narrowing of the paced QRS duration is a poor predictor of the cardiac resynchronization response.^{7,11,33} In other words, the degree of QRS narrowing or its absence does not correlate with the long-term hemodynamic benefit of biventricular pacing^{7,11,33} because the paced QRS does not reflect the underlying level of mechanical dyssynchrony. In this respect, some patients with monochamber LV pacing exhibit an equal or superior degree of

mechanical resynchronization compared to biventricular pacing despite a very wide paced QRS complex.^{7,11,34}

Usefulness of the Frontal Plane Axis of the Paced QRS Complex

Table 2 shows the importance of the frontal plane axis of the paced QRS complex in determining the arrangement of pacing during testing of biventricular pacemakers.⁹⁻¹¹

Biventricular Pacing with the RV Lead Located at the Apex

The shift in the frontal plane QRS axis during programming the ventricular output is helpful in

Table 2. Change in Frontal Plane Axis of Paced QRS When Programming from Biventricular to LV and RV Pacing

Pacing Site	QRS in Lead I	QRS in Lead III	Axis Shift
BiV → RV	Greater positivity	Greater negativity*	Clockwise
BiV → LV	Greater negativity	Greater positivity	Counterclockwise

*QRS in lead III is more negative than in lead II.
BiV = biventricular; RV = right ventricle; LV = left ventricle.

Figure 6. Biventricular pacing with the RV lead in the outflow tract. There was a very prominent R wave in lead V1 during monochamber LV pacing. Note the typical absence of a dominant R wave in lead V1 and the presence of right axis deviation, an uncommon finding during biventricular pacing with the RV lead at the apex. The presence of ventricular fusion with the spontaneous conducted QRS complex was ruled out. Compare this tracing with the one in Figure 3A.

determining the site of ventricular stimulation in patients with first-generation devices without separately programmable RV and LV outputs (Table 2). The frontal plane QRS axis usually moves superiorly from the left (RV apical pacing) to the right superior quadrant (biventricular pacing) in an anti-clockwise fashion if the ventricular mass is predominantly depolarized by the LV pacing lead^{1,10,11} (Fig. 3A). The frontal plane axis may occasionally reside in the left rather than the right superior quadrant during biventricular pacing.

The QRS is often positive in lead V1 during biventricular pacing when the RV is paced from the apex. Barring incorrect placement of lead V1 (too high on the chest: as in Fig. 2A), lack of LV capture, LV lead displacement, or marked latency (exit block or delay from the stimulation site, an important but poorly studied phenomenon with LV pacing) associated with LV stimulation and ventricular fusion, a negative QRS complex in lead V1 probably reflects different activation of an heterogeneous biventricular substrate (ischemia, scar, His-Purkinje participation in view of the varying patterns of LV activation in spontaneous LBBB, etc.) and does not necessarily indicate a poor (electrical or mechanical) contribution from LV stimulation. In this situation, it is imperative to rule out the presence of coronary venous pacing via the middle cardiac vein¹⁶

or even unintended placement of two leads in the RV.³⁵

Biventricular Pacing with the RV Lead in the Outflow Tract

In our limited experience, we have found that during biventricular pacing with the RV lead in the outflow tract, the paced QRS in lead V1 is often negative and the frontal plane paced QRS axis is often directed to the right inferior quadrant (right axis deviation) (Fig. 6). Further studies are required to confirm these preliminary findings and to determine the significance of these ECG patterns of biventricular pacing according to the RV pacing site.

Q or q and QS Configuration in Lead 1

Georger et al.²⁹ observed a q wave in lead I in 17 of 18 patients during biventricular pacing (Fig. 3B & C). As indicated previously, a q wave in lead I during uncomplicated RV apical pacing is rare and these workers observed it in only 1 patient. Loss of the q-wave in lead I was 100% predictive of loss of LV capture.²⁹ It therefore appears that analysis of the Q or q wave or a QS complex in lead I may be a reliable way to assess LV capture during biventricular pacing.

Ventricular Fusion Beats with Native Conduction

In patients with sinus rhythm and a relatively short PR interval, ventricular fusion with competing native conduction during biventricular pacing may cause misinterpretation of the ECG, and a common pitfall in device follow-up¹¹ (Fig. 7). QRS shortening mandates exclusion of ventricular fusion with the spontaneous QRS complex, especially in the setting of a relatively short PR interval. The presence of ventricular fusion should be ruled out by observing the paced QRS morphology during progressive shortening of the AS-VP interval in the VDD mode or the AP-VP interval in the DDD mode. The AS-VP interval should be programmed (with rate-adaptive function) to ensure pure biventricular pacing under circumstances that might shorten the PR interval, such as increased circulating catecholamines.

Long-Term ECG Changes

Many studies have shown that the paced QRS duration does not vary over time as long as the LV pacing lead does not move from its initial site.^{7,11,36} Yet, surface ECGs should be performed periodically because the LV lead may become displaced into a collateral branch of the coronary sinus. Dislodgement of the LV lead may result in loss of LV capture with the ECG showing an RV pacing QRS pattern with an increased QRS duration and superior axis deviation. Ricci et al.³⁶ suggested that variation of the QRS duration over time may play a determinant role if correlated with remodeling of the ventricles by echocardiography. Finally, the underlying spontaneous ECG should be exposed periodically to confirm the presence of a LBBB type of intraventricular conduction abnormality. In this respect, turning off the pacemaker could potentially improve LV function in patients who have lost their

Figure 7. (A) Narrowing of the paced QRS complex (well seen in V1) due to ventricular fusion with the spontaneous conducted QRS complex. This ECG was the initial recording taken upon arrival to the pacemaker follow-up center. AV delay = 100 ms. The marked narrowing of the QRS complex in lead V1 suggests ventricular fusion rather than QRS narrowing from satisfactory biventricular pacing. (B) The ECG taken 15 minutes later (same parameters and AV delay) when the patient was more relaxed shows no evidence of ventricular fusion. (C) Immediately after the tracing in panel (B), ventricular fusion was demonstrated only when the AV delay was lengthened to 130 ms. The serial tracings illustrate the dynamic nature of AV conduction (emotion, catecholamines, etc.) and the importance of appropriate programming of the AV delay to prevent ventricular fusion with the spontaneous conducted QRS complex.

B

C

Figure 7. Continued.

Figure 8. Anodal capture during first-generation biventricular pacing. There is anodal capture on the left (three pacing sites). It disappears on the right (two pacing sites) with reduction of the common ventricular output revealing pure biventricular pacing (reproduced from Garrigue, Barold, and Clémenty¹¹ with permission).

intraventricular conduction defect through ventricular remodeling.

ANODAL STIMULATION

Although anodal capture may occur with high output traditional RV pacing, this phenomenon is almost always not discernible electrocardiographically. Biventricular pacing systems generally utilize a unipolar lead for LV pacing via a coronary vein. The tip electrode of the LV lead is the cathode and the proximal electrode of the bipolar RV lead often provides the anode for LV pacing. This arrangement creates a common anode for RV and LV pacing. A high current density (from two sources) at the common anode during biventricular pacing may cause anodal capture manifested as a paced QRS complex with a somewhat different configuration from that derived from pure biventricular pacing.³⁷⁻⁴² Anodal capture during biventricular pacing disappears by reducing the output of the pacemaker or when the device (even at high output) is programmed to a true unipolar system with the common anode on the pacemaker can. Anodal capture was recognized in first-generation transvenous biventricular pacemakers (without separately programmable RV and LV outputs) when three dis-

tinct pacing morphologies were observed exclusive of fusion with the spontaneous QRS complex: Biventricular with anodal capture (at a high output), biventricular (at a lower output), and RV (with loss of LV capture) or rarely LV (with loss of RV capture)^{41,42} (Fig. 8). Anodal capture involving the ring electrode of the bipolar RV lead can also occur in second-generation biventricular pacemakers with separately programmable ventricular outputs. Anodal stimulation may occur during biventricular pacing as with first-generation devices. However, during monochamber LV pacing at a relatively high output, the paced QRS complex becomes identical to that registered with biventricular pacing below the anodal stimulation threshold^{37,39,41,42} (Fig. 9). Occasionally, this type of anodal capture prevents electrocardiographic documentation of pure LV pacing if the LV pacing threshold is higher than that of anodal stimulation. Anodal stimulation may complicate threshold testing and should not be misinterpreted as pacemaker malfunction. Furthermore, anodal stimulation during biventricular pacing may interfere with a programmed interventricular (V-V) delay (often programmed with the LV preceding the RV) aimed at optimizing cardiac resynchronization because RV and LV^{31,32} are activated simultaneously.^{37,39,41,42} If the LV threshold

Figure 9. On the left, there is anodal pacing in the DDD mode of a second-generation biventricular pacemaker seen during monochamber LV pacing with an LV output of 3.5 V and 0.5 ms LV. The ECG pattern was identical to that recorded during biventricular pacing. On the right, anodal stimulation alternates with pure LV pacing when the LV output is slightly less than 3.5 V at 0.5 ms (reproduced from Herweg and Barold³⁸ with permission).

is not too high, appropriate programming of the pacemaker output should eliminate anodal stimulation in most cases.

UPPER RATE RESPONSE OF BIVENTRICULAR PACEMAKERS

The upper rate response of biventricular pacemakers differs from the traditional Wenckebach upper rate response of conventional antibradycardia pacemakers because CHF patients generally do not have sinus bradycardia or AV junctional conduction delay. The upper rate response exhibits two forms according to the location of the P wave in the pacemaker cycle: (1) A pre-empted Wenckebach upper rate response with AS-VS sequences and the P wave beyond the postventricular atrial

refractory period (PVARP).⁴³ (2) AR-VS sequences with the P wave sensed (but not tracked) within the PVARP.

Pre-empted Wenckebach Upper Rate Response

In a traditional Wenckebach upper rate response, a dual chamber pacemaker (where upper rate interval > total atrial refractory period (TARP)) delivers its ventricular stimulus only at the completion of the (atria-driven) upper rate interval (Fig. 10). The AV delay initiated by a sensed P wave increases progressively because the ventricular channel waits to deliver its output at the end of the upper rate interval. Eventually, a P wave falls in the PVARP, a pause occurs, and the ventricular paced

Normal upper rate response of the Wenckebach type

The pre-empted Wenckebach behavior with short sAVI

Figure 10. Top: Traditional pacemaker Wenckebach upper rate response. Bottom: Repetitive pre-empted Wenckebach upper rate response. See text for details (reproduced from Barold, Garrigue, and Israel¹² with permission).

sequence repeats itself. In patients with pacemakers implanted for CHF, the Wenckebach upper rate response (or more precisely the manifestation of upper rate > total atrial refractory period) assumes a form that is not immediately recognizable because no paced beats are evident.

In patients with normal or near normal sinus node function and AV conduction and a relatively short PVARP, a pacemaker Weckenbach upper rate response takes the form of a repetitive pre-empted process which consists of an attempted Wenckebach upper rate response with each cycle, associated with continual partial or incomplete extension of the programmed AV interval^{12,43} (Figs. 10 and 11). The conducted spontaneous QRS complex

continually occurs before completion of the upper rate interval. It is therefore sensed by the pacemaker, and ventricular pacing is pre-empted. In other words, the pacemaker cannot time out the upper rate interval and thus cannot emit a ventricular stimulus at its completion. This form of upper rate response tends to occur in patients with relatively normal AV conduction, a short programmed AV delay, a relatively slow programmed (atrial-driven) upper rate, and a sinus rate faster than the programmed (atrial-driven) upper rate (Fig. 11). It is therefore more likely to emerge on exercise or during times of distress when adrenergic tone is high. Consequently, the pre-empted Wenckebach upper rate response has become important recently

Figure 11. Stored markers showing development of a pre-empted Wenckebach upper rate response during biventricular pacing. Upper rate interval (URI) = 460 ms. (A) There is 1:1 atrial tracking and biventricular (BV) pacing with the programmed AS-VP delay. (B) When the spontaneous ventricular rate exceeds the programmed upper rate (VS-VS < URI), a pre-empted Wenckebach upper rate response supervenes. AS conducts to VS so that AS-VS becomes longer than the programmed AS-VP interval. Note that the sinus P wave is sensed beyond the postventricular atrial refractory period. AS = atrial-sensed event; VS = ventricular-sensed event; BV or VP = biventricular pacing event.

because biventricular pacemakers are now implanted in patients with CHF (or hypertrophic cardiomyopathy) where there is commonly relatively normal sinus node function and AV conduction. The occurrence of a pre-empted Wenckebach response in such patients defeats the very purpose of this type of cardiac stimulation. Because patients with CHF are susceptible to sinus tachycardia (especially during decompensation despite beta-blocker therapy), it is particularly important to program a relatively fast upper rate during biventricular pacing to avoid a pre-empted Wenckebach upper rate response with resultant loss of cardiac resynchronization manifested by the emergence of the patient's spontaneous conducted QRS in the electrocardiogram.

In summary, a pre-empted Wenckebach upper rate response has no paced events and is characterized by three features: (1) VS-VS interval < atrial-

driven upper rate interval (VS = ventricular-sensed event), (2) PR interval (AS-VS) > programmed AS-VP. The spontaneous PR interval remains relatively constant (AP = atrial paced event, AS = atrial-sensed event), and (3) there are no unsensed (or refractory sensed) P-waves as in a typical Wenckebach upper rate response in the presence of AV block.

Upper Rate Limitation with P-Wave in the PVARP

When the P wave falls within the PVARP, a device may not assume 1:1 atrial tracking immediately when the sinus rate drops below the programmed upper rate if the P wave remains in the PVARP.² The reason lies in the fact that AR-VS (spontaneous AV conduction) > programmed AS-VP interval. Therefore, the total atrial refractory

Figure 12. Diagram showing an upper rate response with the P wave falling within the postventricular atrial refractory period (PVARP) in the setting of normal AV conduction. Cardiac resynchronization (CRT) occurs with AS–VP sequences when the sinus rate is below the maximum tracking rate (MTR). When the atrial rate exceeds the MTR at point 1, the P wave falls within the PVARP (AR marker) and CRT is lost and AR–VS sequences take over with AR conducting to VS. When the sinus rate falls below the MTR at point 2, no CRT occurs because the timing cycles of the device force the continuation of AR–VS sequences. Failure of CRT at this stage results from the longer prevailing total atrial refractory period (TARP) which is equal to [(AR–VS) + PVARP] longer than the programmed TARP = [(AS–VP) + PVARP]. CRT with AS–VP sequences is restored at point 3 when the sinus interval (P–P) > [(AR–VS) + PVARP], at a sinus rate substantially lower than the MTR. AS = atrial-sensed event; VS = ventricular-sensed event; VP = biventricular paced event; AR = atrial-sensed event in the atrial refractory period of the pacemaker where tracking cannot occur.

period during AR–VS operation, (AR–VS) interval + PVARP must be longer than the programmed TARP which is the sum of (AS–VP) interval + PVARP (Fig. 12). The pacemaker will continue to operate with AR–VS cycles below the upper rate until the sinus interval drops below the duration of the (AR–VS) interval + PVARP interval thereby allowing escape of the sinus P wave out of the PVARP. Therefore, restoration of resynchronization will occur at a rate slower than the programmed upper rate. This problem is worse in patients with first-generation devices due to double counting where 1:1 atrial tracking (AS–VP pacing) will return only when the sinus interval becomes longer than [(AR–VS) interval + PVARP + ICD (ICD = interventricular conduction delay or the interval between the RV and LV electrograms both sensed by the device)].² These considerations are important in CHF patients who develop substantial increases in sinus rates with exercise or states of increased circulating catecholamines. Special algorithms based on beat to beat PVARP shortening upon sensing a P wave in the PVARP are now available in the latest devices

to promote 1:1 atrial tracking just below the upper rate (discussed later).

OPTIMAL PROGRAMMING OF BIVENTRICULAR DEVICES

Cardiac resynchronization depends on meticulous programming of the device (Table 3). The most important parameters are the AV delay, PVARP, and the maximum tracking rate.¹⁰

Table 3. Programmability of Biventricular Pacemakers

Parameter	Management
AV delay	<ol style="list-style-type: none">1. A long AV delay should not be used.2. Optimize the AS–VP delay and avoid ventricular fusion with the spontaneous conducted QRS complex.3. Program rate-adaptive (dynamic) AV delay off during temporary pacing for testing (with VDD mode slower than sinus rate to sense atrial activity).4. Program rate-adaptive AV delay for long-term pacing.
Atrial sensing and PVARP	<ol style="list-style-type: none">1. Short PVARP (aim for 250 ms). May have to use algorithms for the automatic termination of endless loop tachycardia.2. Program off the post-VPC PVARP extension.3. Automatic mode switching off in devices using a relatively long PVARP mandated by the mode switching algorithm.
Upper rate	Relatively fast upper rate so the patient does not have “breakthrough” ventricular sensing within their exercise zone. Initial upper rate of 140/min is often appropriate in the absence of myocardial ischemia during pacing at this rate.
AV conduction	<ol style="list-style-type: none">1. Use drugs that impair AV conduction to avoid ventricular fusion or double counting in devices with a common sensing channel.2. Consider ablation of the AV junction in refractory double counting (common sensing channel) or patients with a long PR interval difficult to manage.

Table 4. Causes of Double Counting in Devices with Common Sensing

1. Loss of tracking of sinus rhythm. This includes sinus tachycardia above programmed upper rate and sinus P waves buried in a relatively long postventricular atrial refractory period (especially with activation of the automatic postventricular atrial refractory extension by a pacemaker defined ventricular extrasystole). Far-field sensing of left atrial activity by the LV lead, and near-field sensing of the T-wave can also induce double counting of the ventricular electrogram
 2. Double-counting of the QRS during supraventricular tachyarrhythmias (with intrinsic AV conduction) at a rate below the cut-off point
- Double-counting of QRS during ventricular tachycardia at rates below the cut-off point (resulting in detection of ventricular fibrillation)
- The above diagnosis of double counting of the ventricular electrogram requires exclusion of (a) displacement of the RV lead toward the tricuspid valve with far-field RV sensing of atrial activity and (b) oversensing of diaphragmatic myopotentials by using testing with deep respiration, coughing, laughing, and the Valsalva maneuver

SIMULTANEOUS SENSING OF THE VENTRICULAR ELECTROGRAM FROM BOTH VENTRICLES

First-generation biventricular pacemakers utilized a parallel dual cathodal system where pacing and sensing occur simultaneously in the two ventricles. Such systems (using a conventional DDD pacemaker and a Y-adapter) are still being implanted in various countries because of cost considerations. The common sensing channel predisposes the pacemaker to double sensing of the ventricular electrogram^{12,44} (Table 4). Double counting of the QRS complex usually involves the conducted QRS complex (as most patients have left bundle branch block and relatively normal AV conduction).²⁻⁷ Less commonly, double counting of the ventricular complex occurs when there is a loss of LV capture with preservation of RV pacing. Both situations produce temporal separation of the RV and LV electrograms. The degree of separation depends on the severity of the interventricular conduction delay and the location of the electrodes. During AV synchrony, a device can sense the LV electrogram only if it ex-

tends beyond the relatively short ventricular blanking period initiated by the prior detection of the RV EGM. With ventricular rhythms, the LV EGM may precede that from the RV. The consequences may be serious and include ventricular inhibition (with denial of beneficial resynchronization) and inappropriate therapy including shocks in the case of biventricular ICDs (excluding double counting from lack of LV capture)⁴⁴⁻⁵¹ (Fig. 13). The diagnosis is important because double counting can often be corrected by appropriate programming and therapy.

Causes of Double Counting of the Ventricular Electrogram

Barring isolated loss of LV pacing, Table 4 outlines the possible causes of double counting of the ventricular EGM in systems with simultaneous sensing from the RV and LV in the presence of an undisplaced RV lead. Double counting of the RV electrogram is uncommon with sensing only from the RV. In the case of biventricular pacemakers, the ventricular blanking period (after ventricular sensing) if programmable could be lengthened to contain the entire ventricular electrogram. This option is generally not available in biventricular ICDs where a long ventricular blanking period might promote undersensing of ventricular tachyarrhythmias. A new algorithm based on programming an interventricular refractory period (IRP) (Medtronic Inc.) was designed for pacemakers (not ICDs) to prevent double counting when sensing between the LV tip and the RV tip.¹² This feature prevents restarting the ventricular refractory period, postventricular atrial blanking and refractory periods, and upper rate timers when a second sensed depolarization is seen in the ventricular refractory period following a sensed event (as in LBBB) (Fig. 14). When the second sensed depolarization occurs within the interventricular refractory period, the refractory periods and timing intervals are not reset, thus preventing the second sensed depolarization from limiting upper tracking rates as seen in first-generation devices. In other words, it rectifies the problem of inappropriate extension of the atrial refractory period due to sensing of a delayed LV potential during the ventricular refractory period. The interventricular refractory period functions like a ventricular sensing blanking period which itself is not programmable in these devices.

Figure 13. Double counting of the ventricular electrogram in a patient who had received inappropriate shocks by an implanted Guidant Contak CD biventricular ICD that senses from both ventricles simultaneously. The atrial and ventricular electrograms are on top. The first 2 ventricular complexes are paced. The atrial rate then exceeds the programmed upper rate and a repetitive pre-empted Wenckebach sequence starts. The device then senses each conducted QRS twice. The second last cycle terminates with a paced ventricular beat because of slight sinus slowing. AS = atrial-sensed event; VP = ventricular paced event; VS = ventricular-sensed event; VT = ventricular tachycardia; VF = ventricular fibrillation (reproduced from Barold, Garrigue, and Israel¹² with permission).

Figure 14. Diagrammatic representation of the interventricular refractory period (IRP) of the Medtronic InSync III biventricular pacemaker. The IRP prevents sensing of a second ventricular depolarization when the RV and LV do not depolarize simultaneously. Thus, a sensed event in the IRP (either following a ventricular paced event or a non-refractory sensed event) does not initiate new timing cycles. A = atrium; S = non-refractory sensed event; P = paced event; R = refractory-sensed event. Note the short P-P intervals representing the V-V delay or the timing difference between LV and RV stimulation (reproduced from Barold, Garrigue, and Israel¹² with permission).

Table 5. Indicators for Far-Field P wave Sensing in Devices with Common Sensing Channels

1. Recurrence or development of symptoms of CHF
2. Inappropriately short AS–VP delay on surface ECG
3. Unexpected inhibition of ventricular output (DDD mode) with a PR (AS–VS) interval > programmed AS–VP delay (at rates *below* the maximum tracking rate)
4. Event markers recorded with simultaneously telemetered ventricular electrogram and surface ECG.

Far-Field Sensing of Atrial Depolarization Causing Double and Triple Counting

A device with common RV and LV sensing may sense the far-field atrial electrogram via an LV lead located (or displaced from its original site) in

one of the coronary veins because of its proximity to the AV groove and the left atrium.^{11,12,52–56} In the first-generation devices with a common sensing channel, far-field atrial sensing by the ventricular channel inhibits biventricular pacing and induces the emergence of spontaneous AV conduction (Table 5). A wide ventricular electrogram inducing double counting together with the sensed atrial signal results in triple counting at the ventricular level (Fig. 15). Far-field atrial sensing can also cause inappropriate discharge of a biventricular ICD (Fig. 16). This complication is devastating in pacemaker-dependent patients who have undergone ablation of the AV junction for permanent atrial fibrillation prior to the implantation of a biventricular device because of the risk of ventricular asystole and inappropriate therapy including shocks.⁵⁶

Figure 15. Far-field atrial sensing resulting in triple counting of a biventricular pacemaker. Simultaneous recordings (from top to bottom) of the ECG, a marker channel, and telemetered ventricular electrogram of a (dual-cathodal with a common sensing ventricular channel) Medtronic InSync DDDR biventricular pacemaker programmed to the ODO mode (25 mm/s). There is sinus rhythm with 1:1 AV conduction and ventricular inhibition. The LV lead detects the late portion of the P wave because of its proximity to the coronary sinus and left atrium (VS follows each AS closely), resulting in complete inhibition of ventricular pacing. Every P wave is conducted and produces a wide QRS sequentially sensed by the RV lead, then by the LV lead, as a function of the distance between the leads, the long interventricular conduction time, and the duration of the blanking period. Therefore, there are three ventricular-sensed markers associated with each QRS complex—the first VS is the far-field atrial signal, the second VS and third VS markers depict the two near-field components of the ventricular electrogram originating from the RV and LV leads, respectively. In the DDDR mode, the two signals generated by ventricular depolarization were recorded as VR events (refractory sensed) by the marker channel. VS = ventricular-sensed event; AS = atrial-sensed event (reproduced from Lipchenka, Garrigue, Glikson, et al.⁵² with permission).

Figure 16. Double jeopardy during biventricular pacing. Simultaneous recording of lead II ECG (top), bipolar ventricular electrogram between the right and left ventricular electrodes (middle), and annotated markers (bottom) showing far-field oversensing of atrial fibrillation potentials by a Guidant PRIZM VVIR ICD modified for biventricular pacing with a Y-adaptor (25 mm/s). The recording shows a “double whammy” with ventricular asystole and the delivery of an inappropriate shock. VT = ventricular tachycardia (intervals between 300 and 500 ms); VF = ventricular fibrillation (intervals < 300 ms); VS = ventricular-sensed event. The ventricular shock terminated atrial fibrillation and produced atrial standstill. Atrial fibrillation returned a few seconds later (reproduced from Garrigue, Barold, Clementy, et al.⁵⁶ with permission).

Figure 17. Ventricular triggered mode of third-generation Medtronic biventricular ICD (VVIR mode). The device triggers a biventricular output upon sensing the RV electrogram in an attempt to provide resynchronization upon sensing. The pacemaker stimuli (RV and LV) deform the sensed ventricular premature beats. The degree of electrical resynchronization cannot be determined from this tracing. Note that in the DDD/DDDR mode, the ventricular triggered mode functions only with ventricular sensing in the AV delay.

Figure 18. Ventricular resynchronization upon RV sensing by the ventricular triggered mode of a Medtronic biventricular ICD in the DDD mode. The patient has intra-atrial conduction delay so that the atrial electrogram sensed in the atrial appendage occurs late during the isoelectric portion of the PR interval (dotted vertical line). The AS–VS interval during AV conduction measures only 50–60 ms. The patient did not tolerate an AS–VP interval of 40 ms to produce biventricular pacing which in all likelihood occurred with some degree of fusion with the spontaneous conducted QRS complex. This situation calls for one of the two options: (1) ablation of the AV junction and (2) using the triggered mode upon sensing. A trial of the triggered mode produced marked clinical improvement. Consequently, the pacemaker was programmed to the ventricular triggered mode for long-term pacing. AS is followed by VS (smaller downward deflection) which triggers VP (larger downward deflection); AEGM = atrial electrogram; AS = atrial-sensed event; VS = ventricular-sensed event; VP = biventricular paced event.

Devices Susceptible to Double Counting

Conventional dual chamber ICDs used in an “off-label” fashion with a Y-connector for biventricular pacing (simultaneous RV and LV pacing and sensing) can exhibit double counting.^{12,50} One commercially available first-generation Guidant biventricular ICD (Contak CD) system used a conventional dual cathodal system and sensed simultaneously from the RV and LV causing double counting in about 7% of cases.¹² All contemporary devices allow programming of the sensing function of the individual ventricular channels to prevent double counting (RV and LV electrograms) or triple counting (far-field P wave, RV and LV EGMs). Biventricular ICDs now sense only from the RV to avoid double sensing and inappropriate ICD therapy.

RESYNCHRONIZATION DURING SENSING BY THE TRIGGERED RESPONSE

The ventricular triggered mode provides cardiac resynchronization in the presence of ventricular sensing. When a biventricular pacemaker programmed to the DDD/T mode (triggered at the ventricular level) detects ventricular activity in the AV delay, the signal triggers an output² (Fig. 17). In other words, a ventricular-sensed event initiates an

immediate emission of a ventricular or usually a biventricular output (according to the programmed settings) in conformity with the programmed upper rate interval.² The stimulus will be ineffectual in the chamber where sensing was initiated because the myocardium is physiologically refractory. The triggered stimulus to the other ventricle thus attempts to provide resynchronization activation in the setting of intraventricular dyssynchrony. Figure 18 shows how this modality avoided AV junctional ablation in a patient with intra-atrial conduction delay.

In atrial fibrillation, some devices provide some degree of ventricular resynchronization by the triggered mode and attempt regularization of the paced beats up to the programmed maximum tracking rate. Activation of this algorithm does not result in control of the ventricular rate, and should not be a substitute for ablation of the AV junction in patients with drug-refractory rapid ventricular rates.

RESTORATION OF ATRIAL TRACKING BY PVARP ABBREVIATION

The delivery of ventricular resynchronization and 1:1 atrial tracking can be disrupted under certain circumstances (below the upper rate) where

Figure 19. Stored markers showing the development of a locked P wave within the postventricular atrial period (PVARP) of a Medtronic biventricular ICD. AS-VP = 130 ms, upper rate = 130/min (upper rate interval = 460 ms). (A) Normal atrial tracking and biventricular pacing at the programmed AS-VP delay. (B) The P wave is locked into the PVARP despite a ventricular rate slower than the programmed upper rate (VS-VS = 470 ms and upper rate interval = 460 ms). The algorithm shown in Figure 20 would make the diagnosis of the P wave trapped in the PVARP and would restore atrial tracking by PVARP abbreviation.

- 1 Atrial events occur during PVARP and are not tracked.
- 2 After eight qualifying AR-VS cycles, ATR intervenes to break the pattern. PVARP is shortened.
- 3 The intervention continues until proper A-V tracking at the programmed SAV value resumes.

Figure 20. Medtronic's atrial tracking recovery algorithm during biventricular pacing. The algorithm recognizes VS-AR sequences only when the VS-VS interval is longer than the programmed upper rate interval. Abbreviation of the postventricular atrial period promotes recovery of atrial tracking and restores ventricular resynchronization. This algorithm would therefore restore atrial tracking from point 2 to point 3. Figure 19 also shows a situation where the algorithm would be useful.

atrial events are locked into the PVARP. The P wave becomes trapped into the PVARP at atrial rates below the programmed upper rate in the presence of normal AV conduction. This leads to AR-VS cycles where AR is sensed in the PVARP (Fig. 19). Resumption of 1:1 atrial tracking and resynchronization require slowing of the sinus rate so that the P-P or sinus interval exceeds the sum of intrinsic PR interval (AR-VS) + PVARP (prevailing intrinsic TARP).² A special algorithm to restore 1:1 atrial tracking at rates slower than the programmed upper rate works when the devices detect AR in the PVARP (Fig. 20). The algorithm temporarily shortens the PVARP and therefore the intrinsic TARP to permit sensing of the P wave beyond the PVARP so as to restore 1:1 atrial tracking. This algorithm may be useful in patients with sinus tachycardia and first-degree AV block in whom prolonged locking of the P waves inside the PVARP is an important problem.⁵⁷ Such patients are sometimes best treated with ablation of the AV junction.

OMISSION OF LEFT VENTRICULAR STIMULUS

Theoretically (but unlikely), lack of LV sensing could result in competitive pacing and arrhythmia induction. Such a situation might occur if a premature ventricular complex originates near the LV sensing site and at specific time before the P wave: If ventricular activation initiated by the VPC conducts to the RV sensing site with a marked delay, it will be unable to inhibit the scheduled ventricular pacing pulse (triggered by the P wave) thereby delivering the ventricular stimulus beyond the absolute myocardial refractory period. For this reason, Guidant has incorporated an algorithm in their devices to prevent unsafe LV pacing into the vulnerable period. An LV-sensed event initiates an LV protection period during which the LV stimulus is inhibited. The LV protection period is programmable between 300 and 500 ms after an LV-sensed event. LV sensing is used only for inhibition of LV pacing.

BIVENTRICULAR PACING WITH CONVENTIONAL PACEMAKERS

Although conventional dual chamber pacemakers are not designed for biventricular pacing and generally do not allow programming of an AV delay of zero or near zero, they are being used

Table 6. Loss of Cardiac Resynchronization During DDD or DDDR Pacing in the Presence of Preserved LV Pacing

A. Intrinsic

Atrial undersensing from low amplitude atrial potentials
T wave oversensing and other types of ventricular oversensing such as diaphragmatic potentials
Long PR interval
Circumstances that push the P wave into the PVARP such as a junctional rhythm
New arrhythmia such as atrial fibrillation with a fast ventricular rate
First-generation devices with a common sensing channel: ventricular double counting and sensing of far-field atrial activity

B. Extrinsic

Inappropriate programming of the AV delay or any function that prolongs the AV delay such as rate smoothing, AV search hysteresis, etc.
Low maximum tracking rate
Functional atrial undersensing precipitated by an atrial premature beat or ventricular premature beat. Long PVARP including post VPC automatic PVARP extension
Intra-atrial conduction delay where sensing of AS is delayed in the right atrial appendage. A short AS-VP interval may not be able to achieve biventricular pacing

with their shortest "AV delay" (0–30 ms) for ventricular resynchronization in CHF patients with permanent atrial fibrillation.⁵⁸ Their advantages include programming flexibility, and cost considerations. When a conventional dual chamber pacemaker is used for biventricular pacing, the "atrial" channel is generally connected to the LV and the "ventricular" channel to the RV.⁵⁸ This arrangement provides (1) LV stimulation before RV activation (LV pre-excitation) and (2) protection against ventricular asystole (but not sensing of atrial activity) related to oversensing far-field atrial activity. The DVI(R) mode behaves like the VVI(R) mode except that there are always two closely coupled stimuli (or electrocardiographically fused stimuli if the "AV delay" is very short) thereby facilitating evaluation of pacemaker function. Furthermore, the DVI(R) mode provides absolute protection against far-field sensing of atrial activity in the case of LV lead displacement. The short delay between LV and RV stimulation imposed by the shortest "AV delay" may not be a significant limitation in many patients because it is LV pacing that generally provides the salutary effect of biventricular pacing.

CONCLUSION

The advent of biventricular (triple-chamber) devices has added new complexity to the evaluation of pacemaker function and follow-up. Patients with severe CHF benefit greatly from small improvements in hemodynamics. Consequently, one must be cognizant of the various disturbances that can disrupt ventricular resynchronization (Table 6). The presence of interatrial conduction block or delay in CHF patients complicates therapy because it requires additional atrial resynchronization and therefore four-chamber devices for optimal hemodynamic benefit.⁵⁹ The true value of ventricular resynchronization with monochamber LV pacing remains unclear at this time. There is more to learn about the various patterns of the 12-lead ECG in the assessment of lead location and the efficacy of electrical versus mechanical resynchronization especially with the new capability of varying the stimulation interval (V-V) between the two ventricles. The recent introduction of triple ventricular pacing (LV and two sites in the RV) for patients refractory to standard biventricular (RV + LV) resynchronization promises to add more complexity to the interpretation of pacemaker function with electrocardiography.⁶⁰

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