

Marked First-Degree Atrioventricular Block: A New Indication for Cardiac Pacing

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Schüller and Brandt¹ defined the pacemaker syndrome in terms of “symptoms and signs present in the pacemaker patient which are caused by inadequate timing of atrial and ventricular contractions”. This characterization also applies to patients without an implanted pacemaker when “inadequate timing of atrial and ventricular contractions” causes a similar hemodynamic derangement². Thus isolated marked first-degree AV block can cause symptoms similar to the pacemaker syndrome especially in the presence of normal LV function³⁻⁹. A P wave too close to the preceding QRS complex produces basically the same hemodynamic derangement as VVI pacing with retrograde VA conduction¹⁰. In this regard, Chirife et al³ have called the hemodynamic disturbance produced by marked first-degree AV block as the “pacemaker syndrome without a pacemaker” and other workers have referred to this entity as the “pseudopacemaker syndrome”⁴.

Several reports have now documented the benefit of dual chamber pacing in patients with symptomatic marked first-degree AV block and normal left ventricular function³⁻⁹. A number of the reported patients developed their problem secondary to ablation of the fast pathway for the treatment of AV nodal reentry tachycardia^{4,5}. In symptomatic patients from a long PR interval (LPRI) as an isolated abnormality the clinician must establish that the benefit of optimizing AV synchrony with a shorter AV

delay outweighs the loss of left ventricular (LV) function produced by pacing induced aberrant ventricular depolarization¹¹⁻¹³. This determination can sometimes be made clinically and noninvasively but a hemodynamic study with temporary pacing may be required (Figures 1-3).

Differential diagnosis

1. The diagnosis of LPRI can be overlooked when the PR interval is very long because the ECG may be interpreted as showing a junctional rhythm with retrograde conduction. In questionable cases, sinus rhythm is easily demonstrated by demonstrating its characteristic pattern of right atrial activation (from high to low right atrium) during an electrophysiologic study (Figure 1).
2. Patients with a LPRI are susceptible to sinus tachycardia in response to the abnormal hemodynamics. During sinus tachycardia, the ECG may be interpreted as showing supraventricular tachycardia because the sinus P wave close to the tail end of the QRS complex (Figure 2)¹⁴.
3. Some patients, a long interatrial conduction time and a long PR interval may already have appropriately timed mechanical left AV synchrony, and in such a situation improvement would not be expected with pacing¹⁵.

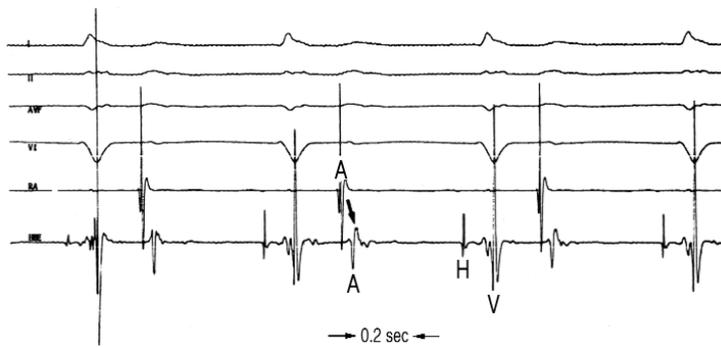


Figure 1. Surface ECG and intracardiac recording from a patient with symptomatic marked first-degree AV block. RA=high right atrial electrogram, HBE= electrogram at site of His bundle recording. Note the sequence of atrial activation (RA to HBE) is consistent with sinus rhythm and rules out retrograde atrial activation. The AH interval is markedly prolonged.

Significance of mitral regurgitation

In patients with LPRI atrial systole with premature and incomplete “atriogenic” closure of the mitral valve causes varying degrees of end-diastolic mitral regurgitation (MR) in mid or late diastole¹⁶⁻²¹. This MR is inconsequential in the normal heart but may be important in patients with severe LV dysfunction¹⁷.

Indications for pacing

Wharton and Ellenbogen²² have argued that “symptomatic first-degree AV block with symptoms suggestive of pacemaker syndrome” should be a Class I indication for permanent pacing. They emphasized that “symptoms can be subtle in some patients or may be of sufficiently long duration that temporary pacing may be indicated to document improvement or reversal of longstanding problems. However, the 1998 ACC/AHA guidelines for pacemaker implantation state that “first-degree AV block (>0.30 sec.) as a Class IIa indication when patients display “... symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary AV pacing” (Figure 3)²³.

Patients with LPRI may or may not be symptomatic at rest. They are more likely to become symptomatic with mild or moderate exercise when the PR interval does not shorten appropriately and atrial

systole shifts progressively closer towards the previous ventricular systole. Patients with subtle symptoms should undergo a treadmill stress test to determine the hemodynamic disadvantage of the LPRI. The class IIa recommendation for permanent pacing does not apply to patients with a long PR interval associated with dilated cardiomyopathy, and congestive heart failure (CHF). Such patients are best treated with a DDDR pacemaker providing biventricular stimulation. The necessity and appropriateness of a temporary AV pacing study in LPRI patients are questionable, especially if the PR interval is very long and does not shorten on exercise. During a resting study it may not be possible to demonstrate symptomatic improvement, and the execution of exercise studies with temporary dual-chamber pacemaker in place is difficult. Therefore a permanent pacemaker may be recommended in selected patients without a temporary pacing study that would add unnecessary risk and cost²⁴.

AV Delay vs asynchronous activation

Iliev et al²⁵ recently compared the AAI and DDD modes in patients with sick sinus syndrome (DDD pacemakers) and native but *long* AV conduction in otherwise normal hearts. At a pacing rate of 70 ppm at rest, there was no overall difference in the aortic

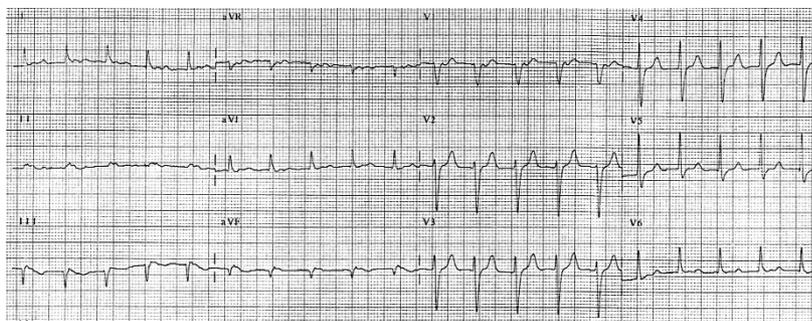


Figure 2. Twelve-lead ECG in the same patient as in Fig 1. During sinus tachycardia the sinus P wave is close to the preceding QRS complex on the initial portion of the ST-segment. This pattern mimics a reentrant supraventricular tachycardia.

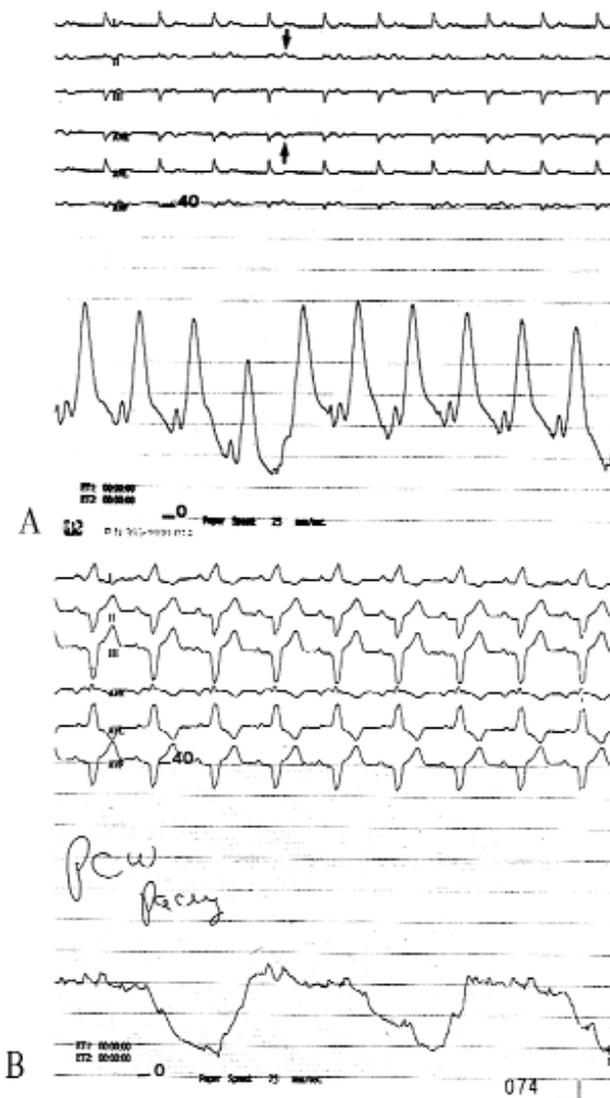


Figure 3. Same patient as Figures 1 and 2. **A.** Pulmonary capillary wedge pressure shows large cannon waves during sinus rhythm with a very long PR interval (Scale 0-40 mmHg). **B.** Note the normal pulmonary capillary wedge pressure after temporary dual chamber pacing with a physiologic AV delay (Scale 0-40 mm Hg).

flow time velocity integral (which reflects cardiac output) during AAI and DDD pacing. However when the patients were divided according to the AV interval (AVI), those with AVI [270 ms showed a higher aortic flow velocity integral during AAI pacing. When the AVI >270 ms, the aortic flow velocity integral was higher during DDD pacing. They established that during DDD pacing that the longer the native AV interval is, the larger the resultant increments in CO. Conversely with a normal or near normal PR interval, a higher CO was found

during AAI pacing with a conducted QRS complex and spontaneous ventricular depolarization. Knowing the physiology of the LPRI syndrome, it is not surprising these workers found that at a pacing rate of 90 ppm, DDD was superior to AAI pacing. These observations should be considered before pacemaker implantation to optimize the AV interval because in some patients deterioration of LV function secondary to paced (asynchronous) ventricular depolarization may outweigh the benefit of optimized AV synchrony.

Asymptomatic and mildly symptomatic patients

Slightly symptomatic patients with LPRI (>300 ms) without a clearcut “pacemaker syndrome” should be investigated with an exercise test and echo-Doppler examination. A temporary pacing study may then be required to make a decision about permanent pacing. There are no data on the prognosis of asymptomatic patients with LPRI. Pacing is generally not recommended in asymptomatic patients with isolated LPRI even with abnormal hemodynamics. However, a truly asymptomatic patient with moderately severe hemodynamic abnormalities probably deserves a pacing study and if it shows good improvement with restoration of optimal AV synchrony, a permanent pacemaker should be considered.

Long PR interval, dilated cardiomyopathy and congestive heart failure

Over the last decade long-term studies with conventional DDD pacing and a short AV delay in heterogeneous groups of patients with congestive heart failure (CHF) of various etiologies have generally yielded disappointing results. In some patients conventional DDD pacing causes further deterioration of LV function related to pacing-induced abnormal ventricular depolarization. In patients with 1st degree AV block, conventional DDD pacing abolishes presystolic mitral regurgitation and increases the time for forward flow. Elimination of diastolic mitral regurgitation plays as yet an undefined role in the overall hemodynamic benefit of conventional DDD pacing in selected patients with severe LV dysfunction, CHF and 1st degree AV block. Abolition of diastolic mitral regurgitation may result in more optimal hemodynamic performance because of a lower left atrial pressure and higher LV preload at the onset of systole.

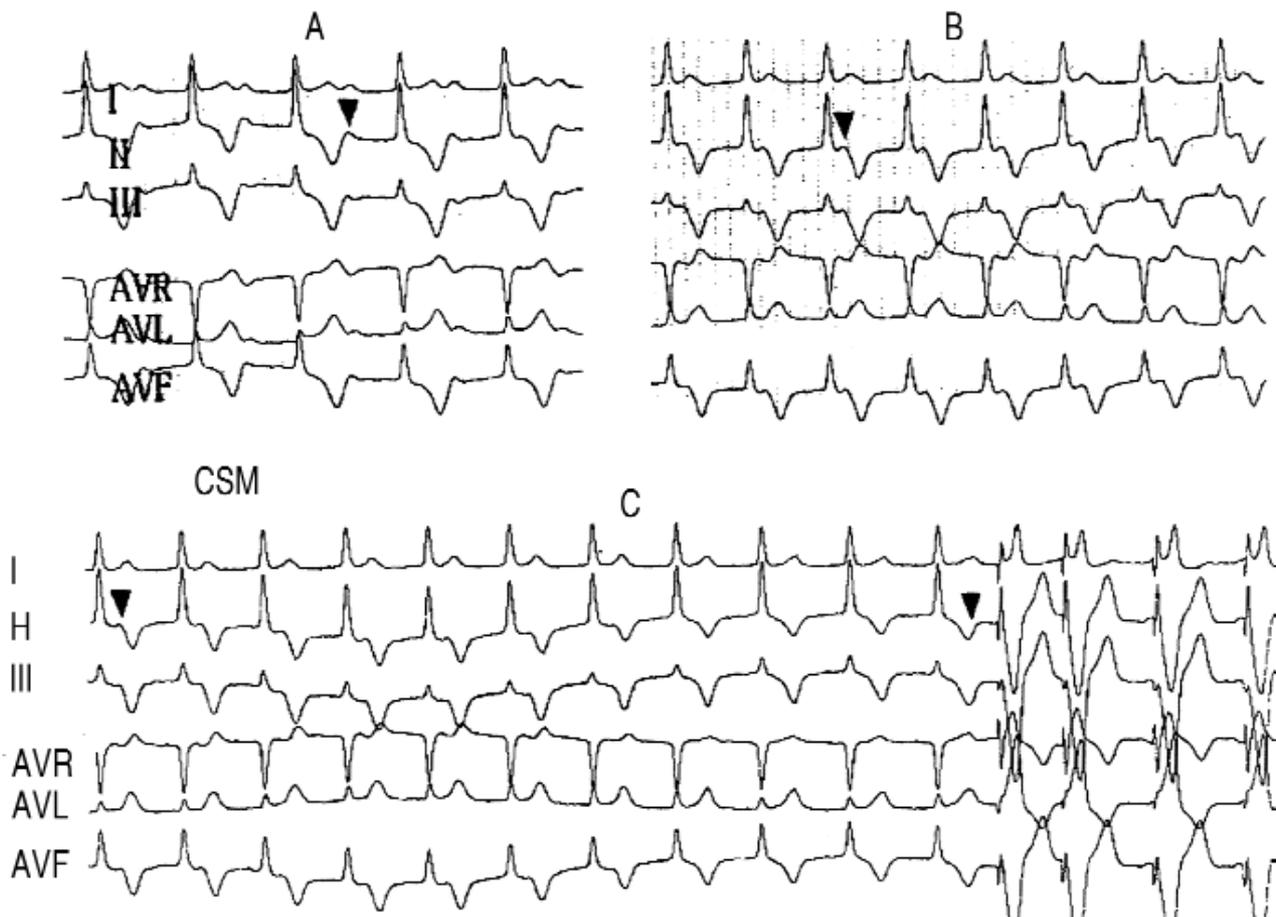


Figure 4. Six-lead ECG showing marked first-degree AV block and sinus tachycardia in a patient with a DDD pacemaker. The postventricular atrial refractory period (PVARP) was 360 ms. The arrow heads point to sinus P waves. **A.** Sinus rhythm at a rate of 88 bpm with an R-P interval of approximately 360 ms. Note the positive P wave near the end of the T wave in leads I, II, III, and aVF. **B.** Sinus tachycardia at a rate of 112 bpm and a longer PR interval than in panel A. The R-P interval now measures only 150 ms. At that time the PVARP was shortened to 200 ms but the tachycardia continued. **C.** Carotid sinus massage (CSM) caused a gradual slowing of the sinus rate with eventual restoration of AV synchrony. The arrow head points to a P wave beyond the 200 ms PVARP. The succeeding 2 ventricular stimuli occur after completion of the upper rate interval. The basic AV delay was 120 ms and the rate-adaptive function was programmed on. (from ref 13 with permission).

The 1998 ACC/AHA guidelines advocate conventional dual-chamber pacing as a class IIb indication in patients with “symptomatic, drug-refractory dilated cardiomyopathy with prolonged PR interval when acute hemodynamic studies have demonstrated hemodynamic benefit of pacing”²³. Neither the degree of acceptable PR prolongation nor the QRS duration is stated. This recommendation is presently controversial especially in the absence of a major intraventricular conduction delay. Patients with refractory CHF, a long PR interval and QRS >130 ms should be considered for either left ventricular or biventricular DDDR pacing. The role of pacing in patients with a long PR interval and no

major intraventricular conduction delay is generally disappointing²⁶. If pacing is considered in this situation, an acute study involving VDD pacing should demonstrate improvement with either conventional, left ventricular or biventricular pacing. However acute improvement does not guarantee long-term benefit in these patients.

Pacemaker technology

The pacing system must prevent migration of the P wave into the postventricular atrial refractory period (PVARP) where it cannot be sensed resulting in loss of AV synchrony (Figures 4, 5).

Functional atrial undersensing

Bode et al²⁷ recently reported the problems associated with pacing patients with 1st degree AV block. They studied 255 patients with Holter recordings and found 9 patients with atrial undersensing despite an adequate atrial signal. The P waves fell continually within the PVARP (PVARP of 276 ms; no PVARPs functioned with automatic extension in response to ventricular extrasystoles). All 9 patients exhibited substantial delay of spontaneous AV conduction (284 ms, range 230-410 ms). The combination of a relatively fast sinus rate and prolonged AV conduction provides the appropriate setting for the development of functional atrial undersensing during which the ECG shows sinus rhythm, a long PR interval and conducted QRS complexes but no pacemaker stimuli. The conducted QRS complexes activate the ventricle while the P waves remain trapped in the PVARP. The pacemaker itself acts as a “bystander” in that it can initiate the pacemaker syndrome but the ECG then shows no pacemaker activity. Bode et al²⁷ also observed that functional atrial undersensing could be initiated and terminated by appropriately timed atrial and ventricular extrasystoles. Barring disruption of the self-perpetuating process by atrial or ventricular extrasystoles, and assuming no change in AV conduction, functional atrial undersensing should theoretically continue indefinitely as long as the atrial rate remains relatively fast and constant. It will however terminate when slowing of the sinus rate produces a P-P in-

terval longer than the sum of the intrinsic PR interval and PVARP (Figure 4).

Heart rate

Bode et al²⁷ recorded a mean sinus rate of 105 ± 3 bpm during functional undersensing because a relatively fast atrial rate facilitates displacement of the P wave toward the PVARP. Some patients with functional atrial undersensing develop marked sinus tachycardia probably as a response to the hemodynamic derangement created by the loss of optimal AV synchrony. The tachycardia often subsides quickly upon restoration of a physiologic AV delay by the pacemaker. As a rule a very long PR interval does not shorten significantly in situations causing sinus tachycardia. Therefore with a fixed PR interval sinus tachycardia may create a vicious cycle because it pushes the P wave closer to the preceding ventricular complex and if this arrangement creates a more unfavorable VA relationship, it will in turn aggravate the sinus tachycardia (Figures 4, 5).

Pacemaker syndrome

During functional atrial undersensing 5 of the 9 patients reported by Bode et al²⁴ developed complaints suggestive of the pacemaker syndrome. Bode et al²⁷ prevented functional atrial undersensing in 7 of their 9 patients by shortening the PVARP and AV delay and previously symptomatic patients became asymptomatic. The other 2 patients exhibited less atrial undersensing.

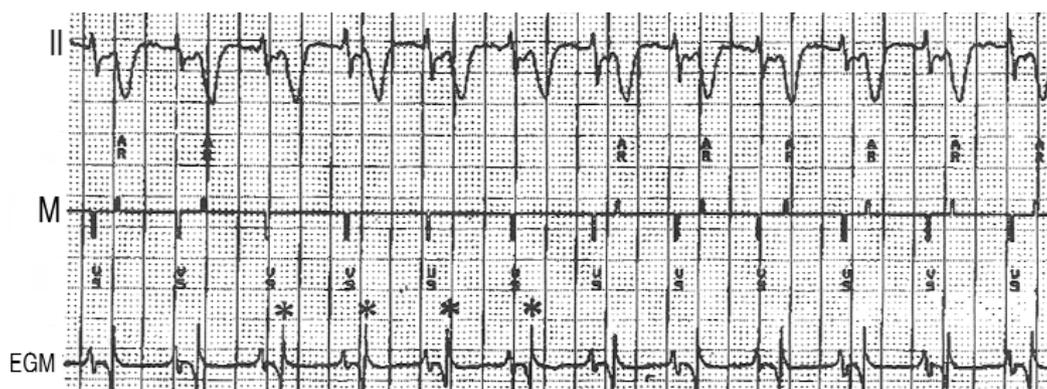


Figure 5. Same patient as in Fig 4. PVARP=360 ms. There is sinus tachycardia at a rate of 110 bpm and a long PR interval. The R-P interval was about 150 ms. The pacemaker detects P waves in the 360 ms PVARP but not when the R-P interval becomes shorter than the 150 ms postventricular atrial blanking period (no AR representation-asterisks). AR=atrial event sensed in the PVARP, AS=atrial sensed event, EGM=electrogram, M=markers, VS=ventricular sensed event. Paper speed=25 mm/sec. (from ref 13 with permission).

Significance of PVARP extension

Functional atrial undersensing can occur with a *short* PVARP whenever a ventricular extrasystole activates an automatic PVARP extension.²⁸⁻³¹ In this situation an unsensed P wave within the extended PVARP gives rise to a conducted QRS complex which the pacemaker interprets as a ventricular extrasystole whereupon it generates another PVARP extension. The extended PVARP is perpetuated from cycle to cycle as long as the pacemaker interprets the conducted QRS as a ventricular extrasystole.

Prevention of functional atrial undersensing

A relatively short PVARP can often be used to prevent functional atrial undersensing because retrograde ventriculoatrial (VA) block is common in patients with LPRI. Other measures³² include: 1. Capability of programming off the automatic PVARP extension after a ventricular extrasystole. Most patients do not need this function in the absence of VA conduction. 2. Elimination of the PVARP extension whenever the pacemaker detects a P wave within the PVARP immediately before the next sensed ventricular beat. 3. Noncompetitive atrial pacing with the delivery of a premature but appropriately delayed atrial stimulus whenever the pacemaker senses activity in the PVARP. In this function an atrial stimulus is emitted 300 ms after the pacemaker senses atrial activity in the PVARP beyond the postventricular atrial blanking period. This process promotes AV resynchronization³³. 4. Ablation of the AV junction with resultant complete AV block in difficult situations. With a pacemaker already in place, this procedure is relatively simple^{6,34}.

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