Cardiac Pacing in First-Degree Atrioventricular Block

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ABSTRACT

This article will summarize the cardiac pacing in first-degree atrioventricular block.

KEYWORDS
Cardiac pacing, first-degree atrioventricular block

Birinci Derece Atriyoventrüküler Blokta Kalp Pili

ÖZET

Bu yazıda birinci derece atriyoventrüküler blokta kardiyak pacing özetlenecektir.

ANAHTAR KELİMELER
Birinci derece atriyoventrüküler blok, kardiyak pacing

İLETİŞİM ADRESİ
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First-degree atrioventricular (AV) block, often considered a relatively benign arrhythmia, can occasionally be associated with severe symptoms. Although there is little evidence to suggest that pacemakers improve survival in patients with isolated first-degree AV block, it is now recognized that marked (PR ≥ 0.30 sec) first-degree AV block can lead to symptoms similar to those in the pacemaker syndrome even in the absence of higher degrees of AV block (1-4). Uncontrolled trials have shown that many symptomatic patients with a PR interval ≥ 0.30 sec can be improved with dual chamber pacing especially in patients with normal left ventricular (LV) function.

**Echocardiography**

Prolonged AV conduction causes atrial systole too early in diastole which then causes an ineffective or decreased contribution of atrial systole to cardiac output. Such a decrease in cardiac output is poorly tolerated in heart failure patients. Atrial contraction begins in early diastole resulting in atrial systole superimposed upon the early left ventricular (LV) filling phase and much earlier than the onset of LV systolic pressure. There is therefore delay of the E wave with resultant fusion between the E and A waves producing shortening of the LV diastolic filling time (Fig 1). The delay in AV conduction also induces diastolic mit-
ral regurgitation (Fig 2). In a normal heart, atrial systole occurs immediately before ventricular systole. In the setting of a long PR interval, the atrium begins to relax and atrial pressure drops after atrial systole. This causes the mitral valve to remain open because of delayed LV systole. With the mitral valve open, the LV end-diastolic pressure rises and exceeds left atrial pressure thereby producing diastolic mitral regurgitation, a decrease in preload (LV end-diastolic pressure) at the onset of LV systole and, ultimately, a decrease in LV dP/dt max and cardiac output. This type of mitral regurgitation appears inconsequential in the normal heart but may be important in patients with severe LV dysfunction.

**Pacemaker-like Syndrome**

AAI(R) pacing with a very long PR interval may produce pacemaker syndrome. This form of pacemaker syndrome has resurfaced more recently in patients with intermittent marked first-degree AV block during managed ventricular pacing to minimize right ventricular pacing. In this pacing mode, a device permits the establishment of very long PR intervals without the emission of a ventricular output.

A long PR interval (≥ 0.30 sec) without a pacemaker shares the same pathophysiology as VVI pacing with retrograde ventriculoatrial conduction or an AAI-induced pacemaker syndrome. Some workers have called the hemodynamic disturbance produced by marked

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**FIGURE 2**

Continuous-wave Doppler echocardiographic recording of transmirtal blood flow velocity demonstrating diastolic mitral regurgitation in a patient with first degree AV block with a markedly prolonged PR interval of 360 msec. The low velocity regurgitant signal is seen in diastole following the atrial filling wave and prior to the onset of systolic mitral regurgitation.
first-degree AV block a “pacemaker syndrome without a pacemaker”, and others have labeled this entity as “pseudopacemaker syndrome.” These characterizations are potentially confusing. The term “pacemaker-like” syndrome is probably more appropriate.

Although patients with symptomatic marked first-degree AV block (≥ 0.30 sec), and normal LV function can benefit from dual chamber pacing. Some patients with a long PR interval and interatrial conduction delay may be already hemodynamically compensated because delayed conduction to the left atrium may be associated with an appropriately timed mechanical left AV synchrony, a situation where improvement would not be expected with pacing.

Clinical evaluation.

Patients with marked first-degree AV block 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 preceding LV systole. Patients with subtle symptoms should undergo a treadmill stress test to determine the hemodynamic disadvantage of the long PR interval (defined as ≥ 0.30 sec). In some patients the long PR interval itself may occasionally become manifest only on exercise. Adaptation of the PR interval on exercise is unlikely in patients with a PR interval ≥ 0.30 sec.

In symptomatic patients with marked 1st-degree AV block (as an isolated abnormality) it is important to realize that the benefit of pacing with optimized AV synchrony (with a shorter AV delay) may outweigh the risk of impaired LV function produced by right ventricular pacing with resultant LV dyssynchrony. This risk is impossible to evaluate on a short-term basis. An acute determination of the clinical status and need for permanent pacing can often be made noninvasively. Occasionally a therapeutic decision may be facilitated by an invasive hemodynamic study with temporary right atrial and ventricular pacing to assess the hemodynamic response to AV interval optimization (Fig 3). However, an acute hemodynamic

![Figure 3](image-url)

**FIGURE 3**

Hemodynamic abnormalities in a symptomatic patient with marked first-degree AV block and hemodynamic improvement with temporary dual chamber pacing. 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). (Reproduced with permission from Barold SS. Acquired atrioventricular block. In: Kusomoto FM, Goldschlager N (Eds), Cardiac Pacing for the Clinician, Philadelphia, PA, Lippincott Williams and Williams, 2001;229-251).
improvement with a more physiologic AV delay does not guarantee long-term benefit because of the potential risk of harmful long-term effects of continual right ventricular pacing. Furthermore, it may be impossible during a resting pacing study to demonstrate symptomatic improvement, and the performance of an exercise study with a temporary dual-chamber pacemaker in place is difficult. Therefore it is reasonable to recommend a permanent pacemaker in many symptomatic patients without a temporary pacing study which itself carries unnecessary risks and cost.

Asymptomatic patients

There are no data on the prognosis of asymptomatic patients with a long PR interval (≥ 0.30 sec). Pacing is generally not recommended in truly asymptomatic patients.

Differential diagnosis

1. The diagnosis of marked first-degree AV block can be overlooked when the PR interval is very long because the ECG may be misinterpreted to show a junctional rhythm with retrograde ventriculoatrial conduction (Fig 4). In questionable cases, sinus rhythm can be easily documented during an electrophysiologic study by delineating the pattern of right atrial activation which travels from high to low right atrium in sinus rhythm (Fig 5).

2. Patients with a long PR interval may respond to the abnormal resting hemodynamics with sinus tachycardia which may be misinterpreted as supraventricular tachycardia because the P-wave is placed so close to the preceding QRS complex.

AV delay vs. asynchronous ventricular activation

Optimization of the AV interval may be harmful in some patients because of deterioration of LV function secondary to paced ventricular depolarization. It is unwise to program a slow pacing rate and a very long AV delay to avoid right ventricular pacing in a patient who remains symptomatic. Programming a short AV
delay for hemodynamic benefit requires periodic evaluation of LV function to determine the cumulative effect of right ventricular pacing.

Long PR interval, and congestive heart failure

Studies with conventional DDD pacing and a short AV delay in heterogeneous groups of patients with LV systolic function and congestive heart failure of various etiologies have generally yielded disappointing results. In only a few patients with severe congestive heart failure and first-degree AV block, implantation of a conventional dual chamber pacemaker may occasionally improve cardiac performance but DDD pacing may also cause further long-term deterioration of LV function secondary to pacing-induced abnormal LV depolarization. Conventional DDD pacing abolishes presystolic mitral regurgitation and increases the time for forward flow. However, elimination of diastolic mitral regurgitation plays as yet an undefined but probably small role in the overall hemodynamic benefit but it may result in more optimal hemodynamic performance because of a lower left atrial pressure and higher LV preload at the onset of systole. Long-term deterioration of LV function during DDD or DDDR pacing may eventually necessitate an upgrade to biventricular pacing.

Indications for Pacing

The 2008 ACC/AHA/HRS Guidelines (3) state: “Although there is little evidence to suggest that pacemakers improve survival in patients with isolated first-degree AV block, it is now recognized that marked (PR more than 300 milliseconds) first-degree AV block can lead to symptoms even in the absence of higher degrees of AV block. When marked first-degree AV block for any reason causes atrial systole in close proximity to the preceding ventricular systole and produces hemodynamic consequences usually associated with retrograde (ventriculo-atrial) conduction, signs and symptoms similar to the pacemaker syndrome may occur. With
marked first-degree AV block, atrial contraction occurs before complete atrial filling, ventricular filling is compromised, and an increase in pulmonary capillary wedge pressure and a decrease in cardiac output follow. Small uncontrolled trials have suggested some symptomatic and functional improvement by pacing of patients with PR intervals more than 0.30 seconds by decreasing the time for AV conduction. Finally, a long PR interval may identify a subgroup of patients with LV dysfunction, some of whom may benefit from dual-chamber pacing with a short(er) AV delay. Although echocardiographic or invasive techniques may be used to assess hemodynamic improvement before permanent pacemaker implantation, such studies are not required. The table in the guidelines shows that first-degree AV block is a class IIa indication as follows: Permanent pacemaker implantation is reasonable for first- or second-degree AV block with symptoms similar to those of pacemaker syndrome or hemodynamic compromise.” It would seem more prudent at this juncture to consider the initial implantation of a biventricular DDD device when the LV ejection fraction is ≤ 35% even in the presence of a narrow QRS complex.

The guidelines of the European Society of Cardiology (4) state that: “In patients with first-degree AV block, cardiac pacing is not recommended unless the PR interval fails to adapt to heart rate during exercise and is long enough (usually .300 ms) to cause symptoms because of inadequate LV filling, or an increase in wedge pressure, as the left atrial systole occurs close to or simultaneous with the previous LV systole. In such cases small, uncontrolled studies have shown an improvement in patient’s symptoms.” The guidelines also recommend pacing with a IIa indication.

REFERENCES