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Review Article

Electrocardiographic features: Various atrial site pacing

Asit Das*

Department of Cardiology, IPGME&R and SSKM Hospital, Kolkata, India

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ABSTRACT

Atrial pacing is done for either symptomatic sinus node dysfunction (SND) or for maintenance of atrio-ventricular synchrony in a dual chamber pacemaker. Conventionally, atrial lead is placed in the right atrial appendage. Atrial conduction disorder in patients with permanent pacing results in higher incidence of atrial fibrillation. Atrial septal pacing has emerged as a solution to this problem. So, it is extremely important to understand the different features of paced P wave from various atrial pacing sites. Conventional right atrial appendage pacing in presence of atrial conduction disorder results in marked latency with prolonged P wave duration with reduced amplitude. The morphology is similar to sinus rhythm. Atrial septal pacing causes short and sharp P wave with negative polarity in inferior leads and positive polarity in lead V1 in lower septal pacing, whereas positive polarity in inferior leads and negative polarity in lead V1 during pacing from upper septum.

Contents

1. Introduction

Atrial pacing is done for either symptomatic sinus node dysfunction (SND) or for maintenance of atrio-ventricular synchrony in a dual chamber pacemaker for atrio-ventricular conduction disease or both sinus and atrio-ventricular disease.[1] Conventionally, atrial lead is placed in the right atrial appendage (RAA). In addition to abnormalities at the sinus node, SND is associated with widespread structural and electrophysiological changes in the atria.[2] Atrial conduction disorder (ACD) (P wave duration traditionally measured in lead II of more than 120 ms) in patients with permanent pacing results in higher incidence of atrial fibrillation.[3] Atrial electrophysiological alterations in persistent atrial fibrillation (AF) diminish rapidly although incompletely during maintained sinus rhythm after cardio version.[4] The occurrence of AF after pacemaker implantation in SND is associated with an increased risk of stroke, systemic embolism, heart failure, and mortality.[5] Permanent atrial pacing also has been proposed as one of the nonpharmacological alternative approaches in selected patient

* Corresponding author at: Flat – B1, GB – 43, Narayantala (West), DB Nagar, Kolkata 700059, India.
E-mail address: dradascard@rediffmail.com (A. Das).

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groups with presumed bradycardia-dependent atrial fibrillation (AF).[6] Atrial preference pacing algorithm, decreases the number of atrial premature beats and the P-wave dispersion, thereby reduces the onset and perpetuator factors of AF episodes and decreases the AF burden in patients who underwent dual chamber pacemaker implantation for various degree of atrio-ventricular blocks and documented atrial fibrillation.[7] On the other hand, the development of novel atrial pacing techniques such as biatrial, bifocal right atrial and single site inter-atrial septum pacing have extended the indications for pacemaker therapy to symptomatic drug refractory AF by prevention of paroxysmal AF. So, it is extremely important to understand the different features of paced P wave from various atrial pacing sites.

2. Normal activation sequence

The right atrium (RA) and left atrium (LA) are activated nearly simultaneously (within 50–80 ms) during sinus rhythm. Normally the spread of activation within each atrium and from right to left follow circumferential and longitudinal muscle bundles, but they are generally not considered to be part of the specialized conduction system. The sites of electrical connections between the right and the left atrium are the high septal right atrium or Bachmann's bundle (BB), the region of Koch's triangle, with left posterior extension of the AV node, the fossa ovalis, and the coronary sinus (CS) level with connections between the coronary sinus muscle cover and the left atrium.[8] One recent study using magnetocardiography (validating with intracardiac electroanatomic mapping) confirmed that left atrial activation occurs in majority through BB either solitary route or in combination with other routes.[9] In the right atrium, the crista terminalis and its medial extension favor the spread of activation in a longitudinal direction, whereas transverse conduction is limited. In the left atrium, circumferential muscle fibers along the mitral annulus as well as longitudinal fibres propagate the wave of depolarization slowly.

The contributions of right- and left-atrial activation to the beginning, middle, and end of the P wave are indicated in Fig. 1. The P wave normally appears entirely upright in leftward- and inferiorly oriented leads such as I, II, III, aVF, and V4 to V6 except V1 and possibly V2. In the short axis view provided by lead V1, which best distinguishes left- versus right-sided cardiac activity; the divergence of right- and left-atrial activation typically produces a biphasic P wave. It is negative in aVR because of the rightward orientation of that lead and is variable in other standard leads.

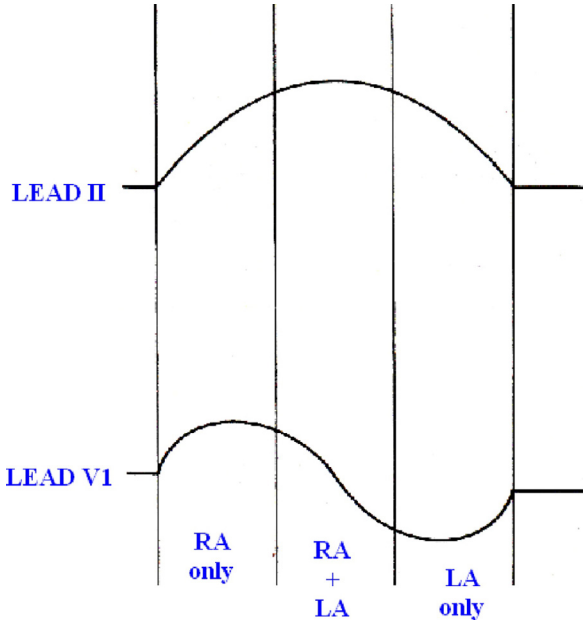

Fig. 1. Typical normal P wave. Lead II provides long-axis and lead V1 provides short axis view.

3. Normal activation sequence

ACD is defined electrocardiographically as P wave duration (traditionally measured in lead II) of more than 120 ms. ACD can be due to either intra- or inter-atrial conduction delay. Intra- and interatrial conduction disturbances are well known factors predisposing to AF development and/or maintenance. The most common site for conduction delay in patients with intra-atrial conduction delay is Koch's triangle. Inter-atrial conduction delay is less common than intra-atrial conduction delay. It is due to conduction delay in the region of BB.

4. Different sites of atrial pacing

Conventionally, atrial lead is placed in the RAA. As discussed earlier, pacing from RAA in patients with atrial conduction disorder results in higher incidence of AF. Susceptibility to paroxysmal lone AF is associated with propagation of atrial signal to LA via margin of fossa ovalis or multiple pathways. Altered and alternative conduction pathways may contribute to pathogenesis of lone AF.[10] Studies have shown that pacing atria from multiple sites (high right atrium with coronary sinus ostium/high right atrium with distal coronary sinus) in patients with atrial conduction disorder results in less incidence of AF.[11,12] Spencer et al. showed that pacing in the interatrial septum at its anterior and superior region, close to BB, results in a symmetric activation of both the atria simultaneously.[13] Several electrophysiological studies have shown that in patients with paroxysmal AF, single-site pacing at lower interatrial septum (LAS) is more effective than biatrial or dual-site atrial pacing in the prevention of AF induction by shortening atrial activation times, and avoiding the undesirable prolongation of the inter-atrial conduction in patients with AF.[14] The prevention of atrial arrhythmias by alternate site pacing or multi-site atrial pacing is by several different mechanisms: improvement of inter- and intra-atrial conduction; increasing the coupling interval of atrial premature beats; homogenous atrial depolarization; proper timing of atrial systole.

5. Status of alternate site atrial pacing

Bailin and colleagues randomized 120 patients of SND with history of paroxysmal AF (with or without AV node ablation) to either conventional RAA or BB pacing and found compared to RAA pacing, BB pacing significantly delayed the onset of permanent AF.[15] Padeletti and his colleagues studied 46 patients with paroxysmal AF randomized to either RAA pacing or LAS pacing and showed superiority of LAS pacing to RAA pacing in reducing AF burden over a 3 months period.[14] Taken together these studies suggest that pacing at the inter atrial septum (high or low) shortens the P wave duration and reduces incidence of AF compared to the RAA site. However, in 2005 interdisciplinary working group of American Heart Association in collaboration with the Heart Rhythm Society commented that until large multicenter clinical trials are available use of alternative site atrial pacing should be considered unproven and experimental.[16] Two large prospective randomized studies have shown that LAS pacing is

superior to RAA pacing in preventing persistent or permanent AF in patients with SND and intra atrial conduction delay.[17,18] On the contrary, Kugacka et al. have shown that single site CS pacing causes echocardiographic pacemaker syndrome in the right heart because of simultaneous retardation of RA contraction and earlier ventricular activation, while single sit BB pacing provides the best atrial contraction synchrony in patients with ACD, and has a comparable effect on global function to multisite atrial septal pacing.[19] However, Choudhuri et al. have suggested that in patients with intra-atrial conduction delay get most benefits from lower atrial septal pacing whereas patients with interatrial conduction delay require pacing at the region of BB.[20] A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society in 2012 stated that dual-site right atrial pacing or alternate single-site atrial pacing from unconventional sites (e.g., atrial septum or Bachmann's bundle) may offer additional benefits to single-site right atrial pacing from the appendage in patients with symptomatic drug-refractory AF and concomitant bradyarrhythmias; however, results from these studies are also contradictory and inconclusive.[21]

6. Electrocardiographic features of different atrial site pacing

During evaluation of a patient with atrial pacing, the 12-lead ECG yields important information about the site of pacing as well as the effect of pacing on atrial depolarization in term of duration and vectoral change. The atrial based paced ECG should be evaluated for latency, paced P wave morphology, duration and its vector.

6.1. Latency

The latency is defined here as the interval from the pacemaker stimulus to the onset of the earliest paced P wave. The demonstration of latency requires a 12-lead ECG taken at fast speed (50 mm/s) for diagnosis. In absence of any atrial conduction delay pacing from right atrial appendage does not produce any significant latency. However, in right atrial appendage pacing in presence of atrial conduction disorder, atrial muscle bundles take considerable time to propagate the stimulation from right atrial appendage to the right atrial free wall then to the crista terminalis and whole right atrium and subsequently to the left atrium across the atrial septum. So, there is a marked latency period. In Bachmann's bundle pacing (junction of the roof right atrium atrial septum), the wave of depolarization rapidly traverses to the left atrium via the bundle and excites the left atrium and in the right atrium via crista terminalis to the whole right atrium. So, there is no latency period and the P wave duration is short. Pacing from lower atrial septal region also exhibit very short latency period as the spread of activation from the pacing site rapidly traverses to the left atrium via coronary sinus musculature and simultaneously right atrium gets stimulated in a reverse direction by right atrial musculature.

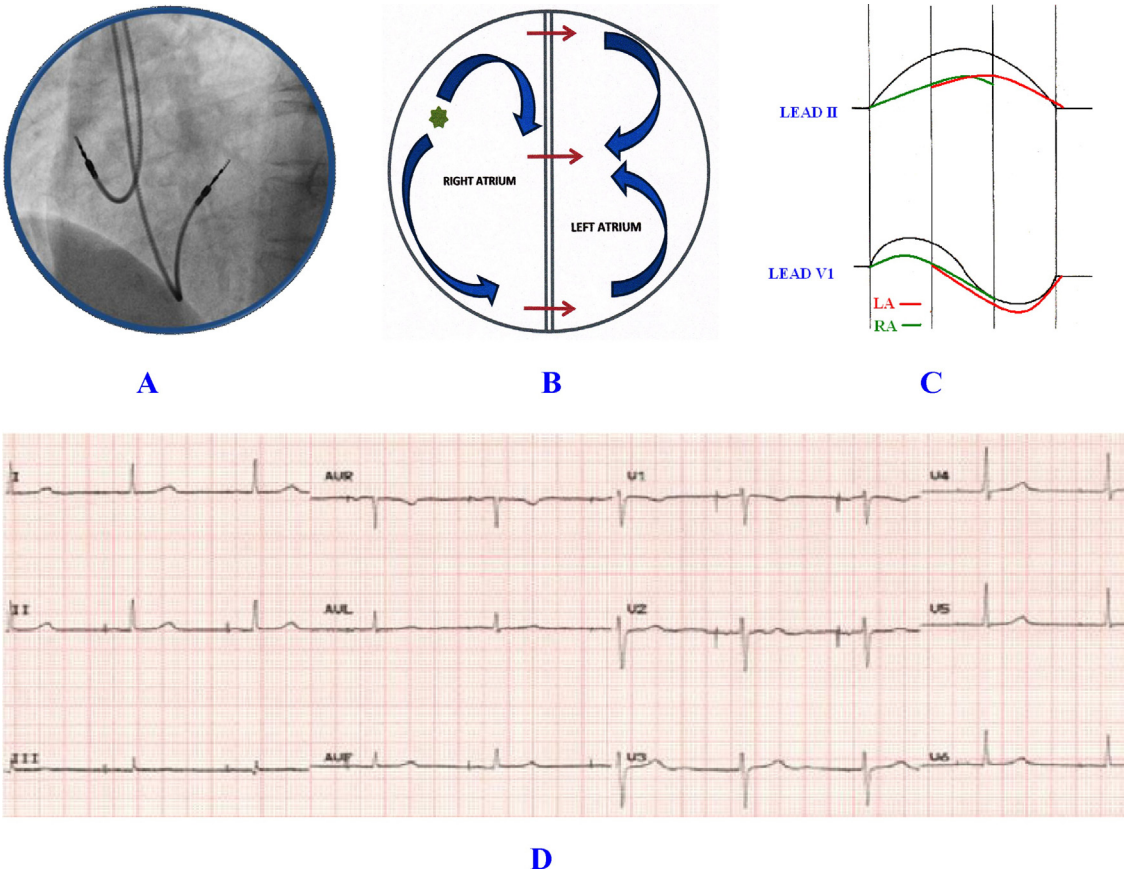

Fig. 2. Conventional right atrial appendage pacing: A, Fluoroscopic left anterior oblique (LAO) view showing atrial lead in right atrial appendage and ventricular lead in interventricular septum, B, Spread of activation wave-front, C, Paced P wave morphology: black line indicates P wave morphology in sinus rhythm. Green line indicates right atrial contribution during pacing and red line indicates left atrial contribution. D, 12 lead surface ECG during atrial pacing at 25 mm/s.

6.2. P wave duration and vector

The typical P-wave morphology in right atrial appendage pacing shows an inferior vector in the frontal plane and the P wave duration is prolonged (equal or more than the sinus P wave duration). The part of the atrial septum on which the lead is screwed is determined according to the polarities of the paced P wave in aVF: total positivity, initial positivity, initial negativity, and total negativity corresponding to four distinct sites starting from the highest to the lowest part of the septum. Pacing from the upper septum (Bachmann's bundle) results in positive paced P-wave in leads I, II, and III which is shorter than the sinus P-wave (expected to be 15–20 ms shorter than sinus P waves). Both the atria get depolarized simultaneously as through the Bachmann's bundle the stimulus goes to the left atrium rapidly and the crista terminalis spread the activation longitudinally in the right atrium from the site of pacing resulting in shortest P wave. When paced from the low atrial septum, the P-wave vector is directed superiorly in the frontal plane (negative in lead II, III and aVF) as the spread of activation occurs in caudo-cranial direction while in the precordial leads the vector is directed anterior and to the right. The P wave duration is short as the left atrium starts depolarizing very early (simultaneously or may be earlier than the right atrium) thereby taking less time for total atrial depolarization.

6.3. P wave morphology in lead V1

The P wave contour is normally smooth and is either entirely positive or entirely negative in all leads except V1 and possibly V2. In the short axis view provided by lead V1, which best distinguishes left- versus right-sided cardiac activity; the divergence of right- and left-atrial activation typically produces a biphasic P wave. The mechanisms of P wave morphological changes in pacing at different sites of right atrium are illustrated in Figs. 2–4. The typical morphology of the P-wave in lead V1 in right atrial appendage pacing is almost identical to sinus rhythm morphology with a terminal negative deflection, caused by the normal activation sequence of the atria, right before left. The P wave in lead V1 in low atrial septal pacing is positive if monophasic this suggests that left atrium is unable to cancel the RA forces and terminally positive if biphasic suggesting that the left atrium negates the initial forces of the right atrium and the terminal forces are formed mainly by the right atrium. An alternative explanation is that the left atrium is depolarized prior to the right atrium when stimulated from the low atrial septum as suggested earlier. Pacing from the upper atrial septum results in a small negative deflection in lead V1.

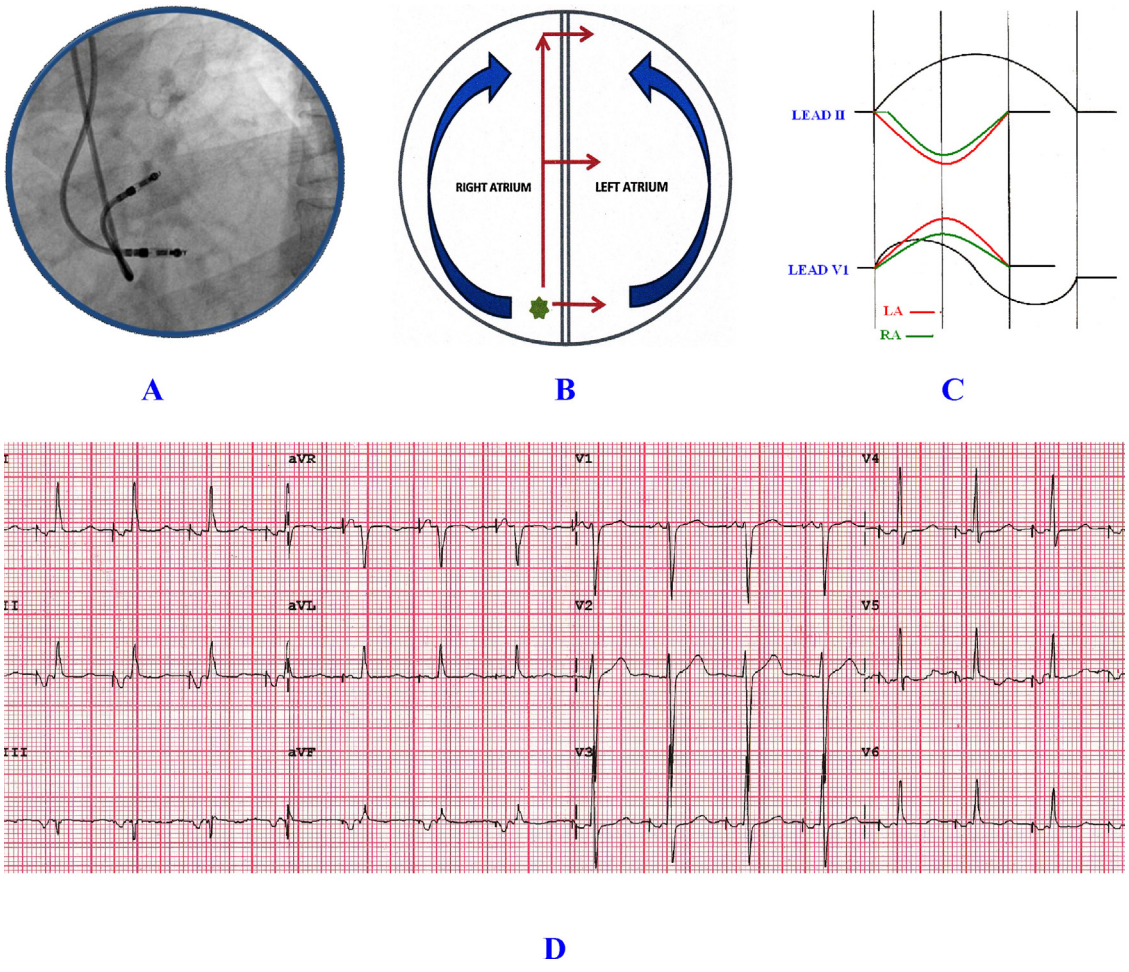

Fig. 3. Lower atrial septal pacing: A, Fluoroscopic left anterior oblique (LAO) view showing atrial lead in lower atrial septum and ventricular lead in interventricular septum, B, Spread of activation wave-front, C, Paced P wave morphology: black line indicates P wave morphology in sinus rhythm. Green line indicates right atrial contribution during pacing and red line indicates left atrial contribution. D, 12 lead surface ECG during atrial pacing at 25 mm/s.

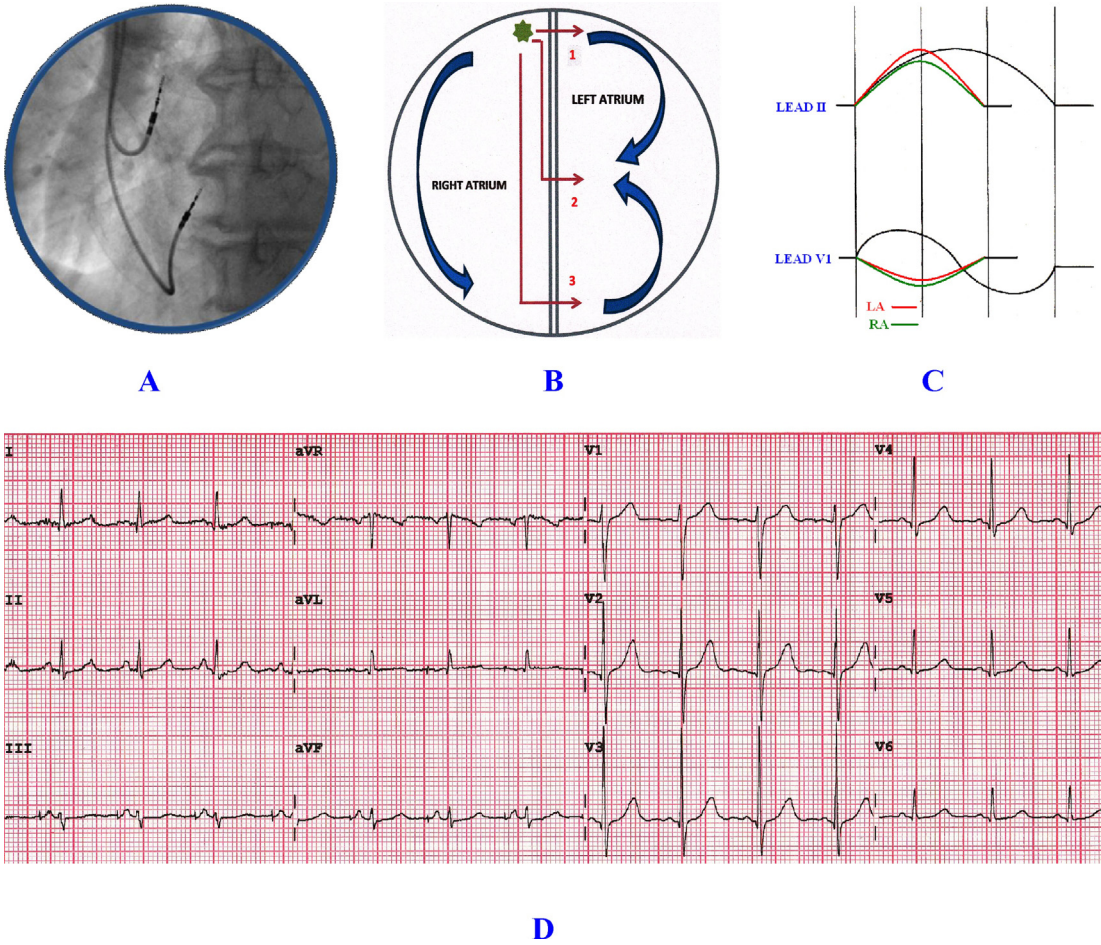

Fig. 4. Bachmann's bundle pacing: A, Fluoroscopic left anterior oblique (LAO) view showing atrial lead in upper atrial septum and ventricular lead in interventricular septum, B, Spread of activation wave-front, C, Paced P wave morphology: black line indicates P wave morphology in sinus rhythm. Green line indicates right atrial contribution during pacing and red line indicates left atrial contribution. D, 12 lead surface ECG during atrial pacing at 25 mm/s.

7. Conclusion

Conventional right atrial appendage pacing results in P wave morphology similar to sinus rhythm with prolonged duration and reduced amplitude. It also results in marked latency in patients with ACD. Atrial septal pacing causes short and sharp P wave. Upper septal pacing results in positive P wave in inferior leads and a small negative deflection in lead V1. Lower septal pacing results in negative P wave in inferior leads and a positive deflection in lead V1.

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