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Original Article

Low interatrial septal pacing: A simple method

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ABSTRACT

Background: Sinus node disease is associated with widespread structural and electrophysiological changes in the atria in addition to abnormalities at the sinus node. The atrial conduction disorder in patients with atrial pacing results in higher incidence of atrial fibrillation. Studies have shown that low interatrial septal pacing is superior to right atrial appendage pacing in preventing persistent or permanent atrial fibrillation in these patients. However, implantation of active fixation lead in low interatrial septal position is difficult and time consuming with conventional stylet, inhibiting application of this method in routine practice.
Method: The technique of implanting atrial pacing lead in low interatrial septum with hand-made stylet is presented in this study with emphasis on fluoroscopic landmark and electrocardiographic P wave pattern.
Results: The results indicate acute and short-term success of low interatrial septal pacing in 10 patients out of 11 patients without major complications. Pacing parameters during implantation and 3 months post procedure were within normal limits.
Conclusion: The initial favorable results of this study indicate low interatrial septal pacing with conventional active fixation lead using fluoroscopic landmark and electrocardiographic characteristics is feasible and reproducible with a simple technique.

1. Introduction

Atrial lead insertion during permanent pacemaker implantation 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 In addition to abnormalities at the sinus node, SND is associated with widespread structural and electrophysiological changes in the atria.2 Atrial conduction disorder is electrocardiographically defined as P wave duration (traditionally measured in lead II) of more than 120 ms.3 This atrial conduction disorder in patients with permanent pacing results in higher incidence of atrial fibrillation.4 The occurrence of atrial fibrillation (AF) after pacemaker implantation in SND is associated with an increased risk of stroke, systemic embolism, heart failure, and mortality.5 Saksena et al. suggested dual-site atrial pacing using electrodes positioned in the high right atrium and at the coronary sinus ostium ("dual-site RA pacing") for prevention atrial fibrillation in these patients.6 However, it requires one extra pacing lead. Two large prospective randomize studies (EPASS and SAFE) have shown that low inter atrial septal (IAS) pacing is superior to right atrial appendage (RAA) pacing in preventing persistent or permanent AF in patients with SND and intra-atrial conduction delay.7,8 De Voogt has described the technique and electrocardiographic features of low IAS pacing in several studies.9 However, for this procedure we need two types of special delivery systems; first is a steerable stylet (Locator) and the second system is a catheter delivery system (Select Secure pacing lead system) which is not widely available.10,11 So, we prepared and used a hand-made stylet (applying a generous curve to the distal end of the straight stylet resulting in a "C" type curve) in this study. Feasibility and safety of this kind of hand-made stylet for lower atrial septal pacing are not well validated.

2. Method

In our hospitals, from 1st February 2015 till 31st January 2016 we had recruited 15 patients for low IAS pacing who were undergoing dual chamber pacemaker. We selected the patients with sinus P wave duration of more than 120 ms in lead II (atrial

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conduction disorder). With hand-shaped ('C" type) stylet we positioned the atrial lead in the target area. The lead parameters (P wave sensitivity, threshold, lead impedance) were noted during implantation and 3 months after the procedure. We used conventional active fixation leads whichever was available at the time of implantation.

After having venous access to the left Subclavian vein, the ventricular lead was placed in the right ventricular apex. The position was observed in left anterior oblique (LAO) fluoroscopic view which served as the reference. The target region for the implantation of the lead in low IAS pacing was the part of the atrial septum just above the coronary sinus ostium (Fig. 1). To locate the lower IAS region, the atrial lead was first advanced into the lower part of the right atrium in Postero-anterior projection. Then the fluoroscope was moved to the 40° LAO position. A generous curve was applied then to the distal end of the conventional straight stylet to form a "C" shapes (Fig. 2). Iterative adjustment to the curve was done according to the shape of the loop of the RV lead in 40° LAO view. This hand made stylet was then advanced through the lead and a counter–clock wise rotation was given on the lead which advanced the lead to the target site. The fluoroscope was then positioned in the 40° right anterior oblique (RAO) view, and the lead was fixed in the anterior position. In both left anterior oblique as well as left lateral view it faced posteriorly towards spine. Fig. 3 demonstrates LAO and RAO view of the leads (ventricular lead positioned at the RV apex and atrial lead positioned at the lower atrial septum).

The fluoroscopic criteria we used for low IAS pacing were: (i) the position of the lead tip in the posterior–anterior view near the indentation of the right ventricular lead at the base of its descendant curve from the superior vena cava (inferior border of tricuspid annulus) and; (ii) in the 40° left anterior oblique view the lead tip pointing posteriorly towards the spine.

The electrocardiographic criteria for low IAS pacing were (i) P wave morphology: negative paced P-wave in inferior leads (lead II, III, and aVF), (ii) Absence of latency: starting immediately with the pacing spike and (iii) P wave duration: shorter (30 ms shorter than sinus P waves measured on the 6-limb lead electrocardiogram).

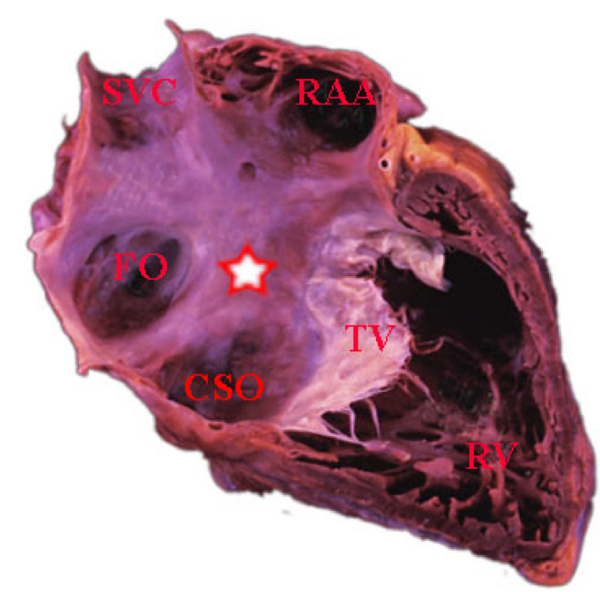


Fig. 1. An anatomic specimen of heart: the right atrial and ventricular free wall has been removed to show interatrial septum as seen in antero-posterior view. SVC = superior vena cava, RAA = right atrial appendage, FO = fossa ovalis, TV = tricuspid valve leaflet, CSO = coronary sinus opening, RV = right ventricle. Star indicates target site for low interatrial septal pacing.

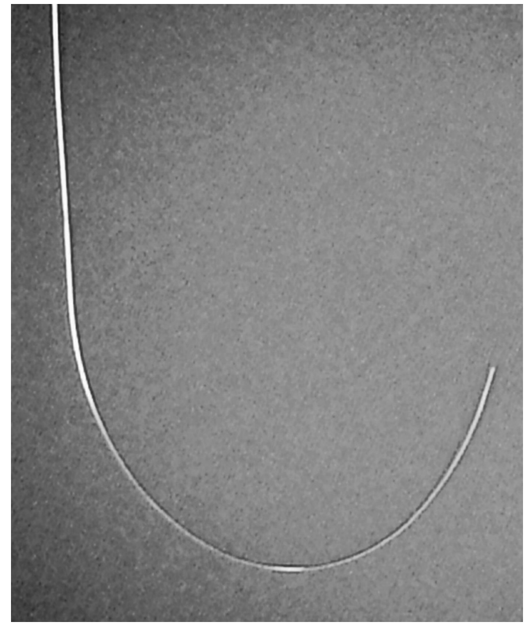


Fig. 2. Hand-made stylet with "C" curve.

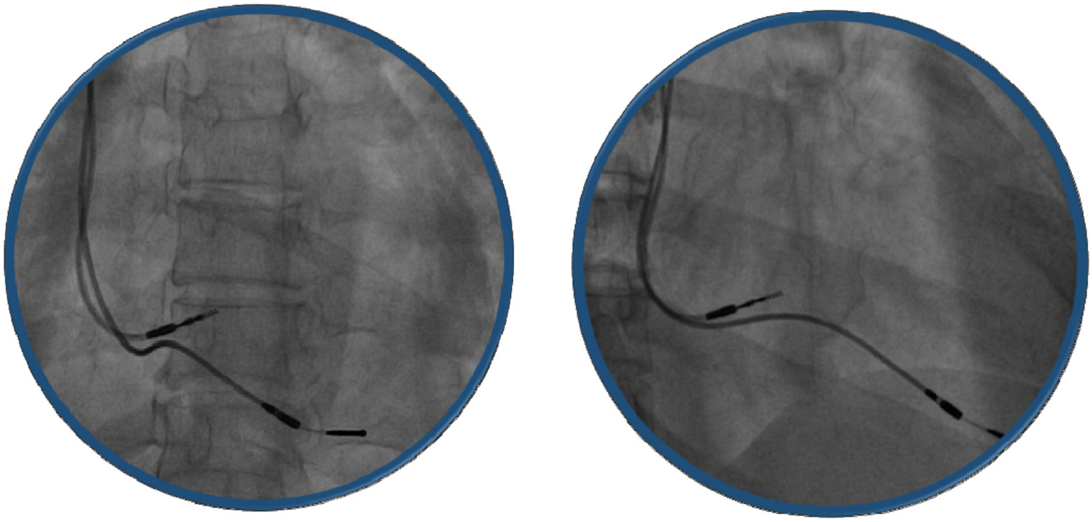
Collected data were analyzed using SPSS 21 statistical package. The test was used to compare the normally distributed continuous variable between the lead parameters during implantation and 3 months post procedure. A p value of less than 0.05 was used to indicate significance. The test was also used to analyze paced P-wave characteristics. The correlation of paced P-wave in lead II, III, aVF versus lead V1 had been seen.

3. Results

We successfully implanted the atrial lead in lower IAS in 14/15 patients. In one patient we fail to position the lead in the target area because of inaccessibility and the lead was placed in right atrial appendage. The right atrium was hugely dilated in this patient. Underlying condition was corrected transposition of great arteries (CCTG). There was no procedural complication. Of these 14 patients, 8 were male and 6 were female. Mean age was 65.5 years (95% CI; 62.16–68.84). The lead position in the low IAS region was electrocardiographically confirmed by noting the paced P wave characteristics (duration, latency, and vector) in 12 lead surface ECG during implantation and on 2nd post-implantation day. The ECG was taken with 50 mm/s paper speed to better calculation of the intervals. Table 1 summarizes the paced P wave features of these 14 patients.

The mean P wave sensitivity at implantation and 3 months post procedure were 2.84 mV (95% CI; 2.48–3.20) and 2.75 mV (95% CI; 2.43–3.07) respectively. All the data had been collected in the bipolar configuration. The change were insignificant (p value is 0.12). Mean pacing threshold at implantation and 3 months post procedure were 0.76 V (95% CI; 0.55–0.95) and 0.76 V (95% CI; 0.65–0.87) respectively. The change in pacing threshold were not significant (p value is 1.0). The mean lead impedance at implantation and 3 months post procedure were 842.5 ohm (95% CI; 768.67–916.33) and 775 ohm (95% CI; 730.04–819.96) respectively. The changes were significant (p value is 0.009).

The mean shortening paced P-wave duration was 40 ms with 95% CI of 40.23–49.77 ms. The two-tailed p- value (compared to an expected mean shortening of at least 30 ms) is 0.0011. This is considered to be statistically significant. The correlation of paced P-wave morphology in lead II, III, aVF versus lead V1 shows a value



A: LAO view **B: RAO view**

Fig. 3. A & B: Fluoroscopic 40° left anterior oblique (LAO) and 40° right anterior oblique (RAO) view in a patient with dual chamber pacemaker showing RV lead positioned at the RV apex and atrial lead positioned at the lower atrial septum.

Table 1
Paced P wave characteristics of 10 patients with pacing lead in low interatrial septum.

Serial No.	Age (Yrs)	Sex	P wave duration (ms)			Latency (ms)	P wave morphology		Lead parameters					
			Sensed (S)	Paced (P)	Shortening (S-P)		Lead II, III, aVF	Lead V1	P wave sensitivity (mA)		Pacing threshold (Volt)		Lead impedance (Ohms)	
									During implantation	After 3 months	During implantation	After 3 months	During implantation	After 3 months
1.	70	Male	120	70	50	0	Negative	Positive	2.2	2.0	0.8	1.0	820	800
2.	72	Male	130	75	55	10	Biphasic II, negative III, aVF	Positive	2.6	2.7	0.6	0.8	795	710
3.	60	Female	120	85	35	0	Negative	Positive	3.1	2.9	1.2	1.0	990	850
4.	64	Male	125	80	45	0	Negative	Positive	3.6	3.5	0.9	0.7	810	765
5.	65	Male	130	85	45	90	Negative	Positive	2.8	2.8	0.4	0.5	1015	815
6.	68	Female	120	80	40	0	Negative	Positive	3.5	3.1	0.7	0.6	910	885
7.	71	Male	135	85	50	5	Negative	Positive	2.0	2.1	0.9	0.7	725	710
8.	58	Female	125	75	50	10	Negative	Positive	2.7	2.6	1.0	0.8	790	770
9.	63	Male	125	80	45	0	Negative	Positive	3.0	3.1	0.6	0.8	705	695
10.	64	Female	120	85	35	0	Negative	Positive	2.9	2.7	0.5	0.7	865	750
11.	66	Male	130	90	40	0	Negative	Positive	2.8	2.9	0.9	0.9	840	820
12.	69	Female	125	95	30	5	Negative	Positive	2.9	2.6	0.8	0.8	782	750
13.	59	Female	120	85	35	0	Negative	Positive	2.6	2.9	0.6	0.6	828	730
14.	68	Male	130	95	35	0	Biphasic II, negative III, aVF	Positive	3.0	2.9	0.8	0.8	920	800

of correlation co-efficient (R) of 0.85 (<0.5 indicates poor co-relation). This result indicates very significant inverse relationship, which means when paced P wave morphology in lead II, III, aVF is negative and negative, there is very high probability of V1 being positive.

4. Discussion

The right and left atria are activated nearly simultaneously (within 50–80 ms) during sinus rhythm. Electrocardiographically P wave duration is traditionally measured in lead II and a value of more than 120 ms is abnormal. Atrial conduction disorders 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.12 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 Bachmann's bundle (BB). Several studies have shown the beneficial effects of low IAS pacing in patients with intra-atrial conduction delay in term of prevention of atrial fibrillation.7–9

4.1. Effects of right atrial appendage pacing

Atrial conduction delay either spontaneous or induced by right atrial appendage pacing, delays left atrial systole and modifies, or even in extreme cases cancels, its contribution to ventricular filling with the resultant risk of left heart AV asynchrony. As a result, this increases risk of diastolic mitral regurgitation and atrial fibrillation. Besides the reduction or loss of atrial contribution and the proportional reduction of overall cardiac performance, delayed left atrial contraction may also induce major neurohumoral changes that contribute to lowering blood pressure through atrial reflexes activated by increased atrial stretch and pressure causing elevation of atrial natriuretic peptides.13 These hemodynamic disorders are particularly important when interatrial conduction delay is long, shifting atrial systole after the beginning of ventricular contraction and against a closed mitral valve, resulting in a derangement similar to 1:1 ventriculoatrial conduction during ventricular pacing. Besides the reduction or loss of atrial contribution and the proportional reduction of overall cardiac performance, delayed left atrial contraction may also induce major neurohumoral changes that contribute to lowering blood pressure through atrial reflexes activated by increased atrial stretch and pressure causing elevation of atrial natriuretic peptides.14,15

Right atrial appendage (RAA) pacing is used to treat Sinus node dysfunction and suppress atrial fibrillation, but may further aggravate the atrial physiology that underlies these conditions and may worsen atrial dyssynchrony.16–18 Pacing the interatrial septum at either upper part (Bachmann's bundle region) or at lower part (above the coronary sinus ostium) has been advocated to achieve or maintain atrial synchrony and prevent atrial fibrillation.19,20

Two large prospective randomize studies (EPASS and SAFE) have shown that low inter atrial septal (IAS) pacing is superior to RAA pacing in preventing persistent or permanent AF in patients with SND and intra-atrial conduction delay.7,8 For patients with SND without atrial conduction delay with history of AF, trial reports are controversial. Wang M et al., have demonstrated that right low AS pacing in SND patients with paroxysmal AF who have a dual-chamber pacemaker achieve better regional right and left atrial active mechanical properties and LA hemodynamic performance compared with those with RAA pacing.21 Inter-atrial electromechanical dyssynchrony is also reduced with right low IAS pacing. Padeletti et al. showed that rate-adaptive pacing at the triangle of Koch is more effective than RAA pacing in preventing symptomatic recurrences of paroxysmal AF in patients with sinus bradycardia and a history of AF.22 However, 2 subsequent prospective, randomized studies in similar patient populations failed to demonstrate this superiority of IAS to conventional appendage pacing.23,24

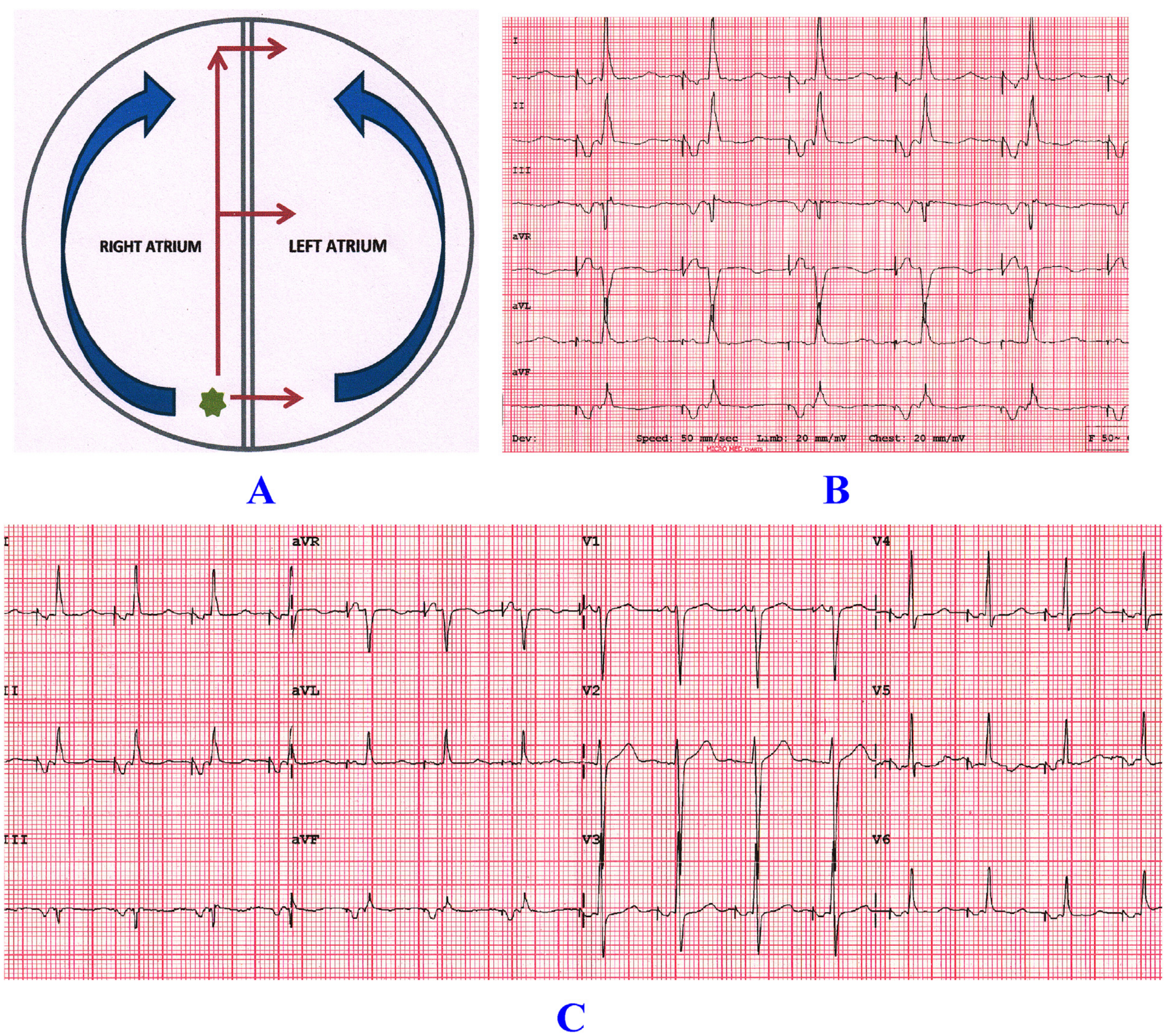


Fig. 4. Low Interatrial Septal pacing: A; Spread of activation wave-front – the depolarization wave front rapidly crosses to the left atrium along the musculature around the proximal coronary sinus. Both the atria get stimulated in a caudo-cranial direction (reverse to normal sinus rhythm and just opposite of Bachmann's bundle pacing). So, it produces P waves with superior axis (negative in inferior leads). As, the propagation time is short and both the atria get stimulated simultaneously, it produces narrow P wave, B; 6 lead surface ECG shows appearance of P wave during low IAS pacing with a paper speed of 50 mm/sec, C; 12 lead surface ECG during low IAS pacing with a paper speed of 25 mm/s.

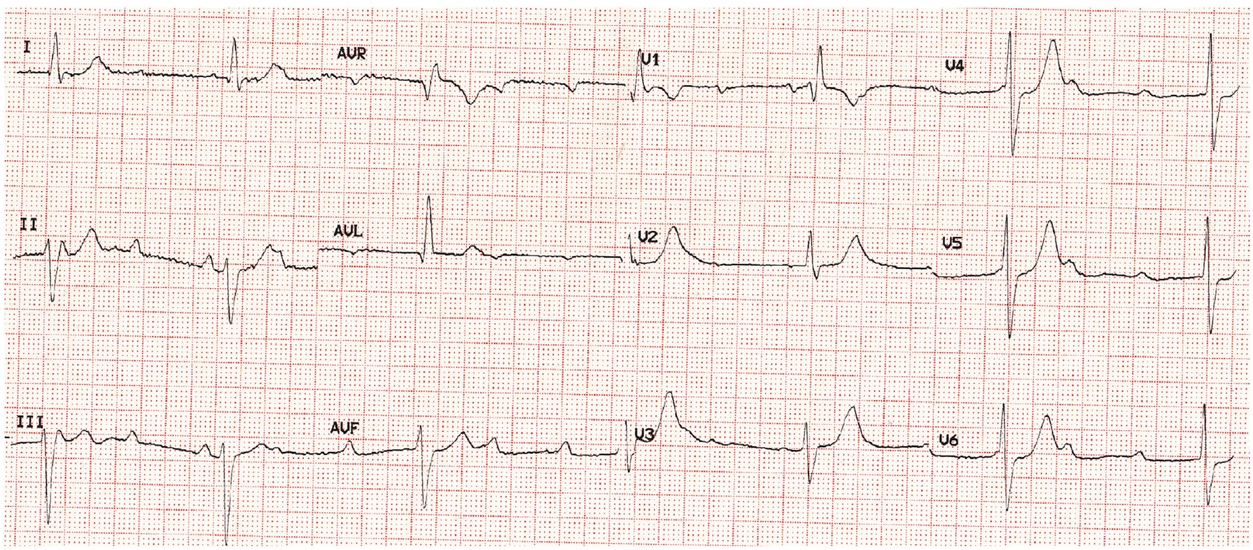
4.2. Advantages of low IAS pacing

The main benefits that this selective site for right atrial pacing provides are[1]: a very short interatrial conduction delay and a significant decrease in P-wave duration[2]; a reduction in dispersion of atrial refractoriness[3]; a more homogeneous recovery of excitability and atrial activation.

However, as previously suggested low RA septal pacing may be effective only in patients with intraatrial conduction delay whereas patients with interatrial conduction delay needs pacing at the upper atrial septum near the region of Bachmann's bundle.[7–25]

4.3. Electrocardiographic characteristics

In patients with right atrial appendage pacing, 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 with prolonged paced P wave equal or more than the sinus P wave duration.[26] In low IAS pacing (above the coronary sinus ostium), the wave of depolarization rapidly traverses to the left atrium via the musculature around the proximal part of the coronary sinus and excites the left atrium in a caudo-cranial



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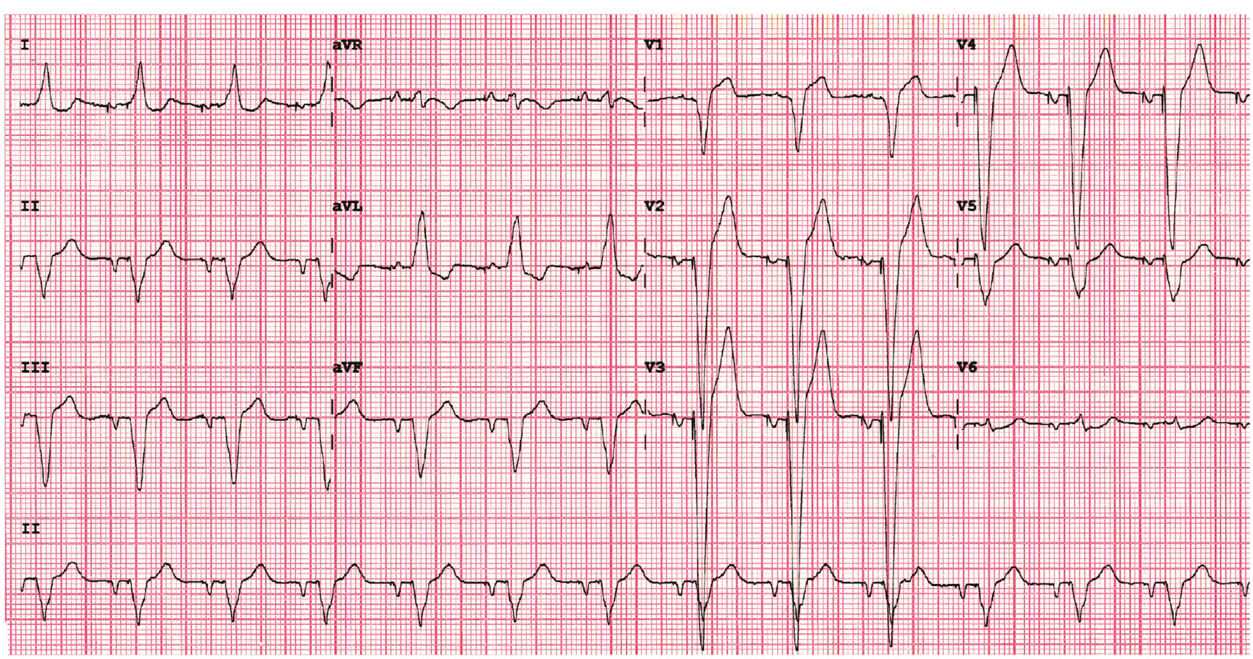

B

Fig. 5. Low IAS pacing: A; 12 lead base-line ECG shows advanced AV block with a background of trifascicular block with atrial conduction disorder, B; 12 lead surface ECG during DDD pacing. Note the paced P wave morphology in lead II, III, aVF and V1. Also, note the duration of paced P wave.

direction and in the right atrium via crista terminalis to the whole right atrium. So, there is no latency (starting immediately with the pacing spike) period and the P wave duration is short (shorter than the sinus P-wave; expected to be more than 20 ms shorter than sinus P waves) (Fig. 4).27 The paced P-waves in low IAS pacing are negative in leads II, III, aVF and small positive deflection in lead V1 (Fig. 5). In the short axis view provided by lead V1, the P wave in lead V1 in low interatrial 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.

5. Conclusion

Delivering the atrial lead to the lower atrial septum is difficult with conventional stylets. Special tools like steerable stylet or steerable catheters are required frequently. The method of lower IAS pacing with conventional stylets is not well elucidated. Our study showed that the lower IAS pacing is feasible and reproducible with a simple technique (hand-made stylet).

Conflict of interest statement

None.

Study limitations

The present study has several limitations: 1) very small sample size, 2) short term follow up (for only 3 months), and most importantly 3) didn't include data collection during follow up with respect to the occurrence of atrial arrhythmia and pacing burden. Further studies that involve larger sample of patients and longer follow up period are required to confirm the present findings. Additionally, preprocedural echocardiographic evaluation to provide an idea about the required curve for particular patient should be investigated

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