Single-Site Bachmann’s Bundle Pacing Is Beneficial While Coronary Sinus Pacing Results in Echocardiographic Right Heart Pacemaker Syndrome in Brady-Tachycardia Patients

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**Background:** It has been proposed that multisite atrial pacing (MSAp) restores atrial electrical activation and prevents atrial fibrillation recurrence; however, single-site Bachmann’s bundle pacing (BBp) has also been reported as providing effective atrial resynchronization. Coronary sinus pacing (CSp) leads to reversed impulse propagation within the atria.

**Methods and Results:** Acute echocardiographic examination was performed in 15 healthy subjects, and in 25 patients with sinus node dysfunction and recurrent atrial fibrillation during MSAp (atrial leads in the BB area and CS ostium), and single-site BBp and CSp. Regional atrial synchrony was assessed by tissue Doppler echocardiography. Pacing mode had no effect on stroke volume. CSp resulted in right atrial filling diminution, shortened mechanical atrioventricular delay in the right heart and diminished right ventricular inflow. The magnitude of reversal of the physiological right-to-left atrial contraction sequence was most prominent during CSp (15±11, −12±23, −3±21, −42±23 ms; control, MSAp, BBp, CSp respectively, P<0.0001). BBp provided the best atrial contraction synchrony, and had a comparable effect on global cardiac function to MSAp.

**Conclusions:** Single-site BBp provides comparable hemodynamics to MSAp and is sufficient to restore atrial contraction synchrony. Single-site CSp induced echocardiographic pacemaker syndrome in the right heart. *(Circ J 2010; 74: 1308–1315)*

**Key Words:** Atrial fibrillation; Atrial pacing; Echocardiography; Hemodynamics; Right heart pacemaker syndrome

**Pacemaker syndrome** is a complex of adverse clinical, hemodynamic and electrophysiological signs resulting from disruption of appropriate atrioventricular (AV) synchrony. Typically pacemaker syndrome is described in patients with a single-chamber ventricular pacemaker and preserved ventriculo-atrial conduction, or a dual-chamber pacemaker with inappropriate AV delay timing; however, it has also been noted as a consequence of prolonged intra- or interatrial conduction times. Symptoms associated with pacemaker syndrome may vary from mild to severe, and in many patients are not even noted until the proper timing between atrial and ventricular contractions is restored.

Pacemaker syndrome during coronary sinus pacing (CSp) can be related to a change in impulse propagation within the atria and earlier ventricular activation. Our previous work on the hemodynamic consequences of different atrial pacing modes showed that pacing from the distal CS caused reversal of the physiological right-to-left atrial contraction pattern and diminished right atrial (RA) filling.

The RA appendage is the conventional atrial lead position; however, it has been reported that pacing at this site prolongs interatrial conduction and changes the electrophysiological properties of the atria, which may promote supraventricular arrhythmias. Alternate RA pacing sites, mainly in the region of the Bachmann’s bundle (BB) or the CS area, have been introduced, aimed at improving the atrial electrical synchrony and antiarrhythmic effect. Other investigators have proposed multisite atrial pacing (MSAp) as a method of atrial fibrillation (AF) prevention; however, to date neither the optimal atrial pacing sites nor the advantage of multisite atrial pacing has been determined. Despite numerous studies of the hemodynamic aspects of pacing at alternate atrial sites, a direct comparison of their impact on cardiac function and atrial contraction synchrony has not been...
The aim of the study was to determine the adverse hemodynamic effects of CSp and to investigate which of the 3 atrial pacing modalities (ie, BBp, CSp, or MSAp) provides the best/worst parameters of global cardiac function and synchrony. We assumed that the number of pacing sites, as well as the direction of impulse propagation during pacing at different atrial sites, would have an influence on regional atrial synchrony and global cardiac performance.

The investigation was conducted in patients with sinus node dysfunction, AF recurrence and prolonged sinus P-wave duration on the surface ECG who had been treated with MSAp. Prolonged P-wave duration can indicate the presence of interatrial conduction disturbances. The choice of optimal site of preventive atrial pacing, which preserves atrial and AV contraction synchrony, and has a beneficial influence on atrial remodeling and thus vulnerability to arrhythmias, is an important issue in this population.

Methods

Patient Population

Acute echocardiographic examination was performed in 30 consecutive patients treated with a novel configuration of MSAp incorporating the BB and the CS ostium region. Indications for pacemaker implantation comprised: (1) sinus node dysfunction with class I or II indications for permanent cardiac pacing; (2) documented symptomatic AF recurrence (paroxysmal or persistent); (3) signs of advanced intra- and interatrial conduction delay (ie, sinus P-wave duration ≥120 ms on the surface ECG); (4) no evidence of 2nd or 3rd degree AV block. Five patients were excluded from the echocardiographic study because of severely depressed global left ventricular systolic function (1 patient), moderate mitral valve disease (1 patient), or documented AF within 1 month preceding the examination (3 patients).

A standard dual-chamber pacemaker was implanted in all patients. One atrial lead was connected to the atrial (A) port, and the other to the ventricular (V) port. The atrial lead connected to the A-port was placed in the BB region, and the atrial lead connected to the V-port was implanted at the CS ostium area. The target region to implant the BB lead was the confluence of the RA roof and the interatrial septum. The implantation procedure in the BB area was described previously. The ostial CSp was confirmed on the ECG by negative P-waves in the inferior limb leads. All pacemakers were programmed to the DDD mode, or the DDD-R mode in patients with chronotropic incompetence, with an interatrial delay of 15 ms (the stimulus in the CS delayed by 15 ms with regard to the BB region).

Figure 1. Atrial contraction sequence (PA<sub>m-t</sub>) calculated as the time difference between the beginning of the mitral and tricuspid atrial waves assessed by pulsed-wave Doppler echocardiography. Recording during single-site coronary sinus pacing. Time from the pacing spike till the beginning of the tricuspid atrial wave (PA<sub>t</sub>=129 ms, Left), and mitral atrial wave (PA<sub>m</sub>=69 ms, Right). PA<sub>m-t</sub>=PA<sub>m</sub>−PA<sub>t</sub>=69 − 129 ms = −60 ms. Negative sign indicates reversion of the physiological atrial contraction sequence from right-to-left to left-to-right.
In order to relate the results of atrial contraction sequence and synchrony obtained with different atrial pacing modalities to the physiological pattern, echocardiographic examination was also performed in a control group of 15 subjects with no history of any cardiovascular disease, no pacemaker and with normal ECG recordings.

The local ethical committee approved the study protocol, and all patients gave their written informed consent to MSA pacemaker implantation and participation in the study.

**Study Protocol**

In each patient, the echocardiographic examination was performed during MSAp and single-site atrial pacing (BBp, CSp) evaluated in random order. The basic pacing rate was programmed to exceed the intrinsic rate by at least 10 beats/min and was constant during the study. Evaluation of the echocardiographic parameters was performed after a 5-min resting period following initiation of each mode to allow stabilization of the variables measured, and data acquisition occurred during the next 5–10 min. We also performed the examination during sinus rhythm (SR) in patients with intrinsic SR >40 beats/min.

**Echocardiography**

Studies were performed with a commercially available instrument (Vivid 7, GE Healthcare, Horten, Norway) using a 3.4-MHz phased-array transducer. Doppler studies were made in the left lateral decubitus position. One echocardiographer, blind to the pacing mode, performed all data acquisition and measurements. Mitral and tricuspid inflows were recorded with pulsed-wave Doppler at the level of the tips of the mitral (m) and tricuspid (t) leaflets during apnoea, and the following parameters were assessed: diastolic filling time (DFT) and diastolic velocity time integral (VTI), atrial wave duration (Adur), atrial VTI (VTI-A), deceleration of the atrial wave (DTA), time interval from the beginning of the P-wave or pacing spike on the ECG till the beginning of the atrial wave (PA). Pulmonary and aortic outflow spectra were recorded with continuous-wave Doppler and the VTI at the level of the respective valves was registered (VTI_pulm/VTI_Ao). The following parameters were calculated: the percentage of Adur in relation to the total DFT (%A), mechanical AV delay in the right and left heart, defined as the time between the beginning of the mitral/tricuspid atrial wave and the beginning of the aortic/pulmonary outflow spectrum (AV-RV/AV-LV). Additionally, global atrial contraction sequence was calculated as the time difference between the beginning of the mitral and tricuspid atrial waves: PA_m-t = PA_m - PA_t (Figure 1).

The influence of atrial pacing mode on the atrial electromechanical delay was assessed by means of pulsed-wave tissue Doppler.\(^{19}\) A 6-mm wide sample volume was placed in the middle segment of the lateral left and RA walls. The time distance was measured from the beginning of the P-wave or atrial pacing spike on the ECG until the beginning of the pulsed-wave tissue Doppler A’-wave in the lateral LA (s-LA) and lateral RA (s-RA) walls. The regional interatrial delay (interA) was calculated as the time difference between s-LA and s-RA (Figure 2).

All analyses were performed off-line on an EchoPac workstation (GE, version 2.0.6) with a sweep of 100 mm/s. Parameters obtained from at least 3 consecutive beats were measured and the average was calculated.

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**Figure 2.** Regional interatrial delay (interA) calculated as the time difference between the pacing spike and the beginning of the tissue Doppler A’ wave at the level of the lateral left atrial wall (s-LA = 52 ms, **Right**) and lateral right atrial wall (s-RA = 138 ms, **Left**). \(\text{interA} = \text{s-LA} - \text{s-RA} = 52 - 138 \text{ms} = -86 \text{ms}\). Recording during single-site coronary sinus pacing.


**Table 1. Atrial Contraction Sequence (PA<sub>Asc</sub>) and Regional Interatrial Delay (InterA) in the Control Group and Study Population During SR and Different Atrial Pacing Modes**

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Study group</th>
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<tbody>
<tr>
<td></td>
<td>P value</td>
<td></td>
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<tr>
<td></td>
<td>(ANOVA)</td>
<td></td>
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<tr>
<td>PA&lt;sub&gt;Asc&lt;/sub&gt; (ms)</td>
<td>15±1*</td>
<td>44±13‡</td>
</tr>
<tr>
<td>InterA (ms)</td>
<td>19±13</td>
<td>60±24‡</td>
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Scheffé post-hoc tests for PA<sub>Asc</sub>: *P<0.001 control vs SR and MSAp, †P<0.001 SR vs MSAp, BBp and CSp, ‡P<0.001 CSp vs other; interA: *P<0.0001 SR vs other, †P<0.05 MSAp vs BBp, ‡P<0.0001 CSp vs other. PA<sub>Asc</sub> time difference between the beginning of the mitral and tricuspid atrial wave; InterA, interatrial delay assessed by tissue Doppler echocardiography at the level of the lateral left and right atrial walls; SR, sinus rhythm; MSAp, multisite atrial pacing; BBp, Bachmann’s bundle area pacing; CSp, coronary sinus ostium pacing.

**Table 2. Clinical and Echocardiographic Characteristics of the Study Population**

<table>
<thead>
<tr>
<th>Study population (n=25)</th>
<th>Control group (n=15)</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>66±9</td>
</tr>
<tr>
<td>Males (n)</td>
<td>8</td>
</tr>
<tr>
<td>Structural heart disease (n)</td>
<td>4</td>
</tr>
<tr>
<td>Coronary artery disease (n)</td>
<td>4</td>
</tr>
<tr>
<td>Arterial hypertension (n)</td>
<td>22</td>
</tr>
<tr>
<td>Minor valvular heart disease (n)</td>
<td>3</td>
</tr>
<tr>
<td>LA (mm)</td>
<td>41±5</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>48±9</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>63±8</td>
</tr>
</tbody>
</table>

Data are presented as the number of patients (n), or mean±SD. *P<0.05. LA, left atrium; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction.

**Results**

The characteristics of the study population and the control group are presented in Table 2. Each patient in the study population had a normal left ventricular ejection fraction, and slightly enlarged LA diameter. AF was persistent in 9 and paroxysmal in 16 patients from the study group. At the time of the echocardiographic study, 23 patients were being treated with antiarrhythmic drugs (Class 1: 11; β-blockers: 5; amiodarone: 2; sotalol: 4; calcium-channel blockers, 1). Moreover, 21 patients had received angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, and statins had

**Table 3. Electrocardiographic Parameters at the Time of the Acute Echocardiographic Study in the Control Group and in the Study Population During SR and Different Atrial Pacing Modes**

<table>
<thead>
<tr>
<th></th>
<th>Control group (n=15)</th>
<th>Study group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>(n=17)</td>
<td>(n=25)</td>
</tr>
<tr>
<td></td>
<td>(n=25)</td>
<td>(n=25)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>73±6</td>
<td>53±9*</td>
</tr>
<tr>
<td>P-II (ms)</td>
<td>118±12</td>
<td>142±15†</td>
</tr>
<tr>
<td>PQ (ms)</td>
<td>183±18</td>
<td>194±36</td>
</tr>
</tbody>
</table>

P<0.0001 control group and MSAp vs SR, BBp and CSp.

ECC Measurements

Standard 12-lead ECG was recorded with paper speed of 50 mm/s and 2 mV/cm calibration. P-wave duration and the pacing spike–Q interval (PQ) was measured manually in the II or III limb lead during SR and each atrial pacing modality.

**Statistical Analysis**

Data are presented as mean±standard deviation (SD). For continuous variables with normal distribution, the Student’s t-test for independent samples was used. If variables did not follow normal distribution, the U Mann-Whitney test was applied for comparisons of independent measurements. Differences between categorical variables were tested with the chi-square test. Analysis of variance (ANOVA) for repeated or independent measures was used to compare the hemodynamic variables during different pacing modes. When significant difference (P<0.05) was found, the Scheffé procedure was used as a post-hoc test to locate the difference. The primary aim of the study was to investigate 3 atrial pacing modalities (BBp, CSp or MSAp) and the inference was based on the analysis of variance for repeated measures. The data of the blood pool Doppler parameters in the control group and in the study population during SR were collected and showed for the general sense purposes. Significantly lower heart rate during SR in comparison with the pacing modes and huge patient specificity (mitral and tricuspid inflow depend on age, diastolic heart function, valvular competence, volume status, blood pressure, and many other factors) precludes an objective comparison of the blood pool Doppler parameters between the healthy subjects and the patients with bradycardia and pacing. Comparison of the normal mitral inflow (from the control group) with, for example, the “impaired relaxation” profile from the study population might lead to spurious conclusions. In contrast to the blood pool Doppler parameters, heart rate exerts only minor influence on mechanical dysynchrony, because it deals with the relative activation sequence and in our opinion such a comparison could be performed among the different atrial pacing modes, SR and the control group (Table 1). Those data were related to others by means of ANOVA for independent measures. Spearman’s correlation index was applied to estimate correlations of continuous variables. STATISTICA software (version 8.0, StatSoft Inc) was used to calculate statistics. P<0.05 was considered statistically significant.

**Hemodynamic Consideration of Atrial Pacing**

The primary aim of the study was to investigate 3 atrial pacing modalities (BBp, CSp or MSAp) and the inference was based on the analysis of variance for repeated measures. The data of the blood pool Doppler parameters in the control group and in the study population during SR were collected and showed for the general sense purposes. Significantly lower heart rate during SR in comparison with the pacing modes and huge patient specificity (mitral and tricuspid inflow depend on age, diastolic heart function, valvular competence, volume status, blood pressure, and many other factors) precludes an objective comparison of the blood pool Doppler parameters between the healthy subjects and the patients with bradycardia and pacing. Comparison of the normal mitral inflow (from the control group) with, for example, the “impaired relaxation” profile from the study population might lead to spurious conclusions. In contrast to the blood pool Doppler parameters, heart rate exerts only minor influence on mechanical dysynchrony, because it deals with the relative activation sequence and in our opinion such a comparison could be performed among the different atrial pacing modes, SR and the control group (Table 1). Those data were related to others by means of ANOVA for independent measures. Spearman’s correlation index was applied to estimate correlations of continuous variables. STATISTICA software (version 8.0, StatSoft Inc) was used to calculate statistics. P<0.05 was considered statistically significant.
been administered to 11 patients. The control group was younger, had shorter P-wave duration and smaller LA dimensions than the study population.

Influence of the Atrial Pacing Mode on the ECG (Table 3)
We were able to record SR in only 17 of the 25 patients with MSAp studied because of sinus bradycardia (<40 beats/min), escape nodal rhythm or non-sinus atrial rhythm. Heart rate in the study population was significantly lower during SR (53 ± 9 beats/min) than during atrial pacing or in the control group. P-wave duration during SR in the study population was significantly lower during SR (53 ± 9 ms) compared with SR or BBp modalities. CSp caused a significant delay in the onset of the RA wave (PA), with concomitant abbreviation of the mechanical AV delay in the right heart (AV-RV). Additionally, CSp resulted in diminution of the RA wave: short RA wave duration (Adur, %), low VTI, velocity time integral of the mitral/tricuspid inflow; Adur, atrial wave duration; %A, percentage of atrial wave duration in relation to the total diastolic filling time; VTI-A, atrial velocity time integral; DTA, deceleration of the atrial wave; PA, time interval from the beginning of the P-wave or pacing spike on the ECG till the beginning of the atrial wave; AV-RV/AV-LV mechanical atrio-ventricular delay in the right/left heart; VTI_{booth_m}, velocity time integral at the level of the pulmonary/aortic valve. Other abbreviations see in Table 1.

Cardiac Function
Detailed data on global cardiac function during MSAp and the 2 single-site atrial pacing modes are presented in Table 4. Statistical analysis (ANOVA for repeated measures) was performed only for the pacing modes. No differences were found between MSAp and BBp with regard to the mitral or tricuspid valve inflow parameters, the right or left mechanical AV delay, or the velocity time integral of the pulmonary or aortic valve. Only the onset of the LA wave (PA) in relation to the pacing spike during MSAp occurred earlier than during BBp or CSp modalities.

Several detrimental hemodynamic effects in the right heart were observed with CSp compared with MSp or BBp. CSp caused a significant delay in the onset of the RA wave (PA), with concomitant abbreviation of the mechanical AV delay in the right heart (AV-RV). Additionally, CSp resulted in diminution of the RA wave: short RA wave duration (Adur and %A), low VTI (VTI-A), and prominent deceleration time (DTA) (Figure 3). The premature abbreviation of the RA wave caused diminution of VTI compared with the other pacing modes.

During CSp a significant correlation was observed between %A and AV-RV (R = 0.60; P < 0.01), as well as between %A and the time integral from the pacing spike till the Q wave on the ECG (R = 0.50; P < 0.001). The longer the pacing spike–Q wave interval or AV-RV, the longer the RA wave.

Data obtained in the control group and in the study population during SR are also presented in Table 4, however statistical deduction was based on the analysis of data during three studied pacing modes.

Atrial Contraction Sequence and Synchrony (Table 1)
Important global and regional mechanical interatrial dysynchrony was present in the study group during SR. Single-site
CSp resulted in prolonged electromechanical delay in the lateral RA (s-RA: 108±23, 69±23, 62±20 ms for CSp, BBp and MSAp, respectively; P<0.0001; CS vs other), which led to major regional interatrial delay and reversion of the physiological right-to-left atrial contraction sequence (negative PA_m-t). The electromechanical delay in the lateral LA was longer during BBp than during CSp or MSAp (s-LA: 86±24, 66±22, 62±22 ms for BBp, CSp, and MSAp, respectively; P<0.0001; BB vs other); however, only this mode resulted in comparable (P=NS) atrial contraction sequence and synchrony to the control group. Multisite atrial pacing caused only minor reversion of the physiological right-to-left atrial contraction pattern, because of premature onset of LA contraction (see PA_m in Table 4). During all the examined pacing modes, a significant correlation was observed between the atrial contraction sequence and regional interatrial delay (R=0.53; P<0.0001).

**Discussion**

Our study demonstrated that in patients with sinus node dysfunction, recurrent AF and interatrial conduction disturbances, the atrial pacing site has a major influence on cardiac hemodynamics and contraction synchrony. Single-site CSp resulted in several detrimental hemodynamic effects in the right heart because of significant prolongation of the electromechanical delay in the lateral RA wall, which resulted in retardation of RA contraction. It reversed the physiological right-to-left atrial contraction sequence, shortened the RA wave and diminished right ventricular inflow. In our study, MSAp had no additional beneficial effect over single-site BBp. Parameters of global cardiac function were comparable during these 2 pacing modalities; however, BBp provided more physiological atrial contraction sequence and synchrony.

In patients with interatrial conduction disturbances and AF recurrence, MSAp has been proposed for maintaining SR. The main objective of this therapy is to shorten the total atrial activation time, and diminish the influence of atrial conduction delays on AF induction and/or maintenance. Several methods of MSAp have been proposed, mainly incorporating 1 lead at the RA appendage and the other at the CS ostium (dual-site RA pacing) or in the mid- or distal CS (bi-atrial pacing). Recently, our group proposed a novel method of MSAp incorporating the BB region and CS ostium (“double-resynchronization”). The clinical effects of this technique of MSAp in reducing AF recurrence have been promising, especially if used in combination with antiarrhythmic drugs; however, its main drawback is the number of leads that have to be implanted in the atrium.

To date, the RA appendage has been the conventional location for the atrial lead. However, it has been reported that pacing from this site leads to atrial activation prolongation...
ventricular mechanical AV delay and a decrement in the ventricular activation caused marked shortening of the right ventricular mechanical delay in the lateral RA, which caused retardation of the time sequence of electromechanical delay because of significant prolongation of the electromechanical delay. Single-site CSp was accompanied by an important interatrial conduction delay. Diminished RA contribution to ventricular filling during CSp. We found that the shorter the right ventricular mechanical AV delay, the greater the RA wave diminution. We do not have data on the clinical effect of RA wave shortening with CSp, as in all the patients MSAnp was implemented during long-term follow-up. However, from the echocardiographic point of view, we could observe the right heart pacemaker syndrome with marked abbreviation of the Doppler RA wave, a phenomenon that is nicely demonstrated in Figure 3. Hemodynamic consequences of the typical "pacemaker syndrome" were described in 1889 by McWilliam, but it was not until 1969 that Mitsui et al attributed the clinical symptoms to disturbances in AV contraction synchrony. The clinical consequences of CSp need to be determined in further studies. Data on the harmful effect of CSp on RA function are rare, as most studies have focused on LA function and its beneficial electrical properties. Only a few studies have assessed the RA function during CSp. Kutarski et al investigated the influence of CSp on signal-averaged P-wave time domain parameters and demonstrated that in patients with interatrial conduction disturbances, CSp reduced LA late potentials, but at the same time failed to restore homogeneity of activation within the RA.

As mentioned in the statistical section, data for the blood pool Doppler parameters in the control group and during SR in the study population are provided for general purposes only. However, it can be seen that during SR in the study population the DFT was prolonged and %A diminished. This phenomenon can be attributed to the diastasis phase, which becomes prominent during bradycardia. In order to maintain cardiac output at a constant level, all the VTIs are higher during sinus bradycardia than during pacing. The interval from the beginning of the P-wave till the onset of the A-wave is shorter during sinus bradycardia because of measurement differences (from the beginning of the P-wave and not the onset of the pacing spike) and shorter electromechanical delay. The only parameters that can be compared are Adur, VTI-A and DTA, which are comparable at the mitral level; however, at the tricuspid level the atrial wave during CSp is abbreviated, even in comparison with SR.

In the control group the DFTs are longer because of the younger age and lack of concomitant heart disease. Similarly, the VTIs are larger because of the higher cardiac output. It is even difficult to compare the parameters of the atrial waves because a healthy heart is less atrial-dependent than a sick one, and Adur is shorter. The interval from the beginning of the P-wave till the onset of the A-wave is much shorter than during pacing, but also shorter than during SR in the study population as a consequence of faster electromechanical coupling. The mechanical AV delay is shorter in the control group because of the shorter A-wave.

**Clinical Implications**

In our opinion, appropriate atrial synchrony may improve its mechanical efficacy and have a beneficial long-term effect on pacing-induced remodeling and AF prevention. Our results may have important clinical implications in patients with prolonged P-wave duration on the surface ECG, recurrent AF.
and indications for permanent cardiac pacing. Single-site BBp may be sufficient to restore atrial synchrony, and its global hemodynamic effect is comparable with that of MSAp. Results from the study underline the unfavorable effects of single-site CSp because of the significant abbreviation of the RA contribution to right ventricular filling.

Study Limitations
This was an acute study assessing the hemodynamic effects of different atrial pacing modalities on the echocardiographic parameters of regional and global cardiac function. We can only speculate on the comparable antiarrhythmic effect of BBp and MSAp, and the clinical implications of echocardiographic right heart pacemaker syndrome caused by CSp. Long-term observation of patients in whom permanent BBp or CSp has been implemented would be necessary to determine this issue.

Conclusions
Our data demonstrate that atrial pacing site has an impact on atrial synchrony. Single-site BBp provides the best atrial conduction synchrony in patients with atrial conduction abnormalities, and has a comparable effect on global function to MSAp. Single-site CSp causes echocardiographic pacemaker syndrome in the right heart because of simultaneous retardation of RA contraction and earlier ventricular activation.

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Disclosures
The authors declare no conflicts of interest, and no relationships with industry.

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