Post-Pacing T Loop Abnormalities

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SUMMARY

Vectorcardiograms of paced and unpaced beats were recorded from 18 patients with implanted pacemakers to investigate the characteristics of post-pacing T inversions. The intraventricular conduction during sinus rhythm of unpaced beats was normal in 13 of 18 patients and abnormal in the other 5 (RBBB in 3, LBBB in 2).

The directions of the maximum QRS vector in paced beats (max QRSp.V) and maximum T vector in unpaced beats (max T unp.V) were studied. In 8 of 13 patients with normal intraventricular conduction (group A) and in 3 patients with right bundle branch block (RBBB), the differences between the directions of max QRSp.V and max T unp.V in both frontal and horizontal planes were less than 30 degrees, and in 5 of 13 patients with normal intraventricular conduction (group B) and 2 patients with left bundle branch block (LBBB), differences exceeded 30 degrees. The direction of max T unp.V tended to be superiorly, posteriorly and to the right regardless of max QRSp.V direction.

The similarity in direction of max QRSp.V and max T unp.V supports the hypothesis by Rosenbaum et al that post-pacing T inversions may be, in part, explained on the basis of 'cardiac memory'. However the absence of correlation between the max QRSp.V and max T unp.V suggests that the post-pacing T inversions may be caused by other unknown mechanisms.

Additional Indexing Words:
Pacemaker Post-pacing T loop abnormalities

Massive T inversion in the unpaced electrocardiogram subsequent to ventricular pacing has been known to be referred to as post-pacing T
The precise pathogenesis and clinical significance of post-pacing T inversion are still obscure. It has been postulated by several authors that post-pacing T inversions are not caused by the artificial stimulus used for ventricular pacing, but by ventricular depolarization from an abnormal site. More recently, Rosenbaum and his colleagues noted that the polarities of T waves in unpaced beats were similar to those of QRS waves in paced beats. They suggested a new concept that the sequence of ventricular depolarization in the paced beat modulates the order of repolarization in the unpaced beat. However, additional quantitative studies of post-pacing T inversion are required, as Surawicz indicated in his editorial, “A mere change in T wave polarity in selected leads does not prove that the T wave ‘remembers’ the pattern of previous repolarization.”

In the present study, the T loops in unpaced vectorcardiograms and the QRS loops in paced vectorcardiograms were recorded from 18 patients with implanted cardiac pacemakers and compared. The results suggested that although the post-pacing T inversion might be explained, in part, by Rosenbaum’s theory, it could also be induced by other unknown causes.

**METHODS**

Vectorcardiograms (VCG) and electrocardiograms (ECG) of both paced and unpaced beats were recorded from 18 patients, 13 women and 5 men, ranging in age from 51 to 82 years; mean ± SD, 71.8 ± 9.8, with implanted permanent cardiac pacemakers. As shown by Tables I and II, the clinical diagnoses indicating pacemaker implantation were sick sinus syndrome in 13 cases, Mobitz II A-V block in 2 cases and complete A-V block in 3 cases. Sinus rhythm captured with ventricular complexes sometimes appeared preceding the pacemaker spike, and sometimes continued after the pacemakers were turned off.

All patients with III A-V block had been carefully weaned off the pacemaker and sinus rhythm captured with ventricular complexes could be recorded after stable supraventricular or idioventricular rhythm was re-established. The pacing periods ranged from 3 to 46 weeks; mean ± SD, 15.2 ± 12.7. Conventional 12-lead ECGs and VCGs were recorded in the supine position. VCGs of 15 to 20 cardiac cycles were stored on magnetic tapes. The unpaced ECGs showed sinus rhythm with normal intraventricular conduction in 13 patients, sinus rhythm with right bundle branch block (RBBB) in 3 patients and sinus rhythm with left bundle branch block (LBBB) in 2 patients.

The parameters used for analyses of the VCGs were: 1) the directions of
Table I. Patients with Normal Intraventricular Conduction

<table>
<thead>
<tr>
<th>case No.</th>
<th>age (yrs)</th>
<th>sex</th>
<th>diagnosis</th>
<th>underlying heart diseases</th>
<th>CTR (%)</th>
<th>pacing site</th>
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<td>LVH</td>
<td>64</td>
<td>endo</td>
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<td>79</td>
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<td>SSS</td>
<td>AS</td>
<td>57</td>
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<tr>
<td>3</td>
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<td>—</td>
<td>55</td>
<td>endo</td>
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<tr>
<td>4</td>
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<td>SSS</td>
<td>—</td>
<td>51</td>
<td>endo</td>
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<tr>
<td>5</td>
<td>79</td>
<td>female</td>
<td>Mobitz II A-V block</td>
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<td>endo</td>
</tr>
<tr>
<td>6</td>
<td>79</td>
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<td>—</td>
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<td>7</td>
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<td>—</td>
<td>54</td>
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<td>47</td>
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<td>SSS</td>
<td>—</td>
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<td>10</td>
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<td>SSS</td>
<td>—</td>
<td>55</td>
<td>endo</td>
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<tr>
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<td>71</td>
<td>female</td>
<td>SSS</td>
<td>MR, TR</td>
<td>57</td>
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</tr>
<tr>
<td>12</td>
<td>71</td>
<td>female</td>
<td>SSS</td>
<td>DCM</td>
<td>78</td>
<td>epi</td>
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<tr>
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<td>82</td>
<td>male</td>
<td>III A-V block</td>
<td>DCM</td>
<td>61</td>
<td>epi</td>
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SSS=sick sinus syndrome; LVH=left ventricular hypertrophy; AS=aortic stenosis; MR=mitral regurgitation; TR=tricuspid regurgitation; DCM=dilated cardiomyopathy; CTR=cardiothoracic ratio; endo=endocardial pacing; epi=myocardial pacing.

Table II. Patients with Abnormal Intraventricular Conduction

<table>
<thead>
<tr>
<th>case No.</th>
<th>age (yrs)</th>
<th>sex</th>
<th>diagnosis</th>
<th>underlying heart diseases</th>
<th>CTR (%)</th>
<th>pacing site</th>
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<td>58</td>
<td>endo RBBB</td>
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<td>LVH</td>
<td>64</td>
<td>endo RBBB</td>
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<tr>
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<td>59</td>
<td>male</td>
<td>Mobitz II A-V block</td>
<td>—</td>
<td>54</td>
<td>endo LBBB</td>
</tr>
<tr>
<td>18</td>
<td>66</td>
<td>female</td>
<td>SSS</td>
<td>—</td>
<td>50</td>
<td>endo LBBB</td>
</tr>
</tbody>
</table>

RBBB=right bundle branch block; LBBB=left bundle branch block; IVC during SR=intraventricular conduction during spontaneous rhythm. Other abbreviations same as for Table I.

The maximum QRS vector in the paced beats (max QRSp.V) and of the maximum T vector in the unpaced beats (max T unp.V) in the frontal and horizontal planes and 2) rotation of QRS loops in the paced beats and of T loops in the unpaced beats in all planes.

The patients were classified into 2 groups depending on whether the differences between the directions of max QRSp.V and max T unp.V in both frontal and horizontal planes were less than 30 degrees (group A) or more than 30 degrees (group B).

In addition, QRS duration and corrected QT interval (QTc) were measured from paced and unpaced ECGs. The ventricular gradient (G) vectors were calculated by Ashman’s methods (1 Ashman unit = 4 micro
volt sec) from both pre-paced and post-paced ECGs. The calculation of G vector in the horizontal plane was made from the QRS and T complexes in leads V₂ and V₆. The leads selected and their horizontal axes were determined according to Abildskov's report. Student's t-tests were used to calculate statistical significance and all values are reported as averages ±1SD.

Results

Normal intraventricular conduction

In 13 of 18 patients, the unpaced ECGs showed sinus rhythm with normal intraventricular conduction (Table I), 11 of the 13 patients were being paced from endocardial electrodes in the right ventricle and 2 from myocardial electrodes at the anterior right ventricle. The underlying heart diseases of 5 of the 13 patients were: dilated cardiomyopathy (DCM) in 2, left ventricular hypertrophy (LVH) in 1, aortic stenosis (AS) in 1, mitral regurgitation (MR) and tricuspid regurgitation (TR) in 1.

![Diagram](image)

Fig. 1. Examples of paced and unpaced vectorcardiograms and electrocardiograms of patients with normal intraventricular conduction. Lower panels show electrocardiograms. First QRS-T complexes show paced beat; second QRS-T complexes show unpaced beat. Upper panels show the vectorcardiograms from same patient. Top traces show QRS and T loops in paced beat. Lower traces show QRS and T loops in unpaced beat. Sides of QRS-T loops; amplified T loops shown.
Examples (case 7) of paced and unpaced VCGs and ECGs are shown in Fig. 1. In the ECGs (lower panel), the second QRS-T complexes show unpaced beats, in which the post-pacing T inversions are seen in all leads except aV_{R}, aV_{L} and V_{1}. The VCGs are shown in the upper panels. The top set of tracings shows QRS-T loops in the paced beat and the lower set shows QRS-T loops in the unpaced beat. The QRS loop of the paced VCG is directed superiorly, posteriorly and to the right.

The direction of the T loop of the unpaced VCG is quite similar to that of QRS loop of the paced VCG.

The directions of all measured max QRSp.V and max T unp.V are summarized in Fig. 2. In 11 cases paced from endocardial electrodes (solid lines), the max QRSp.Vs (crosses) were directed superiorly, posteriorly and to the right or left. The max T unp.Vs (circles) were superior in all cases, to the right in 9 and the left in 2, and posterior in 10. The mean directions of max QRSp.V and max T unp.V were respectively, 273.4±38.2 degrees, 247.3±30.8 degrees in the frontal plane, and 262.4±31.4 degrees, 242.6±44.0 degrees in the horizontal plane. Two cases (cases 12 and 13) were paced from myocardial electrodes. The max QRSp.V of case 13 was directed anteriorly, superiorly and to the left. The max T unp.V was also directed anteriorly and superiorly. The respective mean directions of max QRSp.V and max T unp.V were 306.0±8.5 degrees, 223.0±26.8 degrees in the frontal plane, and 171.0±199.4 degrees, 169.0±43.8 degrees in the horizontal plane. Differences between the directions of max QRSp.V and max T unp.V in the frontal and horizontal planes were calculated. All cases were classified into 2 groups in which the differences were either less than, or exceeded 30 degrees.

![Figure 2](image_url)

Fig. 2. Directions of max QRSp.V (crosses) and max T unp.V (open and closed circles) in frontal (left panel) and horizontal (right panel) planes in patients with normal intraventricular conduction. Crosses connected to open or closed circles in same patient by two kinds of lines. Solid lines show patients paced from endocardial electrodes; dotted lines show patients paced from myocardial electrodes. Open circles show patients in group A; closed circles show patients in group B.
in both planes. Of 11 cases paced from endocardial electrodes, 8 had less than 30 degrees difference between max QRSp.V and max T unp.V (open circles in Fig. 2; group A). The angles between these vectors exceeded 30 degrees in 3 of 11 cases paced from endocardial electrodes and the 2 cases paced from myocardial electrodes (closed circles in Fig. 2; group B).

In 8 of 11 cases paced from endocardial electrodes and 1 of 2 cases paced from myocardial electrodes, max T unp.V tended to be directed superiorly, posteriorly and to the right regardless the direction of max QRSp.V.

Agreement of the rotation of QRS loops in paced beats with that of T loops in unpaced beats was 27% in the frontal, 45% in the horizontal and 45% in the left saggital planes.

QRS durations and QTc, ST and T abnormalities in pre-paced ECGs were studied. QTcs in the unpaced beats were slightly greater in both groups than normal. There were no significant differences between groups in QRS duration or QTc in paced and unpaced beats. ST and T abnormalities in the pre-paced ECGs were similar in both groups, but in 2 of 5 group B cases, abnormally large T inversions were seen in the left precordial leads.

Changes in G vectors from pre-paced to post-paced ECGs are illustrated in Fig. 3 (n=8). In the 6 cases paced from endocardial electrodes, the changes in G vectors were directed superiorly, posteriorly and to the right in both group A (open circles) and group B (closed circles). In the 2 cases paced...
from myocardial electrodes, the changes in G vectors were directed superiorly, posteriorly (case 13) or anteriorly (case 12) and to the left.

**Abnormal intraventricular conduction**

Unpaced ECGs revealed 3 patients with sinus rhythm with RBBB and 2 patients with sinus rhythm with LBBB (Table II). All were being paced from endocardial electrodes. Paced and unpaced VCGs of case 17 who had abnormal intraventricular conduction (LBBB) are shown in Fig. 4. In the left panel, the upper tracings show paced VCG and lower tracings show unpaced VCG. The QRS loop in the paced beat was directed superiorly, posteriorly and to the left, while the T loop in the unpaced beat was directed superiorly, anteriorly and to the right. The direction of the T loop in the unpaced beat was similar to a previous description of LBBB.\(^{12}\)

The directions of max \(\text{QRSp.V}\) and max \(\text{T unp.V}\) in these cases are summarized in Fig. 5. In Fig. 5, crosses show the direction of max \(\text{QRSp.V}\). The direction of max \(\text{T unp.V}\) is indicated by triangles (RBBB) and squares (LBBB). In 3 RBBB cases in unpaced beats, the mean directions of max \(\text{QRSp.V}\) and max \(\text{T unp.V}\) were \(291.7\pm 50.6\) degrees and \(291.0\pm 20.1\) degrees in the frontal plane, and \(280.0\pm 66.6\) degrees and \(277.0\pm 64.2\) degrees in the horizontal plane, respectively. In 2 LBBB cases, mean directions of max \(\text{QRSp.V}\) and max \(\text{T unp.V}\) were \(203.7\pm 36.2\) degrees and \(317.3\pm 182.0\)
degrees in the frontal plane, and 170.0±632.2 degrees and 304.3±26.6 degrees in the horizontal plane, respectively. These results support the fact formerly reported that post-pacing T inversions were not seen in patients with LBBB, but did occur with RBBB in paced beats.4,5)

Changes in G vectors from pre-paced to post-paced ECGs in these cases are shown in Fig. 6. In Fig. 6, the change in G vectors was directed superiorly, posteriorly and to the right in 2 cases with RBBB. This direction was similar to that observed in cases with normal intraventricular conduction. In contrast, in 2 cases with LBBB the change in G vectors was directed inferiorly, posteriorly and to the left.
Massive T inversions occurred in unpaced ECGs subsequent to ventricular pacing and generally appeared in leads II, III, aVF and V_{2-5}. The characteristics of post-pacing T inversions are summarized in previous reports. The transient T inversion was independent of atrial rate and QRS duration. The magnitude of T inversion was related to the amount of power used for ventricular pacing as well as to the duration of the electrical stimulation, and the time required for complete recovery was longer than that of the electrical stimulation.

The electrocardiographic leads on which T wave inversions appear depend on the site of electrical stimulation; for example, in cases paced at the outflow tract of the right ventricle from the endocardial electrodes, T wave inversions appeared mainly in leads V_{1,2}, and during pacing of the left ventricle from the epicardial electrodes they appeared in leads I, aVL, V_{5-6}.

Although the genesis of post-pacing T inversion is still obscure, it has been postulated that such T wave changes might be the result of continuous abnormal depolarization and of electrothermic myocardial injury around the stimulating electrodes.

Recently, Rosenbaum and his associates systematically analyzed T wave changes in LBBB evoked by atrial pacing or by intermittent right ventricular pacing. They tried intermittent pacing in patients with rate dependent LBBB and noted that immediately after the cessation of atrial pacing the polarities of the T waves of unpaced beats conducted normally were the same as those of QRS waves of paced beats conducted abnormally (LBBB). From this fact, they concluded that the heart repolarization process can be modulated by the depolarization process.

In the present study, the directions of max QRSp.V and max T unp.V were compared. These parameters do not show the cardiac excitation sequence, but can indicate the direction of additional instantaneous vectors produced by larger excitation and repolarization wave fronts during a cardiac cycle. The directions of max QRSp.V and max T unp.V were similar in group A (68% of cases with normal intraventricular conduction) and in all cases with RBBB paced from endocardial electrodes. The above findings and the fact formerly reported, that post-pacing T inversion depends on the location of the stimulating electrodes, seem to partly support the possibility that the depolarization sequence of paced beats modulates the repolarization sequence. However, in group B and LBBB the directions of max QRSp.V differed from those of max T unp.V by more than 30 degrees. Rosenbaum interpreted this contradiction as indicating that the T wave results from
a balance of two different forces: one that causes a secondary T wave change and the other that causes the primary T wave change. If the secondary T wave change is predominant, the polarity of the post-pacing T waves will not be affected by the primary T wave change that is caused by modulation of the repolarization process. In this study, the underlying heart diseases were complicated in 1 of 8 cases of group A and in 4 of 5 cases of group B as shown in Tables I and II. These findings suggest that it might be difficult to modulate the repolarization process in group B by the depolarization process because of an abnormal myocardium.

Fig. 7 shows the vectorcardiograms of case 18. In this case, both normally and abnormally conducted beats (LBBB) appeared occasionally during pacing. The top traces show the paced vectorcardiograms. The middle and bottom traces show LBBB with normal intraventricular conduction in unpaced vectorcardiograms. The spatial direction of the T loop in the bottom is similar to that of the QRS loop in the top. However, in the middle traces, small round T loops are evident and the spatial direction of the T loop is similar to that of the secondary T loop that originally occurred in a subject with LBBB. This phenomenon indicates that the secondary force induced by abnormal intraventricular conduction in unpaced beats rather than the
primary force induced by the paced beats will predominate.

In cases paced from endocardial electrodes, the direction of max T unp.V and the change in G vectors from the pre- to the post-paced ECGs tended to be directed superiorly, posteriorly and to the right. These spatial directions may not depend on the directions of max QRSp.V in normal intraventricular conduction and RBBB with or without underlying heart diseases. These findings suggest that the post-pacing T inversions may be induced by additional unknown causes.

The following might be possible explanations. The post-pacing T inversions may be due to local tissue damage found around the stimulating electrodes. Similar transient T wave inversions were found in normal conducted beats in the ECGs of patients with intermittent LBBB, post-tachycardia syndrome, Wolff-Parkinson-White syndrome and extrasystole. Such T wave inversions appeared mainly in the chest leads \((V_3 - 6)\) and were often seen in vertical leads \((II, III, aVF)\). The spatial directions of such T wave inversions would be superiorly, posteriorly and to the right. In these cases, however, no local myocardial injury would be produced by the stimulating electrodes. The long-lasting effects of damage to the afferent or efferent cardiac nerves might be considered. Kralios and her associates noted that the polarity of the T wave in the vertical leads recorded on a canine’s body surface was altered by stimulating the cardiac branch of the sympathetic nerves. Unilateral alteration of sympathetic tone caused QT interval alterations. These experimental studies provided an anatomic basis for possible local control of the electrophysiological state and suggest that altered localized autonomic effects might affect the polarity of the T wave and QT interval. Their data support the latter possibility.

**REFERENCES**