**Introduction**

Conduction disturbances of the heart may be originated from the sinoatrial (SAN) and atrioventricular (AVN) nodes and intraventricular conduction system. The chronic conduction disturbances are due to degenerative conditions often without other identifiable myocardial disease (2). Some patients with permanent pacemakers have overt or latent atherosclerotic heart disease, which causes conduction disturbances, probably by inducing conduction system ischemia (3).

We aimed to evaluate the importance of coronary artery disease being as a cause of conduction disturbances, which necessitate permanent cardiac pacemaker implantation.

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**Objective:** In the present study we examined retrospectively the coronary anatomy/pathology of 78 consecutive patients with coronary artery disease (CAD) who underwent permanent pacemaker implantation in order to find a common pathological anatomic basis for conduction disturbances and to compare them with a group of matched patients with angiographically proven CAD.

**Methods:** Study group consists of seventy-eight patients with angiographically documented CAD and permanent pacemaker implantation. Control group included comparable patients with CAD and without a pacemaker implantation. Coronary angiography was performed using standard Judkins approach in all patients within 2 months before pacemaker implantation. The locations of narrowings in the left anterior descending (LAD) and right (RCA) coronary arteries, as the arteries supplying the conduction system, were documented accurately and further classified as follows. Type I: Anatomy not compromising blood supply to the conduction system, namely, either the absence of significant narrowing in the LAD, RCA, left circumflex, posterolateral, or posterior descending arteries or the presence of mid-distal LAD lesions beyond the septal branches. Type II: Pathological coronary anatomy involving septal branches emerging from the LAD (and without significant lesions in the RCA). Type III: Pathological coronary anatomy compromising blood supply to the sinoatrial (SAN) or atrioventricular (AVN) nodes but not compromising blood flow to the septal branches. This subset included patients with distal LAD lesions after the septal branches. Type IV: Combination of types II and III pathological coronary anatomy that compromises blood supply both to the septal branches and SAN and AVN arteries.

**Results:** Occurrence of the type IV coronary anatomy (45%) was significantly higher than type I (19%), type II (24%) and type III (11%) in the study group (p<0.02). Statistically significant differences were found between the two groups (p<0.05): more patients in the study group had type II (24%) and IV (45%) coronary anatomy (p<0.02) while type I (35%) and III (37%) anatomy were more frequently observed in control group (p<0.05). Analysis of flow quality of septal perforators, SAN and AVN arteries, in the study group demonstrated a significant tendency for reduced blood flow in the conduction system.

**Conclusion:** Presence of first perforator lesions with poor quality of flow and right coronary artery lesions shown angiographically should be considered as the risk factors requiring permanent pacemaker implantation in patients with coronary artery disease.

**Key words:** Permanent pacemaker, coronary artery disease, pathological coronary anatomy (Anadolu Kardiyol Derg, 2002, 4:279-83.)
Material and Methods

Study Population:

We studied 78 patients who had pacemaker implantation and angiographically proven coronary artery disease between 1997 and 1999. None of these patients received beta-blockers, calcium channel blockers, amiodarone or other medications responsible for sinus node dysfunction or conduction disturbances. To each patient from the study group (group I), we matched a patient without a pacemaker using the following criteria: sex, age patient, presence or absence of diabetes mellitus, hypertension, and absence or presence of significant (>50%) narrowing in the coronary arteries responsible for supplying blood to the conduction system (Left anterior descending: LAD, right coronary artery: RCA, Left circumflex: LCx). For matching, we used the presence or absence of lesions in each coronary artery but without regard to their locations in specific segments. These cases comprised the control group (group II). Patients who had coronary artery by-pass surgery before pacemaker implantation, dilated cardiomyopathy, valvular heart disease, heart transplantation were not included in this study. Indications for pacemaker implantations were atrioventricular block in 18 patients, atrial fibrillation with low ventricular rate in 16 patients and sick sinus syndrome in 44 patients.

Coronary Angiography:

Coronary angiography was performed with the standard Judkins approach in all patients within 2 months before pacemaker implantation. Angiograms were reviewed by two experienced observers who were unaware of clinical data of patients. Significant coronary artery disease was defined as narrowing of > 50% of the coronary artery luminal diameter. The dominance of the coronary tree was determined and documented according to the criteria of Dodge et al (4). Patient who had dominant LCx coronary artery were not included in the study.

Assessment of Pathological Coronary Anatomy Lesions:

Narrowings in the coronary artery tree were identified in the following arteries and branches: left main, first three perforators, first diagonal, LCx, first marginal, RCA, right ventricular branch, posterolateral (PL), posterior descending coronary artery (PDA), SAN and AVN arteries. Each lesion diameter was compared with an adjacent distal normal-looking segment and lesion severity was graded in the following manner: 0 % to 50 %, insignificant; 50 % to 70 % moderate; 70 % to 90 %, significant; and > 90 %, severe. Left main coronary artery, LAD and LCx were measured in the antero-posterior view; perforators, ramus medianus, and first marginal in the right anterior oblique (RAO) view; first diagonal in the RAO or caudo-cranial left anterior oblique (LAO) view; and RCA, right ventricular branch, PL, PDA, SAN and AVN arteries in the LAO view.

Qualitative Assessment of antegrade and retrograde flows:

Antegrade and retrograde flows in branches supplying the conduction system were graded qualitatively in each patient as good, moderate and poor (5).

Classification of Pathological Coronary Anatomy Supplying the Conduction System:

The location of narrowings in the LAD and RCA as the arteries supplying the conduction system was documented accurately and classified as follows (5).

Type I: Anatomy not compromising blood supply to the conduction system, namely, either the absence of significant narrowing in the LAD, RCA, LCx, PL, or PDA or the presence of mid-distal LAD lesions beyond the septal branches.

Type II: Pathological coronary anatomy involving septal branches emerging from the LAD (and without significant lesions in the RCA).

Type III: Pathological coronary anatomy compromising blood supply to the SAN or AVN but not compromising blood flow to the septal branches. This subset included patients with distal LAD lesions after the septal branches.

Type IV: Combination of types II and III: pathological coronary anatomy that compromises blood supply both to the septal branches and SAN and AV arteries.

Statistics

Comparison of clinical data was performed by use of unpaired-t test. Distribution of study patients
and their matched control cases according to stenosis severity or pathological anatomy types was analyzed with the Chi-square test. Distribution of the study patients and their matched control cases according to the direction (antegrade or retrograde) and quality of blood supply to the conduction system was studied with the Chi-square test.

Results

The study group comprised 78 patients who had permanent pacemaker and coronary artery disease shown by angiography. We matched 78 patients without pacemaker from our angiography database to each 78 patients from the study group. The clinical data of the two groups were comparable (Table 1). There were no significant differences between groups in respect to single vessel, two vessel, and three vessel disease. All the matched patients had dominant right coronary artery. Study group and matched control group did not differ by the distribution of the patients according to lesion severity (Table 2).

The distribution of pathological anatomic types in regard to the conduction system blood supply in studied groups is presented in Table 3. Type IV coronary anatomy (45 %) was significantly higher than type I (19 %), type II (24 %) and type III (11 %) in the study group than in control one (p<0.02). A statistically significant difference was found between the two groups (p<0.05): more patients had type II (24 %) and IV (45 %) coronary anatomy in the study group (p<0.02), and type I (35 %) and III (37 %) anatomy in control group (p<0.05). If one combines the anatomic types that compromise blood flow to septal branches (type II and IV) and the anatomic types that do not (type I and III) in the study group, there could be found a statistically significant difference in distribution of patients between these combinations (54 patients: 70 % vs 24 patients: 30 %, p<0.005, respectively). In the analysis of flow quality of septal perforators, SAN and AVN arteries; it was found that there is a significant tendency for reduced blood flow in the conduction system of the study group (Table IV). The number of patients who had poor flow quality in the septal branches, SAN and AVN arteries was significantly higher in the study group than in control group (p<0.05).

Discussion

Blood supply to the conduction system as well as the pathologic anatomy of the coronary circulation in the presence of intraventricular conduction disturbances has already been reported (6-12). There is still controversy about the association of con-
duction disturbances and underlying coronary anatomy. The relationship between the conduction disturbances and the underlying pathology has been studied at autopsy, but these patients do not represent the general population. Hambly et al. (13) examined 42 patients with ECG conduction disturbances and symptomatic coronary artery disease. Although most patients had a significant lesion in the LAD coronary artery, there was no correlation with any specific lesion. Several anatomical data of the coronary artery have been investigated and related to post-CABG conduction disturbances: the number of diseased coronary arteries was not found to be predictive (14,17). It has been reported that total occlusion of the LAD or right coronary artery or occlusion of both arteries were unrelated to conduction disturbances (14,17). Mosseri et al. has reported that conduction disturbances after CABG are related to specific anatomical lesions involving the first perforating artery which supplies the interventricular septum (5,16).

We hypothesized that an atherosclerotic process in arteries that supply region of conduction may cause disturbances that require pacemaker implantation. We have several main findings; first, the occurrence of type IV coronary anatomy was significantly higher than the other types in the study group and significantly higher than those of control group. Second, incidence of combined anatomic types that compromise blood flow to the septal branches (type II and IV) is significantly higher than the anatomic types that do not (type I and III). Third, the flow quality of the first septal perforator, SAN artery and AVN artery in the study group was significantly poorer than in control group.

Main difference of our study than the previous ones is that we studied only patients who had coronary artery disease proven angiographically. Patients who had CABG, valvular heart disease and heart transplantation were not included in the study. According to our knowledge, our study is the first which evaluates patients who had only CAD without CABG and valvular heart disease. Mosseri et al (16) has reported that conduction disturbances after CABG are found to be related to specific anatomical lesions involving the first perforating artery which supplies the interventricular septum. Furthermore we can suggest that patients who have specific anatomical lesions involving first perforator artery and RCA should be considered as candidates to permanent pacemaker implantation regardless of previous CABG.

Conclusion

Conduction disturbances requiring permanent pacemaker implantation were found to be related to flow quality and specific anatomical lesions involving the first perforator artery and the right coronary artery in patients with coronary artery disease. Patients with CAD and conduction disturbances requiring implantation of permanent pacemakers are more likely to have a poor quality flow in the septal branches of left anterior descending coronary artery and right coronary artery lesions. Presence of first perforator lesions with poor quality of flow and right coronary artery lesions shown angiographically should be considered as the risk factors requiring permanent pacemaker implantation in patients with coronary artery disease.

Table-III: Distribution of coronary anatomy type in the study group and matched control group

<table>
<thead>
<tr>
<th>Type</th>
<th>Study Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>15 (19 %)</td>
<td>27 (35 %) y</td>
</tr>
<tr>
<td>Type II</td>
<td>19 (24 %) t</td>
<td>7 (9 %)</td>
</tr>
<tr>
<td>Type III</td>
<td>9 (11 %)</td>
<td>29 (37 %) y</td>
</tr>
<tr>
<td>Type IV</td>
<td>35 (45 %)*</td>
<td>15 (19 %)</td>
</tr>
</tbody>
</table>

* p<0.05 vs type I, II and III, † p<0.02 vs control group, ‡ p<0.05 vs study group

Table-IV: Flow quality in the study group and control group in septal branches, SAN and AVN arteries

<table>
<thead>
<tr>
<th>Flow Quality</th>
<th>Study group</th>
<th>Control group</th>
</tr>
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<tbody>
<tr>
<td>Poor</td>
<td>34 (44%)*</td>
<td>18 (23%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>26 (33%)</td>
<td>34 (44%)</td>
</tr>
<tr>
<td>Good</td>
<td>18 (23%)</td>
<td>26 (33%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>78</td>
<td>78</td>
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<table>
<thead>
<tr>
<th>Flow Quality</th>
<th>Study group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor</td>
<td>28 (36%) y</td>
<td>16 (20%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>30 (38%)</td>
<td>41 (53%)</td>
</tr>
<tr>
<td>Good</td>
<td>20 (26%)</td>
<td>21 (27%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>78</td>
<td>78</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Flow Quality</th>
<th>Study group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor</td>
<td>41 (53%) y</td>
<td>15 (19%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>15 (19%)</td>
<td>16 (20%)</td>
</tr>
<tr>
<td>Good</td>
<td>22 (28%)</td>
<td>38 (49%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>78</td>
<td>78</td>
</tr>
</tbody>
</table>

* vs control group p<0.02, † vs control group p<0.05, ‡ vs control group p<0.005
References