Dispersion of P wave duration and P wave vector in patients with atrial septal aneurysm

Marianna Janion*, Jacek Kurzawski, Janusz Sielski, Katarzyna Ciuraszkiewicz, Marcin Sadowski, and Edyta Radomska

Swietokrzyskie Centrum Kardiologii WSzZ, ul. Grunwaldzka 45, 25-736 Kielce, Poland

Received 13 November 2006; accepted after revision 10 April 2007; online publish-ahead-of-print 30 May 2007

Aims Atrial septal aneurysm (ASA) may be involved in the genesis of atrial arrhythmias as a consequence of disturbances in the propagation of depolarization, which may be easily assessed by P wave dispersion measurement. The aim of this study is to assess the dispersion of P wave duration and P wave vector in patients with ASA and to determine the effect of associated interatrial shunt on the magnitude of P wave dispersion.

Methods and results The study population consisted of 23 healthy volunteers and 88 patients with ASA base more than 15 mm and protrusion more than 7.5 mm. The size of aneurysms and atria was determined by echocardiography and P wave dispersion was measured on the surface ECG. In ASA patients, dispersion of P wave duration was significantly increased when compared with healthy controls (7.8 ± 12.1 vs. 3.7 ± 3.5 ms; P < 0.01). Dispersion of P wave vector was also significantly increased (8.5 ± 10.1° vs. 4.6 ± 3.6°; P < 0.005). In healthy volunteers, the mean values of both parameters were below the cutoff points.

Conclusion In patients with ASA, there was a significant dispersion of P wave duration and P wave vector. Variation in P wave duration was significantly correlated with the dispersion of P wave vector and age of these patients. Dispersion of P wave vector was significantly decreased in ASA patients with interatrial shunt. P wave dispersion in ASA patients may predispose to the development of atrial arrhythmias.

KEYWORDS Atrial septal aneurysm; P wave dispersion; P wave duration; P wave vector; Interatrial shunt

Introduction

The first autopsy of a case of atrial septal aneurysm (ASA) was reported over 80 years ago in 1934.1 The introduction of diagnostic ultrasound resulted in a marked increase in the number of patients identified with ASA with the detection rate of 0.2–4.0% by transthoracic echocardiography (TTE) and of 2.0–8.0% by omniplane transesophageal echocardiography (O-TEE).2–5 Atrial septal aneurysm is a risk factor for arterial embolism, as frequently co-existing patent foramen ovale (PFO) or atrial septal defect (ASD) provides an interatrial shunt leading to paradoxical embolism.2–6 The presence of an additional structure in the interatrial septum may be involved in the genesis of atrial arrhythmias as a consequence of disturbances in the propagation of depolarization, which may be easily assessed by P wave dispersion measurement. To the best of our knowledge, there is no analysis of these parameters in patients with ASA.

The aim of this study is to evaluate differences in dispersion of P wave duration and P wave vector in patients with ASA and to determine the effect of an associated interatrial shunt on the magnitude of P wave dispersion.

Methods

Patients

The study population consisted of 88 patients—35 (39.8%) men and 53 (60.2%) women—and 23 healthy volunteers who served as the control group. Subjects were selected from 21 588 TTE examinations performed in an echocardiographic laboratory, which serves as a reference centre in the district of over 1 300 000 inhabitants. The mean age of all study patients was 54.3 ± 14.4 and the mean age of the control subjects was 47.5 ± 11.8 years (P < 0.05).

Entry criteria included the presence of ASA by echocardiography and sinus rhythm on the surface ECG. Subjects were excluded if they had a history of transient ischemic attack or stroke, atrial fibrillation, or any other ischemic heart disease. All patients had normal serum ion levels and no other metabolic disorders.

Study protocol

Atrial septal aneurysm was detected by transthoracic (TTE) or transesophageal echocardiography (O-TEE) in the longitudinal and transverse projections, providing clear visualization of the interatrial septum. Patients who had an aneurysm with the base >15 mm and protrusion >7.5 mm (half of the minimal base dimension)
Table 1  Echocardiographic and electrocardiographic parameters in patients with atrial septal aneurysm and in healthy controls

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Study patients (n = 88)</th>
<th>Healthy controls (n = 23)</th>
<th>P &lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size of ASA (cm²)</td>
<td>1.52 ± 0.72</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Left atrial length (M-mode) (cm)</td>
<td>3.93 ± 0.77</td>
<td>3.76 ± 0.41</td>
<td>ns</td>
</tr>
<tr>
<td>Left atrial cavity area (cm²)</td>
<td>23.75 ± 7.90</td>
<td>18.85 ± 3.30</td>
<td>0.005</td>
</tr>
<tr>
<td>Right atrial cavity area (cm²)</td>
<td>19.28 ± 7.38</td>
<td>17.30 ± 2.41</td>
<td>0.05</td>
</tr>
<tr>
<td>Mean P wave duration</td>
<td>114.4 ± 14.3</td>
<td>118.6 ± 9.8</td>
<td>ns</td>
</tr>
<tr>
<td>Dispersion of P wave duration</td>
<td>7.8 ± 12.1</td>
<td>3.7 ± 3.5</td>
<td>0.01</td>
</tr>
<tr>
<td>(tP1–tP2) (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean vector deviation (degree)</td>
<td>54.5 ± 21.0</td>
<td>52.7 ± 20.6</td>
<td>ns</td>
</tr>
<tr>
<td>Dispersion of P wave vector</td>
<td>8.5 ± 10.1</td>
<td>4.6 ± 3.6</td>
<td>0.005</td>
</tr>
<tr>
<td>(Vp–Vp) (degree)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1  Relationship between dispersion of P wave duration and dispersion of P wave vector in the study group (P = 0.003).

Table 1 summarizes the results. Dispersion of P wave duration was significantly increased in patients with ASA when compared with healthy controls (7.8 ± 12.1 vs. 3.7 ± 3.5; P < 0.01). Dispersion of P wave vector was also significantly increased in patients with ASA when compared with healthy controls (8.5 ± 10.1° vs. 4.6 ± 3.6°; P < 0.005). There was no dispersion of P wave duration and P wave vector in the control group.

In the study group, there was a significant correlation between the dispersion of P wave duration and that of the P wave vector: (tP1–tP2) = 0.167(Vp1–Vp2) + 4.704; P < 0.003 (Figure 1) and a positive relationship with patient age (tP1–tP2) = 0.098x + 0.448; P < 0.005 (Figure 2). Cavity area of both atria was also significantly larger in patients with ASA than in healthy controls; however, there was no significant relationship between vector dispersion and left and right atrial size and cavity area.

Of 88 patients with ASA, echocardiography detected interatrial shunting in 47 (53.4%) patients. Of the 47 patients, 28 (57%) subjects had PFO and 21 (43%) ASD. There was no correlation between aneurysm parameters or atrial size and the frequency of PFO or ASD. Dispersion values in patients with and without interatrial shunt are given in Table 2. Dispersion of P wave vector (Vp1–Vp2) in patients without interatrial shunt is significantly higher than that in patients with the shunt.

Twenty-four hour ECG monitoring detected paroxysms of atrial fibrillation and/or flutter in 15 (17.0%) patients with ASA. There was a significant relationship between the

Statistical analysis

Data were expressed as mean ± standard deviation. Student’s t-test was performed to estimate the statistical significance of the results. A P-value less than 0.05 was considered statistically significant.

Ethics

The study complies with the Declaration of Helsinki. The research protocol was approved by the local Ethics Committee. The informed consents of the subjects were obtained.
presence of atrial arrhythmias and the dispersion of P wave duration ($P < 0.005$).

### Discussion

Electrocardiographic recording of sinus P wave provides information on its duration, amplitude, dispersion, and direction of electrical vector. Depending on atrial area, dispersion of P wave duration varies from 0 to 80 ms, whereas dispersion of P wave vector varies from $-50$ to $+60^\circ$. Patients with ASA may be subject to additional risk related to variation in these parameters, because of disturbances in the propagation of depolarization due to the presence of pathological structure.

P wave dispersion has been extensively studied in various disease states. Left ventricular inflow obstruction due to structural or functional changes in the left ventricle may cause P wave dispersion. Pathophysiologic abnormalities in the atria, such as post-ablation injury, give rise to differences in P wave duration. Dispersion has also been found in patients with psychotic disorders such as anxiety and prior to haemodialysis. However, all those patients did not have ASA. The purpose of this study was to estimate dispersion of P wave vector in patients with ASA. Dispersion of P wave duration and P wave vector was significantly increased in the study patients when compared with healthy controls, confirming the suggestion that ASA may be a source of such variation. Furthermore, the presence and magnitude of P wave dispersion were independent of aneurysm and atrial size.

In patients with ASD, dispersion was diagnosed prior to surgical operation and decreased after defect closure. In the present study, P wave dispersion in patients with ASA and associated interatrial shunt was smaller than that in subjects without shunting, and the wall of the aneurysm was markedly deformed because of pressure gradient between the atria. Evidence shows that closure of the defect is not sufficient to eliminate dispersion; it is also necessary to add rigidity to atrial septal structures to eliminate septal pulsation.

Several investigators demonstrated the relationship between dispersion and paroxysmal atrial fibrillation. Dispersion was also found to predict recurrent atrial fibrillation or flutter in patients after cardioversion. In the present study, 17% of patients with ASA had atrial fibrillation/flutter on the ECG monitoring, which is consistent with the results of studies in patients with paroxysmal atrial fibrillation.

There is controversy regarding the impact of atrial cavity size on the magnitude of P wave dispersion. In one study, there was a correlation between atrial cavity size and the magnitude of P wave dispersion, but other investigators did not demonstrate such relationship. The present study corroborates the latter finding.

### Study limitation

The subjects in the control group were significantly younger than those in the study group. Although this was unintentional, only those agreed to serve as controls.

### Conclusions

In patients with ASA, there was a significant dispersion of P wave duration and P wave vector. The differences were independent of the size of the aneurysm. Variation in P wave dispersion was significantly correlated with dispersion of P wave vector and age of these patients. In patients with ASA and interatrial shunt, dispersion of P wave vector was significantly decreased when compared with patients without the shunt. P wave dispersion in ASA patients may predispose to the development of atrial arrhythmias such as atrial fibrillation or flutter.

### Conflict of interest

none declared.

### References


### Table 2

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Interatrial shunt present (n = 47)</th>
<th>Interatrial shunt absent (n = 41)</th>
<th>P &lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dispersion of P wave duration</td>
<td>7.3 ± 11.1</td>
<td>8.4 ± 12.9</td>
<td>ns</td>
</tr>
<tr>
<td>($T_{P1} - T_{P2}$) (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dispersion of P wave vector</td>
<td>4.7 ± 4.1</td>
<td>12.3 ± 12.1</td>
<td>0.001</td>
</tr>
<tr>
<td>($V_{P1} - V_{P2}$) (degree)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Conflicts of Interest:** none declared.