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ORIGINAL RESEARCH

The Effects of Surgical Repair on P-Wave Dispersion in Children with Secundum Atrial Septal Defect

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ABSTRACT

Introduction: Atrial septal defect (ASD) is one of the most common congenital heart diseases in children. P-wave dispersion has been reported to be associated with non-homogeneous propagation of sinus impulses. The heterogeneity of atrial conduction time may predispose the atria to arrhythmias. The aim of this study was to determine the impact of surgical repair on P-wave indices in children with isolated secundum ASD.
Methods: Children with isolated secundum ASD undergoing surgical repair (n=50; mean age, 7.0±3.0 years) and healthy controls (n=51; mean age, 7.6±2.7 years) were compared. Maximum P-wave duration (Pmax), shortest duration (Pmin) and P-wave dispersion (Pd) were measured using 12-lead surface electrocardiography.

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Results: Mean Pmax was found to be significantly higher in children with ASD compared with controls (95.2±10.8 vs 84.1±9.2 msec; $P<0.001$), and Pd before surgery was significantly higher compared with controls (47.4±12.0 vs 38.8±9.7 msec; $P<0.001$). Both P-wave indices were significantly decreased within the first year after surgical closure – the values decreased to those comparable to healthy controls (Pmax, 86.2±9.7 msec; Pd, 39.8±10.7 msec; $P>0.05$).
Conclusion: Surgical closure of ASD in children decreases Pmax and P-wave conduction time. We speculate that earlier closure of the defect may play an important role in avoiding permanent changes in the atrial myocardium and atrial fibrillation in adulthood.

Keywords: atrial septal defect; children; electrocardiography; P-wave dispersion; surgery

INTRODUCTION

Atrial septal defect (ASD) is one of the most common congenital heart diseases in children.[1] Electrophysiological studies in children with secundum ASD demonstrate that sinus node dysfunction begins in early childhood.[2] Atrial arrhythmias, in particular atrial fibrillation, are responsible for the significant morbidity and mortality related to ASD in adults even after surgical repair.[3]

The prolongation of intra- and interatrial conduction time and the non-homogeneous propagation of sinus impulses are well-known electrophysiological characteristics of the atria prone to fibrillation.[4] They have been evaluated using maximum P-wave duration (Pmax) and P-wave dispersion (Pd).[5] Pd is defined as the difference between the longest and the shortest P-wave duration recorded from 12-lead surface electrocardiogram (ECG) leads. Pmax and Pd, measured by ECG, are indicators of interatrial conduction disorder, and non-homogeneous atrial conduction, respectively.[5] Previous studies have shown that Pmax and Pd can predict atrial fibrillation in patients undergoing haemodialysis, aorta-coronary bypass grafting, hypertrophic cardiomyopathy, and in subjects without underlying heart disease.[5–9]

Increases in P-wave duration and Pd have been used as predictors of atrial fibrillation development in various clinical settings.[6–9] Pd has been examined in children with isolated secundum ASD and was found to be significantly higher compared with age-matched controls.[10] In a recent study, Guray et al.[11] demonstrated that Pmax and Pd were significantly higher in adults with secundum ASD compared with healthy controls. Surgical repair significantly improved these indices in patients with secundum ASD. However, to the authors' knowledge, no study has documented P-wave alterations after surgical

closure in children with ASD. The aim of this study was to determine the impact of surgical repair on P-wave indices in children with isolated secundum ASD.

MATERIALS AND METHODS

The study used 50 patients (29 girls, 21 boys; mean age, 7.0±3.0 years) with isolated secundum ASD who underwent surgical repair (via a hatch). Patients were recruited from Istanbul University Paediatric Cardiology Outpatient department between 1998 and 2007. Fifty-one healthy children served as controls, none of which had a history of ASD of any kind (28 girls, 23 boys; mean age, 7.6±2.7 years). The study protocol was approved by the medical ethics committee of Istanbul University. Written informed consent was obtained from all participants.

All of the patients and controls displayed a normal sinus rhythm. Patients were excluded if they demonstrated any of the following: a history of arrhythmias, intake of anti-arrhythmic drugs, valvular heart disease, presence of cardiomyopathies, pulmonary hypertension, or any neurological disease. Patients with other congenital cardiac defects associated with ASD were also excluded. Adverse events (including arrhythmias) were recorded throughout the study. Echocardiographic examination was performed for all control subjects and patients before surgery using a commercially available system (Vivid-3 Expert, GE, USA). Left ventricular end-diastolic diameter was measured from the parasternal long-axis window in M-mode echocardiography.[12] The ejection fraction of the left ventricle was obtained using modified Simpson's method.[13]

Standard 12-lead ECGs were obtained simultaneously using a recorder (Nihon Kohden cardiofax ECG-6511, Japan) set at 50 mm/sec paper speed and 1 mV/cm in a comfortable supine position. For ASD patients, this was recorded before, and 6–12 months after surgery; for controls, this was recorded once at a similar date to baseline recordings. Measurements were calculated using a calliper and a magnifying lens. The P-wave was defined as the distance from the point of the first visible upward or downward departure from the baseline to the return to the baseline. The longest atrial conduction time measured in any of the 12 leads was defined as Pmax; the shortest time was defined as Pmin. The difference between Pmax and Pmin was calculated and defined as Pd (Pd=Pmax–Pmin).

Student *t* tests were used to compare the parametric values between ASD patients (at baseline and after surgery) and controls. For the comparison of the values at baseline and after surgery, paired *t* tests were used. Unless otherwise stated, data are presented as the mean±standard deviation (SD). A P value of <0.05 was considered statistically significant.

RESULTS

The mean age of the ASD patients was not significantly different compared to the healthy subjects (P>0.05). There were no significant differences with regard to left ventricle diastolic diameter (41.9±4.2 vs 40.9±5.5 mm; P>0.05) and left ventricular ejection fraction (67.0%±3.9%

Table 1. P-wave measurements of both controls and children with secundum atrial septal defect. Atrial conduction time was measured using 12-lead surface electrocardiograms.

		Patients (n=50)		Statistical comparison		
Variables	Controls (n=51)	Baseline	After surgery	$P1$	$P2$	$P3$
Pmin, msec	45.3±5.1	47.7±7.2	46.3±6.9	NS	NS	NS
Pmax, msec	84.1±9.2	95.2±10.8	86.2±9.7	<0.001	<0.001	NS
Pd, msec	38.8±9.7	47.4±12.0	39.8±10.7	<0.001	<0.001	NS

Data are presented as mean±standard deviation. Statistical comparison: $P1$=controls vs baseline; $P2$=baseline vs after surgery; $P3$=controls vs after surgery.
NS=not significant; Pd=Pmax–Pmin; Pmax=the longest atrial conduction time; Pmin=the shortest time.

vs 65.9%±4.2%; P>0.05). Pmin was not significantly different between patients and controls (47.7±7.2 vs 45.3±5.1 msec; P>0.05). None of the patients documented arrhythmias during the postoperative follow-up, or any other clinically significant adverse events.

ASD diameters were all larger than 7 mm (mean, 14.8±5.1 mm; range, 8–40 mm) in the patient group. Pmax was significantly longer in children with ASD compared with the controls (P<0.001). Children with ASD also had significantly higher Pd levels compared with controls (P<0.001). In order to investigate the effect of surgical repair on Pd, the patients' baseline and postoperative ECG values were compared. Table 1 shows the baseline and postoperative P-wave duration measurements of children with ASD and controls. Data indicate that both Pmax and Pd significantly decreased within 1 year after surgery when compared with baseline values (P<0.001). After surgery, both Pmax and Pd were not significantly different compared to values in the control group (P>0.05).

DISCUSSION

The results of this study show that Pd was significantly higher in ASD patients at baseline compared with controls. Furthermore, ASD patients' Pd was significantly decreased after surgery. To the authors' knowledge, this is the only study to evaluate the impact of surgery on Pd among children with ASD.

Atrial arrhythmias – especially atrial fibrillations – are frequently encountered in adult patients with ASD and are responsible for substantial morbidity and mortality even after surgical closure.[3,14,15] In patients with isolated ASD, a chronic left-to-right shunt imposes a volume overload on the right-sided cardiac structures and results in their dilation.[16] Dilation of the atrium with age may partially explain why atrial arrhythmias are more common in adult patients.

Pmax is used as an indicator of interatrial conduction disorder, while Pd is used as a marker of regional differences in P-wave durations.[5] The degree of atrial dilation and the size of the ASD have a

positive relation to the changes in atrial conduction time.[10] The heterogeneity of atrial conduction time may predispose the atria to arrhythmias.

In our study, we found that Pmax and Pd were significantly higher in children with isolated secundum ASD compared with controls; we also found significant decreases in both P-wave indices after surgical repair. The results of this study give further support to Ho and colleagues,[10] who demonstrated that Pd was higher in children with ASD. The authors also reported similar Pmax values between ASD patients and controls.[10] In an adult population, Guray et al. found that Pmax was higher in patients with ASD.[11] Changes in atrial myocardium may become more overt with age due to the continual pressure and potential volume overload.[3,15,17] Therefore, the prolonged Pmax values, in contrast to the study by Ho et al., may be attributed to the older age of ASD patients in our study (7.0±3.0 vs 2.9±4.1 years).

Guray et al. studied adult ASD patients with no history of paroxysmal atrial fibrillation (PAF).[11] After surgical closure, patients that did show postoperative PAF tended to be older, with larger Pmax (and higher Pd) at baseline compared with those without postoperative PAF.[11] In the postoperative PAF group, Pmax and Pd values did not decrease significantly within the first year of follow-up after surgery, but they did in those without postoperative PAF.[11] In our study, as in the adult population, we observed a similar decrease in P-wave duration in children after surgery.

The incidence of atrial fibrillation tends to increase with the patient's age.[15] It may therefore be related to the established mechanical and electrophysiological changes of the atrial myocardium at older age.[11] Surgical closure rarely eliminates chronic atrial fibrillation in adult patients with ASD.[14] One of the reasons for recommending early surgical closure of ASD is to prevent atrial arrhythmias arising when the patients become older.[3,14,15] None of the children in our study group had a history of atrial arrhythmias. We believe that the effect of surgery on the alterations of the atrial conduction time in children with ASD may reduce the occurrence of atrial arrhythmias when they become older. This hypothesis has yet to be proven.

One of the limitations of our study was the relatively small number of patients. A second limitation was the calculation of P-wave measurements by manual method, as opposed to the (potentially more accurate) computer-assisted method.

This study revealed that children with ASD have significantly higher Pmax and Pd in comparison to controls and have a significant decrease (down to the level of normal control values) after surgery. To date, only a limited number of studies have been carried out among children and adults with ASD regarding pre-operative ECG changes and the effect of surgery on P-wave conduction time alterations. Therefore, we suggest that further studies are needed on these populations using P-wave indices for the prediction of future atrial fibrillation episodes, and to define the optimal time for surgery – before permanent changes in the atrial myocardium occur.

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