# Prevalence of Interatrial Block in Healthy School-Aged Children: Definition by P-Wave Duration or Morphological Analysis

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**Background:** P waves ≥110 ms in adults and ≥90 ms in children are considered abnormal, signifying interatrial block, particularly in the first case.

**Methods:** To evaluate the prevalence of interatrial block in healthy school-aged children, we obtained 12-lead digital ECGs (Cardioperfect 1.1, CardioControl NV, Delft, The Netherlands) of 664 healthy children (349 males/315 females, age range 6–14 years old). P-wave analysis indices [mean, maximum and minimum (in the 12 leads) P-wave duration, P-wave dispersion, P-wave morphology in the derived orthogonal (X, Y, Z) leads, as well the amplitude of the maximum spatial P-wave vector] were calculated in all study participants.

**Results:** P-wave descriptor values were: mean P-wave duration 84.9 ± 9.5 ms, maximum P-wave duration 99.0 ± 9.8 ms, P dispersion 32.2 ± 12.5 ms, spatial P amplitude 182.7 ± 69.0 $\mu$V. P-wave morphology distribution in the orthogonal leads were: Type I 478 (72.0%), Type II 178 (26.8%), Type III 1 (0.2%), indeterminate 7 (1%). Maximum P-wave duration was positively correlated to age ($P < 0.001$) and did not differ between sexes ($P = 0.339$). Using the 90-ms value as cutoff for P-wave duration, 502 (75.6%) children would be classified as having maximum P-wave duration above reference range. The 95th and the 99th percentiles were in the overall population 117 ms and 125 ms, respectively. P-wave morphology type was not in any way correlated to P-wave duration ($P = 0.715$).

**Conclusions:** Abnormal P-wave morphology signifying the presence of interatrial block is very rare in a healthy pediatric population, while widened P waves are quite common, although currently classified as abnormal.

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P-wave duration; orthogonal P-wave morphology; interatrial block; electrocardiography; vectorcardiography

P-wave durations exceeding 110 ms, the normal cutoff in adults,[1] have been shown to be associated with several deleterious effects such as atrial tachyarrhythmias,[2] ischemic heart disease,[3] left atrial enlargement[4] as well as its electromechanical dysfunction,[5] and embolic stroke.[6] Similar associations have not been as extensively studied in the pediatric population, where such conditions may be relatively uncommon. However, cutoffs for P-wave durations in this population are naturally physiologically lower owing to reduced myocardial mass.[7] Many pediatric investigators and clinicians commonly accept "normal" P-wave values as 70 ms or shorter for infants and 90 ms or shorter for children.[8,9] Indeed, in a series of 232 healthy children, Kose et al.[10] showed that increase in P-wave duration was, in fact, proportionate to age. In a more recent study,[11] P-wave duration was significantly

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associated with age among hospitalized children as well as infants, where particular increase occurred at 10 years of age or older.

Analysis of unfiltered, signal-averaged P waves has revealed differences considering the prevalence of the electrocardiographic (ECG) signs of interatrial block other than P-wave duration between patient groups.[12–14] The three P-wave morphology classes identified[12–14] have been suggested to arise from differences in interatrial conduction.[13] The differences between the observed P-wave morphology classes are seen primarily in the terminal portion of the P wave and therefore likely reflect differences in left atrial activation.[15] A recent study[16] demonstrated a robust agreement between the P-wave morphology obtained by standard 12-lead ECG and left atrial breakthrough site determined using invasive mapping. The results of this study indicated that, in the vast majority of patients, P-wave morphology can be used to correctly identify the type of interatrial block, left atrial breakthrough site, and corresponding route of interatrial conduction.

Aim of this study was to evaluate the prevalence of interatrial block in healthy school-aged children when either the criterion of widened P-wave duration or the presence of abnormal morphology of orthogonal P waves was considered.

## METHODS

The study population consisted of 664 consecutively recruited children from the primary schools of Santorini island, Cyclades, Greece. None of the children had history of any cardiovascular or other disease and received any medications. All children had normal physical examination and a normal 12-lead surface ECG in the supine resting position. Children with left or right bundle branch block, atrioventricular block, ventricular preexcitation, or atrial fibrillation were excluded from the study. The study was approved by the Ministry of National Education and by our hospital's ethics committee. Informed consent was obtained from all children's parents. All study participants underwent a 12-lead digital ECG recording, by previously described techniques.[17]

To optimize the P-wave determination from 12-lead surface ECGs, we used a commercially available computer-based ECG system (Cardioperfect version 1.1; Cardio Control NV, Delft, The Netherlands), which enabled us to record all 12 ECG leads simultaneously at a sampling rate of 1200 Hz and a 12bit A/D conversion rate.[18] From each lead, the average complex was calculated by the MEANS (Modular ECG Analysis) system.[19] Individual average complexes were stored digitally and displayed on a high-resolution computer screen. Each lead was separately magnified with a magnification of 160 mm/s and 60 mm/mV.[18] The start and the end of the P wave were marked with the cursor on a high-resolution computer screen. The onset and offset of the P wave were defined as the junction between the P-wave pattern and the isoelectric line and marked with the cursor. Two independent investigators measured the P waves of all ECGs, without knowledge of subject assignment. The averages of the measurements of the two observers were used for comparisons. If the baseline noise was $>10\ \mu V$ and/or the peak to isoelectric line-P-wave amplitude $<15\ \mu V$, the lead was excluded from the analysis. No attempt was made to correct for missing leads and an ECG with $\leq 9$ measurable leads was excluded from the analysis.[18] After the measurement of all measurable P waves was completed in each ECG, maximum and minimum P-wave duration (see Fig. 1) and mean P-wave duration were calculated. Moreover, P-wave dispersion was calculated as the difference between the longest and the shortest P wave in any of the measured ECG leads.

Intra- and interobserver relative errors (the absolute difference between two observations divided by the mean and expressed as a percentage) for maximum P-wave duration were determined in 50 randomly selected study participants. To define intraobserver errors of measurement, one of the two investigators measured the durations of the P waves of all 50 ECGs twice. The intraobserver relative error for maximum P-wave duration was $5.2 \pm 3.5\%$, and the interobserver relative error was $7.3 \pm 6.8\%$.

To derive vectorcardiographic (VCG) descriptors of P-wave loop, orthogonal X, Y, and Z leads were reconstructed from the standard 12-lead ECGs.[20] The projections of the maximum P-wave vector on the frontal (xy), horizontal (xz), and right sagittal (yz) planes (see Fig. 1) were automatically calculated by our analysis system.[21,22] According to the previously published equations[23] and by use of the Pythagorean theorem, we calculated the amplitude

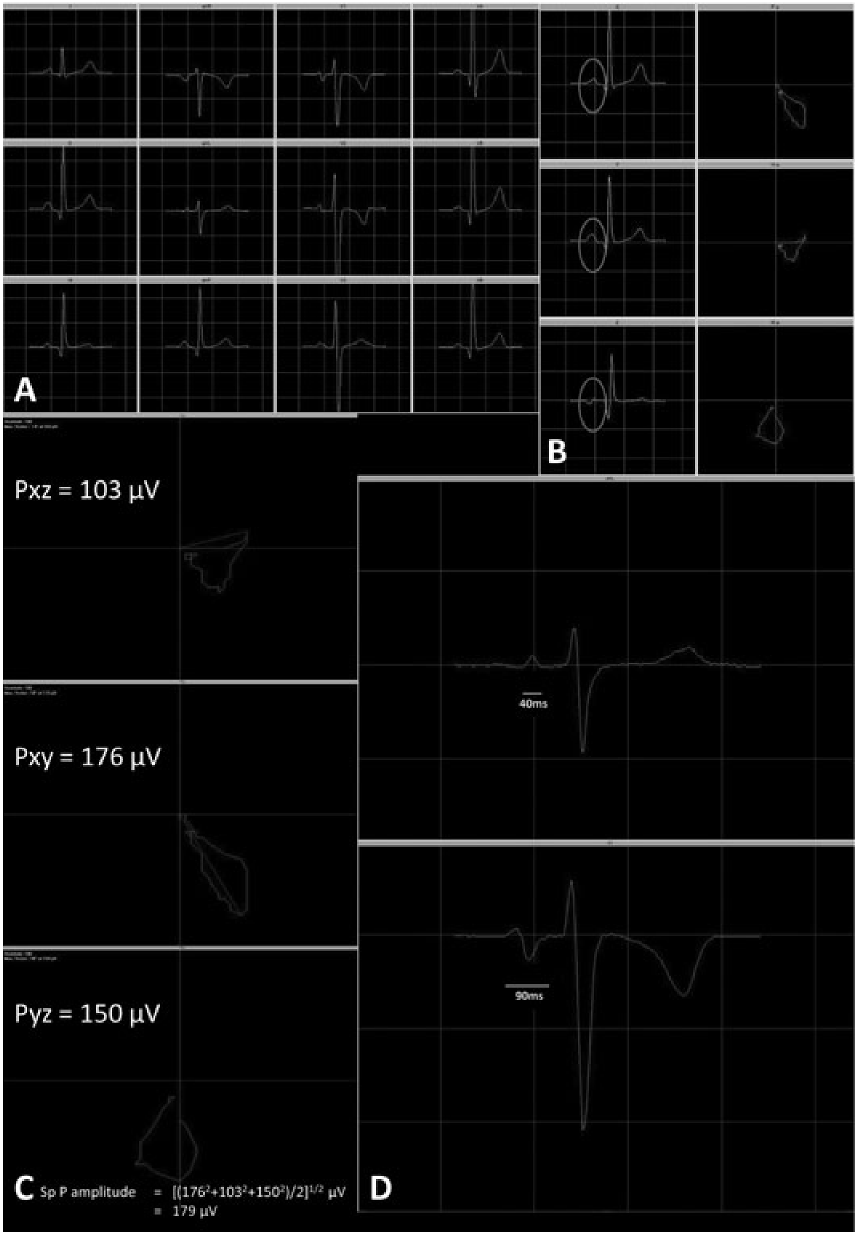


**Figure 1.** (A) Twelve-lead ECG obtained with signal averaging of raw beats by the Cardioperfect software. (B) The derived orthogonal ECG complexes (left) and the projections of the P-wave loop on the three orthogonal planes (right): frontal (Fp), horizontal (Hp) and right saggital (Rp). In this case, the P wave is positive in X and Y leads and biphasic (negative/positive) in Z lead, classifying this child under the *type II* orthogonal P-wave morphology (see text). (C) The projections of the maximum P-wave vector on (from top to bottom) the horizontal (xz), frontal (xy), and right sagittal (yz) planes, as automatically reconstructed by our analysis system. According to previously published equations and by use of the Pythagorean theorem, the amplitude of the maximum spatial P-wave vector (spatial P amplitude) was calculated from the formula: Spatial P amplitude $= [(Pxy^2 + Pxz^2 + Pyz^2)/2]^{1/2}$. (D) The minimum (top) and the maximum (bottom) P-wave duration, measured in this case in aVL and $V_1$ leads, correspondingly, of the 12 leads of Panel A. The quality of the signal was typically good in our data, allowing a fairly clear definition of the beginning and end of the P wave.

of the maximum spatial P-wave vector (spatial P amplitude) from the formula:

$$\text{Spatial P amplitude} = [(Pxy^2 + Pxz^2 + Pyz^2)/2]^{1/2}.$$

From the derived X, Y, and Z leads, the morphology of the orthogonal P-waves was assessed (see Fig. 1). The morphology subsequently was classified into one of the three, previously validated, predefined classes (*type I:* positive leads X and Y and negative lead Z; *type II:* positive leads X and Y and biphasic lead Z (−/+); and *type III:* positive lead X and biphasic signals in leads Y (+/−) and Z (−/+)).[13]

### Statistical Analysis

Continuous variables are summarized as mean ± standard deviation and were compared using the two-sided *t*-test. Categorical variables are expressed as counts and percentages and were compared using the chi-square test. Single linear regression analysis was used to test the correlation of maximum P-wave duration to age and heart rate. Statistical significance was signified by a P value of <0.05. Analyses were made using the SPSS 15.0 software package (SPSS Inc., Chicago, IL, USA).

## RESULTS

### Population

Basic demographic characteristics are presented in Table 1. In short, the studied population consisted of 664 children, with ages ranging from 6 to 14 years old (the vast majority among them, 98.2%, were aged 6–11 years old, corresponding to the six grades of primary school, as shown in Table 1).

### P-Wave Descriptors

The mean values of P-wave duration are shown in Table 1. Maximum P-wave duration was positively correlated to age (Pearson's correlation coefficient 0.252, P < 0.001), indicating a gradual increase of P-wave duration with age (Fig. 2). This parameter did not differ significantly between sexes (98.7 ± 9.2 ms in boys vs 99.4 ± 10.4 ms in girls, P = 0.339). On the other hand, maximum P-wave duration showed a weak ($R^2$ 0.039), but significant, positive correlation to heart rate (Pearson's correlation coefficient 0.197, P < 0.001) (Fig. 3). The correlations of P-wave duration to heart rate and age were independently significant (i.e., they remained significant after adjusting to each other).

**Table 1.** Population Characteristics

| Parameter | |
|---|---|
| N | 664 |
| Age (years; mean ± SD) | 8.5 ± 1.8 |
| Gender (male; N,%) | 349 (52.6%) |
| Age distribution (N) | |
| 6–7 years | 228 (34.3%) |
| 7–8 years | 192 (28.9%) |
| 9–10 years | 205 (30.9%) |
| 11–12 years | 128 (19.3%) |
| 13–14 years | 3 (0.5%) |
| Heart rate (bpm; mean ± SD) | 95.3 ± 15.8 |
| Mean P-wave duration (ms; mean ± SD) | 84.9 ± 9.5 |
| Maximum P-wave duration (ms; mean ± SD) | 99.0 ± 9.8 |
| P-wave dispersion (ms; mean ± SD) | 32.2 ± 12.5 |
| Spatial P amplitude (μV; mean ± SD) | 182.7 ± 69.0 |
| Orthogonal P-wave morphology (N, %) | |
| Type I | 478 (72.0%) |
| Type II | 178 (26.8%) |
| Type III | 1 (0.2%) |
| Indeterminate | 7 (1%) |

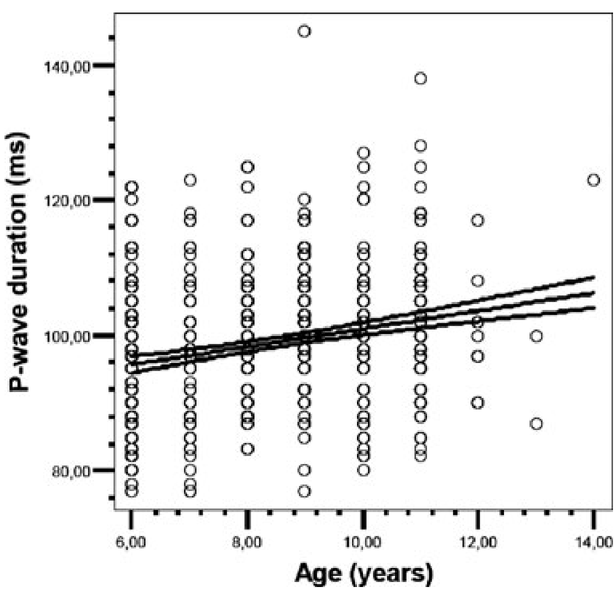


**Figure 2.** Scatterplot of maximum P-wave duration versus age with fitted regression line (±95% confidence intervals), indicating a significant increase with age (standardized beta coefficient = 0.252, P = $4{,}43 \times 10^{-11}$).

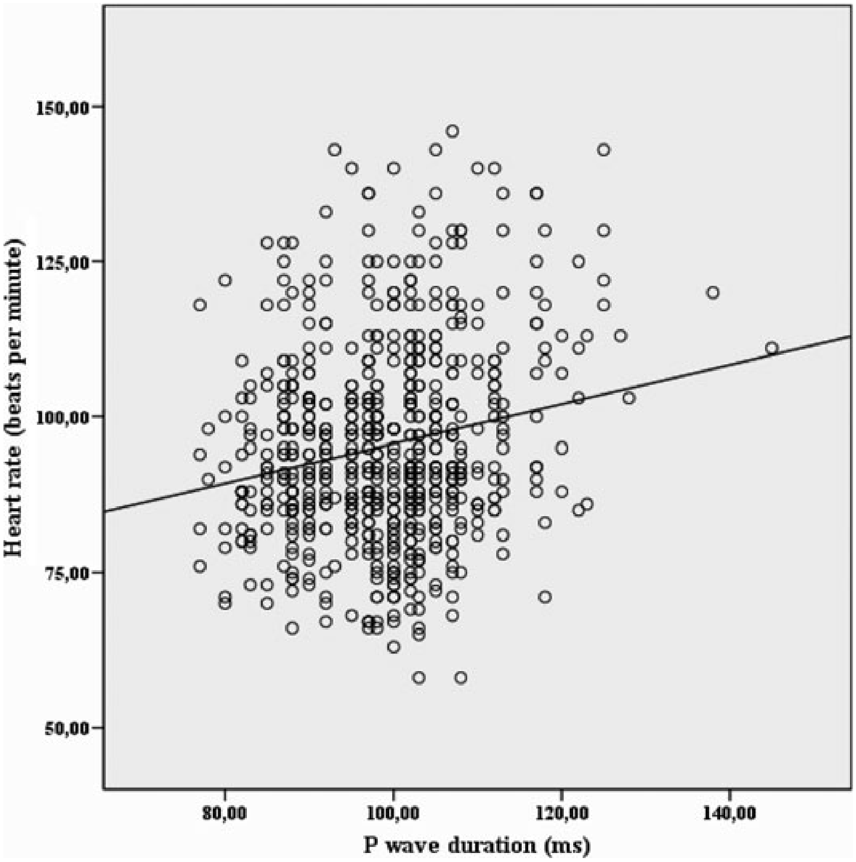


**Figure 3.** Positive correlation of maximum P-wave duration to heart rate (scatterplot with fitted regression line; standardized beta coefficient = 0.197, P = 3,61 × $10^{-7}$).

Using the 90-ms value as cutoff for "reference" P-duration, 502 (75.6%) children would be classified as having a maximum P-wave duration above reference range. Even a 100-ms cutoff would place 297 (44.7%) of the children in the "above-normal" zone, which, evidently, cannot have a true physiological meaning. The 95th and the 99th percentiles, which are frequently used as arbitrary cutoff values in pediatric population were in the overall population 117 ms and 125 ms, respectively. The same parameters per age group are presented in Table 2. It is obvious from this Table that the 95th and the 99th percentile of maximum P-wave duration demonstrate an almost steadily increasing trend with age, with values well above the traditional 90-ms and 100-ms cutoff points of "normal" P-wave duration in children.

The distribution of the three different types of P-wave morphology in the three orthogonal axes is shown in Table 1. There was only one child among the 664 studied with the, generally accepted as pathological, *type III* P-wave orthogonal morphology. *Type II* morphology was present in a large proportion of children (26.8%). There was a small but significant difference in the mean age of children with t*ype I* and t*ype II* P-wave orthogonal morphology (8.4 vs 8.9 years, P = 0.004), with a gradual

**Table 2.** The 95th and the 99th Percentiles of the Average Maximum P-Wave Duration per Age Group

| | Maximum P-Wave Duration (ms) | |
|---|---|---|
| **Age (Years)** | **95th Percentile** | **99th Percentile** |
| 6 | 113.0 | 122.0 |
| 7 | 115.4 | 123.0 |
| 8 | 116.8 | 125.0 |
| 9 | 117.0 | 145.0 |
| 10 | 115.1 | 126.7 |
| ≥11 | 118.0 | 134.8 |

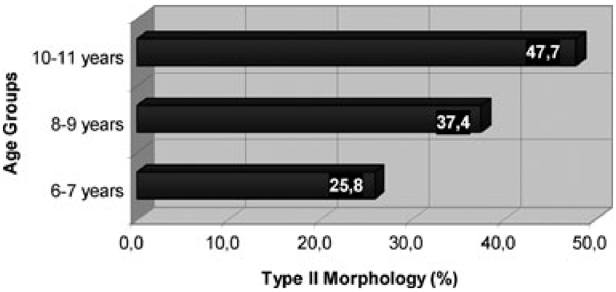


**Figure 4.** Age distribution of Type II P-wave morphology in the pediatric population.

increase in the proportion of *type II* morphology with age (Fig. 4). Contrary to P-wave duration, the P-wave morphology type differed between sexes (*Type II* morphology was significantly more frequent in boys than in girls, 32.4% vs 21.2%, respectively, P = 0.003).

P-wave morphology type was not in any way correlated to P-wave duration. The average value of maximum P-wave duration was 98.9 ms in children with t*ype I* morphology versus 99.2 ms in those with t*ype II* morphology (P = 0.715). There was also no difference in the proportion of children with maximum P-wave duration above the 90-ms cutoff in children with t*ype I* and *type II* morphology (74.1% vs 79.8%, respectively; P = 0.269). The same was true if the 100-ms cutoff was used (43.3% vs 48.3%, respectively; P = 0.346).

The median P-wave dispersion was 30 ms in the overall population (57 ms and 69 ms were the 95th and 99th percentiles, respectively). It did not differ significantly between boys and girls (32.2 ms vs 32.3 ms; P = 0.908), but was weakly correlated to heart rate (Pearson's correlation coefficient 0.087; P = 0.025). This correlation became insignificant when corrected for age (P-wave dispersion itself was significantly inversely correlated to age: Pearson's correlation coefficient 0.431; P < 0.001). P-wave dispersion was similar in children with type I or type II orthogonal P-wave morphology (P = 0.275).

The median spatial P-wave vector amplitude was 172 $\mu$V, with 306 $\mu$V and 401 $\mu$V at the 95th and 99th percentiles, respectively. It did not differ significantly between boys and girls (168.3 $\mu$V vs 170.0 $\mu$V; P = 0.830), but it was weakly correlated to age (Pearson's correlation coefficient 0.108; P = 0.006) and strongly correlated to heart rate (Pearson's correlation coefficient 0.632; P < 0.001). The correlation of the spatial P-wave vector to heart rate remained significant even after adjustment for age (P < 0.001). The amplitude of the spatial P-wave vector did not differ between children with type I and type II orthogonal P-wave morphology (P = 0.339).

## DISCUSSION

Interatrial conduction defects are increasingly being considered a vital contributor to supraventricular arrhythmogenesis.[2,24–28] Impairment of interatrial conduction can be observed as prolongation of the P wave on standard 12-lead ECG,[1,24] or the presence of abnormal P-wave morphology, mainly in orthogonal leads.[12–16] In this study, we considered both criteria for the evaluation of the presence of interatrial block in a healthy school-aged population. Our results indicate that abnormal P-wave morphology signifying the presence of interatrial block is very rare in a healthy pediatric population, while widened P waves are quite common, although currently classified as abnormal.

### Widened P Waves as a Marker of Interatrial Block

Prolonged P-wave durations have been described with certain pediatric medical conditions, although not all series had consistently used similar techniques. In one series where 50 mm/s paper speeds were used for optimization of P-wave measurements, Ozmen et al.[29] compared 43 pediatric patients who had pulmonary stenosis with 33 healthy but otherwise comparable pediatric controls and showed increased P-wave duration with disease. Similarly, Ho et al.[30] compared 94 children with isolated secundum atrial septal defect with healthy controls and observed a significantly increased mean P-wave duration on standard ECGs among those with such defects. In another series of adult patients who had previously undergone Fontan corrective procedure during childhood, Wong et al.[31] demonstrated a similar theme for widened P waves when compared with age- and sex-matched healthy controls. Similar significance of widened P-wave durations among patients with corrected tetralogy of Fallot[32] and children with left atrial abnormality[8,9] has also been noted.

One should consider that cutoffs for P-wave durations in the pediatric population are naturally physiologically lower owing to reduced myocardial

mass.[7] Many pediatric investigators and clinicians commonly accept "normal" P-wave values as 70 ms or shorter for infants and 90 ms or shorter for children.[8,9] Indeed, in a series of 232 healthy children, Kose et al.[10] showed that increase in P-wave duration was, in fact, proportionate to age in a cohort aged 7–15 years. In a more recent study,[11] P-wave duration was significantly associated with age among hospitalized children as well as infants, where particular increase occurred at 10 years of age or older. In that study,[11] the reported overall 27% prevalence of widened P waves among hospitalized children and infants is overwhelmingly high and contrasts the low prevalence of atrial tachyarrhythmias in this pediatric cohort.

In this study, the majority of healthy school-aged children showed P-wave values above the cutoff value of ≥90 ms that was considered as indicating "normality." Even when a 100-ms cutoff value was considered, almost 50% of the children were found to be in the "above-normal" zone. It is obvious that such arbitrarily defined cutoff values do not have a true physiological meaning and are significantly lower than the values of P-wave duration that are encountered in young patients suffering from congenital heart disease and/or atrial arrhythmias.[29–32] The duration of P waves as well as P-wave dispersion in healthy children should be considered an age-related phenomenon since they were found in our study to be positively correlated to age, as previously reported.[10,11] The association between maximum P-wave duration or P-wave dispersion and heart rate has already been reported.[33] The association of spatial P vector amplitude to heart rate and age has not previously been reported. Future studies should verify these results and evaluate their possible clinical significance.

## Interatrial Block and P-Wave Morphology

Current knowledge supports the concept that interatrial block remains largely underappreciated and often mislabeled as an insignificant epiphenomenon on the ECG.[34] In the absence of current standardized ECG software that supports P-wave duration readings, awareness of specific P-wave markers for interatrial block could aid clinicians in recognizing interatrial block where it probably counts most on the bedside ECG.[35] Advanced interatrial block, which has been shown to be caused by complete blockage of the Bachmann bundle,[26] is seen as a prolonged biphasic P wave in the inferior leads.[36,37] Bayés de Luna and coworkers provided the first ECG-criteria for interatrial block, including notes on P-wave morphology.[36] However, the use of standard 12-lead ECG is limited for the study of low-voltage P waves and the need for high-quality signal acquisition has been suggested.[22]

P-wave-triggered signal averaging of unfiltered P waves has previously been used to study orthogonal P-wave morphology in patients with paroxysmal atrial fibrillation, hypertrophic cardiomyopathy, and control patients.[13,14,16,38] Three types of orthogonal P-wave morphology have been observed (as described in the Methods section) and validated.[13,14,16,38] *Type I* is thought to represent an activation sequence directed right-left, superior-inferior, and posterior-anterior. This is best explained by activation via posteriorly located interatrial conduction routes. *Type II* with biphasic lead Z consequently differs in regards to an anterior-posterior-anterior activation sequence, best explained by interatrial conduction via the anteriorly located Bachmann's bundle. In *Type III*, the presence of a biphasic signal in lead Y was always accompanied by a biphasic lead Z, hence the activation direction was characterized not only by the superior-to-inferior-to-superior route but also by posterior-to-anterior-to-posterior propagation, indicating not only Bachmann bundle block but also a block or delayed conduction in the posterior route.[12,16] In a recently published report,[12] *type II* morphology was significantly more common with increasing age with a marked increase in prevalence in healthy subjects after the age of 50, while *type III* morphology was not observed in the healthy population of the same study.

In our study, *type I* morphology of orthogonal P waves prevailed (72%), but *type II* morphology was also present in a large proportion of children (26.8%). Only one child among the 664 studied, demonstrated the, generally accepted as pathological, *type III* P-wave orthogonal morphology. This is largely expected, since this is a clinically healthy population, with no evidence of cardiac disease. There was a small but significant difference in the mean age of children with *type I* and *type II* P-wave orthogonal morphology, with a gradual increase in the proportion of *type II* morphology with age. P-wave morphology type was not in any way correlated to P-wave duration. This observation may be indicative of the fundamentally different theoretical information, regarding atrial depolarization,

offered by P-wave duration and P-wave morphology in the orthogonal leads.

## Limitations

A limitation of our study is that we did not assess the effects of body mass index on either P-wave duration or P-wave morphology. Moreover, our methodology of measuring P-waves duration after signal-averaging and magnification on a high-resolution screen differed from standard manual measurements on paper-printed ECGs. It is possible that our methodology may have affected our results. However, previous studies have demonstrated that onscreen measurements of P waves are consistent with other manual methods and provide more stable results.[18] Finally, orthogonal (X, Y, Z) leads were derived from standard 12 ECG leads by use of the inverse Dower method. This method may be limited for the evaluation of P vector loop features.[39]

## Conclusions

The present findings suggest that P-wave duration in children falls well above cutoff values considered as normal in healthy children. This could mean that higher reference cut-points should be used. On the other hand, abnormal P-wave morphology signifying the presence of interatrial block is very rare in a healthy pediatric population. Future studies are needed to verify our results and to associate the presence of either marker of interatrial block in children with arrhythmogenesis.

## REFERENCES

1. Willems JL, Robles de Medina EO, Bernard R, et al. Criteria for intraventricular conduction disturbances and pre-excitation. World Health Organizational/International Society and Federation for Cardiology Task Force Ad Hoc. J Am Coll Cardiol 1985;5:1261–1275.
2. Dilaveris PE, Gialafos EJ, Sideris SK, et al. Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. Am Heart J 1998;135:733–738.
3. Dilaveris PE, Andrikopoulos GK, Metaxas G, et al. Effects of ischemia on P wave dispersion and maximum P wave duration during spontaneous anginal episodes. Pacing Clin Electrophysiol 1999;22:1640–1647.
4. Munuswamy K, Alpert MA, Martin RH, et al. Sensitivity and specificity of commonly used electrocardiographic criteria for left atrial enlargement determined by M-mode echocardiography. Am J Cardiol 1984;53:829–832.
5. Goyal SB, Spodick DH. Electromechanical dysfunction of the left atrium associated with interatrial block. Am Heart J 2001;142:823–837.
6. Lorbar M, Levrault R, Phadke JG, et al. Interatrial block as a predictor of embolic stroke. Am J Cardiol 2005;95:667–668.
7. Zeek PM. Heart weight. I. The weight of the normal human heart. Arch Pathologica 1942;34:820–832.
8. Park MK, Guntheroth WG. How to read pediatric ECGs (3rd ed.). St. Louis: Mosby, 1992.
9. Garson A. Electrocardiography. In: Garson A, Bricker JT, Fisher DJ, Neish SR (eds.): The science and practice of pediatric cardiology, 2nd Edition. Baltimore, MD, Lippincott Williams & Wilkins, 1998:735–739.
10. Köse S, Kiliç A, Iyisoy A, et al. P wave duration and P dispersion in healthy children. Turk J Pediatr 2003;45:133–135.
11. Loo RM, Ariyarajah V, O'Brien D, et al. High prevalence of widened P waves among pediatric patients in 2 separate hospitals. J Electrocardiol 2008;41:63–67.
12. Havmoller R, Carlson J, Holmqvist F, et al. Age-related changes in P wave morphology in healthy subjects. BMC Cardiovasc Disord 2007;7:22–28.
13. Holmqvist F, Platonov PG, Carlson J, et al. Variable interatrial conduction illustrated in a hypertrophic cardiomyopathy population. Ann Noninvasive Electrocardiol 2007;12:227–236.
14. Platonov PG, Carlson J, Ingemansson MP, et al. Detection of inter-atrial conduction defects with unfiltered signal-averaged P-wave ECG in patients with lone atrial fibrillation. Europace 2000;2:32–41.
15. Lemery R, Birnie D, Tang AS, et al. Normal atrial activation and voltage during sinus rhythm in the human heart: An endocardial and epicardial mapping study in patients with a history of atrial fibrillation. J Cardiovasc Electrophysiol 2007;18:402–408.
16. Holmqvist F, Husser D, Tapanainen JM, et al. Interatrial conduction can be accurately determined using standard 12-lead electrocardiography: Validation of P-wave morphology using electroanatomic mapping in man. Heart Rhythm 2008;5:413–418.
17. Gialafos E, Psaltopoulou T, Papaioannou TG, et al. Prevalence of interatrial block in young healthy men <35 years of age. Am J Cardiol 2007;100:995–997.
18. Dilaveris P, Batchvarov V, Gialafos J, et al. Comparison of different methods for manual P wave duration measurement in 12-lead electrocardiograms. Pacing Clin Electrophysiol 1999;22:1532–1538.
19. Van Bemmel JH, Kors JA, Van Herpen G. Methodology of the modular ECG analysis system MEANS. Methods Inf Med 1990;29:346–353.
20. Carlson J, Havmöller R, Herreros A, Platonov P, Johansson R, Olsson B. Can orthogonal lead indicators of propensity to atrial fibrillation be accurately assessed from the 12-lead ECG? Europace 2005;7(Suppl. 2):39–48.
21. Dilaveris P, Pantazis A, Gialafos E, et al. Determinants of electrocardiographic and spatial vectorcardiographic descriptors of ventricular repolarization in normal subjects. Am J Cardiol 2001;88:912–914.
22. Dilaveris PE, Gialafos JE. Future concepts in P wave morphological analyses. Card Electrophysiol Review 2002;6:221–224.
23. Ishizawa K, Ishizawa K, Motomura M, et al. High reliability rates of spatial pattern analysis by vectorcardiogram in assessing the severity of eccentric left ventricular hypertrophy. Am Heart J 1976;91:50–57.
24. Ariyarajah V, Frisella ME, Spodick DH. Reevaluation of the criterion for interatrial block. Am J Cardiol 2006;98:936–937.
25. Ariyarajah V, Asad N, Tandar A, et al. Interatrial block: Pandemic prevalence, significance, and diagnosis. Chest 2005;128:970–975.
26. Cosío FG, Martín-Peñato A, Pastor A, et al. Atrial activation mapping in sinus rhythm in the clinical electrophysiology

laboratory: Observations during Bachmann's bundle block. J Cardiovasc Electrophysiol 2004;15:524–531.
27. Agarwal YK, Aronow WS, Levy JA, et al. Association of interatrial block with development of atrial fibrillation. Am J Cardiol 2003;91:882.
28. Bayés de Luna A, Guindo J, Viñolas X, et al. Third-degree inter-atrial block and supraventricular tachyarrhythmias. Europace 1999;1:43–46.
29. Ozmen N, Cebeci BS, Kardesoglu E, et al. P wave dispersion in increased in pulmonary stenosis. Indian Pacing Electrophysiol J 2006;6:25–30.
30. Ho TF, Chia EL, Yip WC, et al. Analysis of P wave and P dispersion in children with secundum atrial septal defect. Ann Nonivasive Electrophysiol 2001;6:305–309.
31. Wong T, Davlouros PA, Li W, et al. Mechano-electrical interaction late after Fontan operation: Relation between P-wave duration and dispersion, right atrial size and atrial arrhythmias. Circulation 2004;109:2319–2325.
32. Hallioglu O, Aytemir K, Celiker A. The significance of P wave duration and P wave dispersion for risk assessment of atrial tachyarrhythmias in patients with corrected tetralogy of Fallot. Ann Noninvasive Electrocardiol 2004;9:339–344.
33. Gialafos EJ, Dilaveris PE, Synetos AG, et al. P wave analysis indices in young healthy men: Data from the Digital Electrocardiographic study in Hellenic Air force Servicemen (DEHAS). Pacing Clin Electrophysiol 2003;26(Pt.II):1–6.
34. Ariyarajah V, Puri P, Spodick DH. Clinician underappreciation of interatrial block in a general hospital population. Cardiology 2005;104:193–195.
35. Ariyarajah V, Apiyasawat S, Puri P, et al. Specific electrocardiographic markers of P-wave morphology in interatrial block. J Electrocardiol 2006;39:380–384.
36. Bayes de Luna A, Fort de Ribot R, Trilla E, et al. Electrocardiographic and vectorcardiographic study of interatrial conduction disturbances with left atrial retrograde activation. J Electrocardiol 1985;18:1–13.
37. Castillo A, Vernant P. Disorders of intraauricular conduction due to block of Bachman's bundle. Arch Mal Coeur Vaiss 1971;64:1490–1503.
38. Holmqvist F, Platonov PG, Havmoller R, et al. Signal-averaged P wave analysis for delineation of interatrial conduction – Further validation of the method. BMC Cardiovasc Disord 2007;7:29–36.
39. Guillem MS, Sahakian AV, Swiryn S. Derivation of orthogonal leads from the 12-lead electrocardiogram. Performance of an atrial-based transform for the derivation of P loops. J Electrocardiol 2008;41:19–25.