P-Wave Morphology Is Unaffected by Atrial Size: A Study in Healthy Athletes

Richard Petersson, M.D.,* Hilde M. Berge, M.D.,† Gard F. Gjerdalen, M.D.,‡
Jonas Carlson, M.Sc., Ph.D.,* Fredrik Holmqvist, M.D., Ph.D.,*
Kjetil Steine, M.D., Ph.D.,§ and Pyotr G. Platonov, M.D., Ph.D.*

From the *Department of Cardiology, Lund University and Center for Integrative Electrocardiology at Lund University, Lund, Sweden; †Oslo Sports Trauma Research Center, Norwegian School of Sport Sciences, Oslo, Norway; ‡Section of Vascular Investigations, Oslo University Hospital, Aker, Oslo, Norway and Bjorknes College, Oslo, Norway; and §Department of Cardiology, Akershus University Hospital, Lørenskog, Norway

Background: Orthogonal P-wave morphology has previously been described in different populations, but its relation to atrial size has not been studied in detail. In this study, we investigated whether atrial size affects P-wave morphology in athletes, who are known to have different degrees of atrial enlargement.

Methods: A total of 504 healthy, male, professional soccer players were included (median age 25 years). All underwent echocardiographic and 12-lead electrocardiographic (ECG) recordings. The ECG was transformed into orthogonal leads, using the inverse Dower transform. The association between echocardiographic parameters and standard P-wave measures (i.e., orthogonal morphology, left atrial abnormality assessed as negative P-wave terminal force [PTF] in lead $V_1 > 0.04 \text{ mm} \times s$, and duration) was analyzed.

Results: The vast majority had either type 1 P-wave morphology (75%) (positive leads X and Y and negative lead Z) or type 2 P-wave morphology (22%) (positive leads X and Y and biphasic lead Z [negative/positive]). Left atrial enlargement ($\geq 29 \text{ mL/m}^2$) was found in 79% on echocardiography. There was no significant difference in left atrial end-systolic volume, left or right atrial diameters, or right atrial area between individuals with different P-wave morphologies. ECG signs of left atrial abnormality were found in eight subjects, who did not have significantly larger left atrial dimensions than the rest.

Conclusions: We demonstrated that P-wave morphology does not depend on the size of the atria in young, healthy athletes, and that PTF is not a reliable marker of left atrial enlargement in the current population.

Ann Noninvasive Electrocardiol 2014;19(4):366–373

P-wave morphology; atrium; atrial electrophysiology; electrocardiography; echocardiography

Analysis of P-wave morphology can reveal important information about the three-dimensional propagation of the atrial depolarization.^{1,2} In a population with reduced left ventricular function, abnormal P-wave morphology has been shown to be associated with the development of atrial

fibrillation.³ Three main orthogonal P-wave morphology types have been described, with the major differences primarily seen in the sagittal plane (Fig. 1).^{2,4} The impact of atrial size, in the absence of conduction disturbances, on P-wave morphology has not yet been fully investigated.

Address for correspondence: Richard Petersson, M.D., Department of Cardiology, Skåne University Hospital, SE-22185 Lund, Sweden. Fax: +46-46-15-78-57; E-mail: richard.petersson@med.lu.se

Grants from the Royal Norwegian Ministry of Culture (Oslo, Norway), the South-Eastern Norway Regional Health Authority (Hamar, Norway), the International Olympic Committee (Lausanne, Switzerland), The Norwegian Olympic Committee & Confederation of Sport (Oslo, Norway), Norsk Tipping AS (Hamar, Oslo), the Swedish Heart-Lung Foundation (Stockholm, Sweden), governmental funding of clinical research within the Swedish National Healthcare System (Stockholm, Sweden), Skånes universitetssjukhus stiftelser och donationer (Lund, Sweden), and Södra Regionvårdsnämnden (Lund, Sweden).

© 2014 Wiley Periodicals, Inc.
DOI:10.1111/anec.12132

Figure 1. The three different types of orthogonal P-wave morphologies. Type 1 (predominantly positive leads X and Y and predominantly negative lead Z), type 2 (predominantly positive leads X and Y and biphasic lead Z [negative, positive]), and type 3 (predominantly positive lead X and biphasic leads Y [positive, negative] and Z [negative, positive]).

Holmqvist et al. reported significantly larger left atrial diameters in hypertrophic cardiomyopathy patients with P-wave morphology consistent with complete atrial block, but no difference between patients with other P-wave morphologies.⁵ In the present study, we sought to answer the question of possible pure atrial volume effects on P-wave morphology, as opposed to the known effects of atrial conduction defects. In order to answer our question, we chose to investigate healthy soccer players. It has previously been shown that athletes in general⁶ and soccer players specifically⁷ have greater left atrial volumes than nonathletes. Based on previous knowledge, our hypothesis was that P-wave morphology is primarily a reflection of interatrial conduction, and not so much affected by atrial volumes in healthy individuals.

METHODS

Study Population

In total, 604 male professional soccer players, aged 18 to 40 years, were screened for inclusion in this study. They were all in the Norwegian elite or 1st division leagues. The process of inclusion and examination was performed during a pre-season training camp in La Manga, Spain, from February to April 2008. The size of the study population

was limited to the attending soccer players. All subjects filled out a questionnaire regarding ethnicity. Eleven experienced cardiologists, one resident and one cardiology fellow (both trained in echocardiography) from hospitals in the Oslo region, Norway, performed the echocardiographic screening. The study was approved by the Regional Ethical Committee.

Echocardiographic Studies

All transthoracic echocardiographic recordings were performed with a 2.5-MHz transducer (Vivid 7 and Vivid i; GE Vingmed Ultrasound AS, Horten, Norway). The digital data were transferred to a computer (Dell Optiplex 755) for offline analysis at the core echolab, Oslo University Hospital, Aker, using the software EchoPAC (BT08) (GE Vingmed Ultrasound AS, Horten, Norway). The echocardiographic recordings were performed in standard parasternal long- and short-axis and apical four-chamber views with the study subjects in left lateral supine position.

All measurements were performed as recommended by the American Society of Echocardiography.⁸ Left atrial size was measured as left atrial end-systolic volume from the apical two- and four-chamber views using the area-length method, and left atrial diameter from parasternal long-axis view.⁸ The end-systolic area of the right atrium was measured in an apical four-chamber view. The right atrial end-systolic diameter was measured as the diameter of the right atrium, perpendicular to the longitudinal axis of the atrium.⁹

Data regarding chamber quantification were indexed to body surface area (BSA), in accordance with recommendations.^{8,10} The simplified formula from Mosteller 1987 was used to calculate BSA.¹¹

The echocardiographic analyses were all conducted by one of the authors (GFG).

Electrocardiographic Data Acquisition and Analysis

Standard, 12-lead, 10-second electrocardiograms (ECGs) were recorded from all individuals using CARDIOLINE, RealClick version 3.5.4 (Milan, Italy). These were transformed into derived vectorcardiograms (dVCGs), using the inverse Dower transform, to enable orthogonal lead analysis.¹² A high-pass filter with a cutoff frequency of 0.5 Hz

was applied to the dVCGs to eliminate slow baseline drift due to respiratory movement of the thorax. A 50-Hz band-stop filter was used to reduce power line interference. The QRS complexes were detected automatically. Similar QRS complexes were clustered together using a cross-correlation coefficient of $\rho > 0.9$ in order to exclude artifacts and erroneous beats (e.g., ventricular premature complexes). The cluster with the most QRS complexes was used for further analyses. A 250-ms time-window preceding the QRS complexes was used to extract the P-waves. This time-window was on occasion manually prolonged and/or moved in cases with unusually long PQ time. The individual P-waves were time-shifted to achieve maximum correlation and were then sorted into different clusters based on a cross-correlation coefficient of $\rho > 0.9$. The P-waves in each cluster were then signal-averaged. The beginning and end of the signal-averaged P-waves were defined manually. Finally, the morphology of the signal-averaged P-waves was classified as one of three predefined types (Fig. 1). P-waves that did not match any of the mentioned three types were denoted "atypical." The largest P-wave cluster in a recording was considered for further analysis. Please refer to refs.⁴ and¹³ for details on the above described method, which has been used in previous studies.^{1,2,4} Recordings with less than three P-waves in the largest P-wave cluster were excluded from further analyses. Individuals who did not meet this requirement were excluded.

All ECGs were also automatically analyzed regarding P terminal force in lead V₁ (PTF). PTF was defined as the product of amplitude and duration of the terminal phase of the P-wave (the negative component when the P-wave was biphasic) in lead V₁ (in mm × s).^{14,15} A negative PTF of more than 0.04 mm × s is considered an electrocardiographic marker of left atrial abnormality.¹⁵

Statistical Analysis

Data are presented as median (and lower and upper quartiles), and were analyzed using Kruskal-Wallis to test for significant differences. When comparing only two groups, the Mann-Whitney U test was used, and the Hodges-Lehman analysis was applied to calculate a 95% confidence interval for the difference in medians. For correlation analysis between two variables, the Pearson

correlation coefficient, r , and the coefficient of determination, R^2 , were calculated. Because of the known effects of ethnicity¹⁶ and BMI (body mass index)¹⁷ on P-wave characteristics, linear regression models adjusted for these factors were applied. All tests were two-sided and $P < 0.05$ was considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics version 21.0 for Mac OS X.

RESULTS

Data Availability

Altogether, 595 (99%) professional soccer players agreed to participate in the present study. In 91 cases, the ECG quality was poor, and they were consequently excluded. Ultimately, we ended up with 504 subjects with one unique ECG each.

In 10 and 11 cases, respectively, the left atrial diameter and left atrial end-systolic volume could not be measured because of suboptimal image quality, and they were excluded from these specific analyses.

Study Population

Three-quarters of the subjects had type 1 P-wave morphology and roughly one-fifth had type 2 P-wave morphology. The rest had atypical ($n = 14$) P-wave morphology. None had type 3 P-wave morphology. The median age of the entire study population was 25 years and was not significantly different between the groups with different P-wave morphologies. The vast majority of the study participants were white (84%) and the distribution of ethnicity did not differ between the groups. The median BMI was 23.7 kg/m², the median BSA was 2.0 m² and the median blood pressure was 121/70 mmHg, and none of these significantly differed between the three groups with different P-wave morphologies. Please refer to Table 1 for a full description.

P-Wave Morphology

The median PQ time for the whole study population was 167 ms and did not differ significantly between the groups with different P-wave morphologies. One individual was diagnosed with AV block II, Mobitz type I, and was therefore excluded from the PQ time analyses. The median

Table 1. Clinical Characteristics of the Study Population as a Whole and with Respect to P-Wave Morphology.

	Study Population (n = 504)	P-Wave Morphology 1 (n = 380; 75.4%)	P-Wave Morphology 2 (n = 110; 21.8%)	Atypical P-Wave Morphology (n = 14; 2.8%)	P
Age (years)	25 (21–28)	25 (21–28)	26 (22–28)	26 (22–28)	0.60
Ethnicity (white/black/other)	423 (84%)/45 (9%)/36 (7%)	318 (84%)/35 (9%)/27 (7%)	92 (84%)/9 (8%)	13 (93%)/1 (7%)/0 (0%)	0.83
BMI (kg/m ²)	23.7 (22.9–24.5)	23.7 (22.9–24.5)	23.7 (22.8–24.4)	23.7 (22.4–25.4)	0.92
BSA (m ²)	2.0 (1.9–2.1)	2.0 (1.9–2.1)	2.0 (1.9–2.1)	2.0 (1.9–2.1)	0.30
Systolic BP (mmHg)	121 (115–129)	121 (115–129)	121 (115–129)	129 (119–136)	0.09
Diastolic BP (mmHg)	70 (64–75)	69 (64–75)	71 (65–75)	73 (68–78)	0.07
PQ time (ms)	167 (152–185)*	167 (151–185)	172 (155–190)*	165 (138–188)	0.19
P-wave duration (ms)	132 (123–140)	131 (121–140)	137 (129–145)	122 (105–132)	<0.0001
LA diameter (cm)	3.5 (3.3–3.7)	3.5 (3.2–3.7)	3.5 (3.3–3.7)	3.6 (3.5–3.9)	0.08
LA diameter/BSA (cm/m ²)	1.7 (1.6–1.8)	1.7 (1.6–1.8)	1.7 (1.6–1.8)	1.8 (1.8–1.9)	0.08
LAESV (mL)	71.2 (58.7–85.4)	71.4 (58.7–86.0)	70.2 (55.7–83.0)	79.3 (65.0–100.7)	0.25
LAESV/BSA (mL/m ²)	35.8 (29.6–42.0)	36.2 (29.6–42.1)	33.9 (28.9–40.7)	38.8 (32.9–49.4)	0.14
RAESD (cm)	4.5 (4.1–4.9)	4.5 (4.1–4.9)	4.6 (4.1–4.9)	4.7 (4.2–5.2)	0.43
RAESD/BSA (cm/m ²)	2.2 (2.0–2.4)	2.2 (2.0–2.4)	2.3 (2.0–2.5)	2.5 (2.0–2.5)	0.53
RAESA (cm ²)	21.3 (18.6–24.2)	21.2 (18.5–24.2)	21.8 (18.5–24.4)	21.7 (20.4–24.5)	0.52
RAESA/BSA (cm ² /m ²)	10.6 (9.3–12.1)	10.6 (9.3–12.1)	10.5 (9.3–12.0)	11.3 (10.0–12.6)	0.55

Median (lower and upper quartiles); BMI = body mass index; BSA = body surface area; BP = blood pressure; LA = left atrial; LAESV = left atrial end-systolic volume; RAESD = right atrial end-systolic diameter; RAESA = right atrial end-systolic area. Analyzed using Kruskal-Wallis, except ethnicity, which was analyzed with chi-square test.

*One individual with AV block II, Mobitz type 1, was excluded (PQ time 402 ms).

P-wave duration for the entire study population was 132 ms, and it was significantly longer in subjects with type 2 P-wave morphology (137 ms) compared to subjects with type 1 (131 ms), $P < 0.0001$. However, the two groups overlapped extensively. The median P-wave duration in the group with atypical P-wave morphology (122 ms) was significantly shorter than in subjects with type 1 ($P = 0.006$) and type 2 ($P < 0.001$) morphologies.

Echocardiographic Measurements

The median left atrial end-systolic volume/BSA was 35.8 mL/m². The median left atrial diameter/BSA was 1.7 mL/m². The majority of the subjects (79%) had enlarged left atria, i.e., above the normal reference range, according to the definition by Lang et al. (≥ 29 mL/m²).⁸ There was no significant difference in left atrial end-systolic volume, left atrial end-systolic volume/BSA, left atrial diameter or left atrial diameter/BSA between the different P-wave morphologies (Table 1).

We specifically compared the median of the left atrial end-systolic volume/BSA between the two most common P-wave morphologies, i.e., type 1 (36.2 mL/m²) and type 2 (33.9 mL/m²), and found a nonsignificant difference of 1.6 mL/m² (95% confidence interval -0.4 to 3.6 mL/m²). Looking at the right atrium, the median right atrial end-systolic area/BSA was 10.6 cm²/m² and the median right atrial end-systolic diameter/BSA was 2.2 cm/m². There was no significant difference in median right atrial end-systolic area/BSA ($P = 0.55$) or diameter/BSA ($P = 0.53$) between the different P-wave morphologies.

In a linear regression model adjusted for BMI and ethnicity, P-wave duration ($B = 0.06$ [95% CI: 0.002–0.117]; $P = 0.043$) and PQ time ($B = 0.18$ [95% CI: 0.064–0.293]; $P = 0.002$) were positively correlated to non-BSA-indexed left atrial end-systolic volume. Not adjusting for BMI and ethnicity, P-wave duration ($B = 0.06$ [95% CI: 0.007–0.121]; $P = 0.028$) and PQ time ($B = 0.18$ [95% CI: 0.068–0.294]; $P = 0.002$) were still correlated to non-BSA-indexed left atrial end-systolic volume.

P Terminal Force in Lead V₁

Of the 504 subjects, 144 had a measurable negative PTF. The median negative PTF among them was 0.016 (0.009–0.024) mm × s. There was

no significant correlation between the degree of negative PTF and P-wave duration ($r = 0.161$, $R^2 = 0.026$), $P = 0.054$. Only eight subjects met the criterion for electrocardiographic left atrial abnormality. They did not have a significantly larger median left atrial end-systolic volume or a greater median left atrial diameter than the rest of the population. Neither did they have longer P-wave durations (Table 2). In addition, using a linear regression model adjusted for BMI and ethnicity, we found no significant correlation between non-BSA-indexed left atrial end-systolic volume and the degree of log-transformed negative PTF ($B = -0.001$ [95% CI: -0.003–0.002]; $P = 0.69$) as was the case for the nonadjusted linear regression model ($B = 0$ [95% CI: -0.003–0.002]; $P = 0.76$).

DISCUSSION

As hypothesized, the vast majority of this population of healthy athletes without known conduction abnormalities (one individual was diagnosed with AV block II, Mobitz type 1) had enlarged left atria. They were therefore suitable for studying the effect of left atrial volume on P-wave morphology. We did not find any significant differences in atrial dimensions between subjects with different P-wave morphologies. Thus, our findings strengthen the view that P-wave morphology is indeed a result of interatrial conduction and not dependent on the size of the atria.

P-Wave Morphology and Left Atrial Size

The distribution of P-wave morphologies was roughly what one would expect in young (less than 50 years of age), healthy people¹⁸; i.e., predominantly type 1 and some type 2. Previously, it has been shown that type 1 may be considered the "healthiest,"³ while type 2 is significantly more common among patients with lone paroxysmal atrial fibrillation,¹⁹ and that both type 2 and 3 morphologies are more common among patients with hypertrophic cardiomyopathy.⁵ Atypical P-wave morphology has been demonstrated in patients with arrhythmogenic right ventricular cardiomyopathy.²

The three different P-wave morphologies are likely to, at least in part, represent different atrial depolarization paths.^{1,2} It is reasonable to believe that the morphology of the initial part of the P-wave primarily depends on the variable location

Table 2. Left Atrial Dimensions, Common Atrial ECG Parameters and PTF.

	PTF ≤ 0.04 mm $\times$ s (n = 496)	PTF > 0.04 mm $\times$ s (n = 8)	P
PQ time (ms)	167 (152–185)*	182 (165–192)	0.17
P-wave duration (ms)	132 (123–140)	136 (133–141)	0.15
LA diameter (cm)	3.5 (3.3–3.7)	3.5 (3.2–3.7)	0.74
LA diameter/BSA (cm/m ²)	1.7 (1.6–1.8)	1.7 (1.5–1.9)	0.61
LAESV (mL)	71.2 (58.7–85.3)	71.5 (58.4–88.7)	0.91
LAESV/BSA (mL/m ²)	35.8 (29.6–41.9)	35.5 (28.4–45.3)	0.97

Median (lower and upper quartiles); PTF = P terminal force in lead V₁; BSA = body surface area; LA = left atrial; LAESV = left atrial end-systolic volume. Analyzed with Mann-Whitney U test.

*One individual with AV block II, Mobitz type 1, was excluded (PQ time 402 ms).

of the atrial pacemaker region,²⁰ while the terminal part of the P-wave to a large extent is determined by the left atrial depolarization sequence, largely depending on the transseptal interatrial conduction path.¹ We specifically looked at the two most common P-wave morphologies, i.e., type 1 and type 2, and found no significant difference in left atrial end-systolic volumes. In addition, there was no significant difference in right atrial dimensions between the different morphologies.

Holmqvist et al. have previously reported a significantly larger left atrial diameter in hypertrophic cardiomyopathy patients with type 3 P-wave morphology compared to patients with type 1 morphology, and no significant diameter difference between types 1 and 2 in the same patient material.⁵ Regarding types 1 and 2, the results of the present study are in keeping with those (none had type 3 morphology in our material). It is however important to note that, apart from being younger, healthy and male-only, the individuals in the present study had smaller left atria (median diameter 3.5 cm) than the patients in the study by Holmqvist et al. (mean diameter 4.5 ± 0.8 cm). Our group recently investigated P-wave morphology as a predictor of cardiac resynchronization therapy success in the Multicenter Automatic Defibrillator Implantation With Cardiac Resynchronization Therapy (MADIT-CRT) study population.²¹ This population consisted of patients with ischemic or nonischemic cardiomyopathy, an ejection fraction of 30% or less and prolonged intraventricular conduction (a QRS duration of 130 ms or more). We found that patients with atypical P-wave morphology had significantly smaller mean left atrial volumes than the groups with type 1 and type 2 morphologies pooled together. However, it is difficult to compare that population with the one in the present study because of their

different constitutions. Regarding the left atrial enlargement seen in the healthy soccer players, one may speculate that this is the result of a benign, compensatory mechanism due to volume overload, while it is the result of a pathological process in the cardiomyopathy cases resulting in disturbed interatrial conduction, which presents as a higher prevalence of type 2 and type 3 P-wave morphologies. This line of reasoning is supported by the finding that left atrial enlargement, in the absence of supraventricular tachyarrhythmias, is common in athletes.⁶

Based on their finding of atrial enlargement in athletes, Pelliccia et al. suggested that a transverse left atrial diameter exceeding 5.0 cm in men (4.5 cm in women) should suggest pathological enlargement and not be interpreted as the result of a physiological compensatory mechanism.⁶ Indeed, in our population, none had a left atrial diameter exceeding 4.5 cm.

P Terminal Force in Lead V₁

The concept of PTF was introduced by Morris et al. in 1964.¹⁴ They showed that an abnormal PTF was associated with left-sided heart disease (including, but not limited to, aortic and mitral valve involvement). A negative PTF of more than 0.04 mm $\times$ s is known to be associated with several left atrial abnormalities, such as atrial dilation, atrial muscular hypertrophy, elevated intra-atrial pressures, and delayed conduction. Since these conditions often coexist and express themselves similarly on an ECG, PTF should not be used to diagnose a specific pathology.¹⁵ Several authors, e.g., Hopkins et al., have shown that a negative PTF value of 0.04 mm $\times$ s or more performs best (with a sensitivity of 68.3%) at identifying patients with left atrial enlargement.²² Surprisingly, only

eight individuals met the criterion for abnormal, negative PTF in the present study population, where 79% had enlarged left atria. Importantly, Hopkins et al. used a much stricter definition of atrial enlargement (left atrial diameter/BSA > 2.2 cm/m²). Applying this definition to our material, only eight subjects had enlarged left atria, and none of them had an abnormal, negative PTF (data not shown). As in the present study, the study population analyzed by Hopkins et al. was all male, but in contrast with our study, they were substantially older, with a mean age of 52 (range 21–88) years. This may be, at least part of, the explanation.

There was no correlation between left atrial end-systolic volume and log-transformed negative PTF. In addition, there was no significant difference in median left atrial diameter and median left atrial end-systolic volume between individuals with normal and abnormal PTF. These findings vividly illustrate how uncertain PTF is at identifying pure left atrial enlargement, at least in this population of healthy athletes with no known atrial conduction difficulties. Interestingly, in a study by Forfang et al. on healthy middle-aged men, it was shown that 7.1% at rest and as many as 25.4% after exercise actually had an abnormal PTF.²³

Finally, we could not show any difference in median P-wave duration between individuals with a normal and an abnormal, negative PTF. Neither was there a significant correlation between the degree of negative PTF and P-wave duration. A prolonged P-wave duration has previously been shown to be associated with an abnormal PTF, which is reasonable if an abnormal PTF is an expression of atrial conduction difficulties.²⁴ The reason why we were unable to reproduce this finding was probably that we only had eight subjects with an abnormal PTF. The eight individuals with an abnormal PTF all had type 2 P-wave morphology (data not shown), which is reasonable since this morphology is characterized by a positive terminal deflection in lead Z (directed away from lead V₁, i.e., a negative deflection in this lead).

P-Wave Duration

It has been reported that a total P-wave duration of more than 110 ms in any standard lead provides a sensitivity of 33–62% and a specificity of 86–88% for left atrial enlargement.^{25,26} Truong et al. made

a cardiac computed tomography evaluation of six ECG criteria for left atrial enlargement. Only P-wave duration of more than 110 ms turned out to be independently associated with CT-defined left atrial enlargement.²⁷ We only had 18 persons with a P-wave duration of less than or equal to 110 ms, and half of them actually belonged to the quartile with the largest left atrial volume (data not shown). Hence, we were not able to reproduce those findings. It should be noted that our population differed in many aspects to that studied by Truong et al., such as lower age, men only and no concomitant diseases. Another important aspect is the method used for estimating P-wave duration. The median P-wave duration in the present study was prolonged at 132 ms. It is known that using the method for delineating P-waves applied in the present study (namely including magnification, artifact filtering, and signal averaging) tends to render relatively longer P-wave durations.⁴ The fact that we manually delineate the earliest onset and latest end of the P-wave in any of the three orthogonal leads may also contribute to longer P-wave durations.

Hohnloser et al. investigated potential correlations between P-wave parameters and echocardiographic findings. Only age was shown to be independently and significantly correlated to signal-averaged P-wave duration. In univariate analysis, left atrial and left ventricular end-diastolic diameters also correlated with P-wave duration.²⁸ We found a positive correlation between left atrial end-systolic volume and P-wave duration as well as PQ time, which is in keeping with a previous study.²⁹

We observed a significantly longer P-wave duration in the group with type 2 P-wave morphology than in the type 1 group. However, the two groups overlapped extensively and the relevance of this finding is questionable. It may however reflect different atrial activation routes. The shorter P-wave duration observed in the few cases with atypical morphology may be due to difficulties in delineating the true onset and end of the P-wave because of its atypical appearance.

CONCLUSION

The findings in the present study strengthen the view that P-wave morphology is indeed primarily an expression of interatrial conduction pathways

and not dependent on the size of the atria, at least in young, healthy athletes. We also showed that PTF is not reliable at diagnosing left atrial enlargement in the current population with no known atrial conduction difficulties.

Acknowledgments: *We would like to thank Erik Ekker Solberg, M.D., Ph.D., at Diakonhjemmet Hospital in Oslo, and Thor Einar Andersen, M.D., Ph.D., at Oslo Sports Trauma Research Center (OSTRC) in Oslo, who initiated the preparticipation cardiac screening and monitored the data gathering, and engineer Britt-Marie Gunnarsson at Skåne University Hospital in Lund for valuable technical assistance. Associate Professor Jonas Björk at Skåne University Hospital in Lund deserves special mentioning for vital statistical input.*

REFERENCES

- Holmqvist F, Husser D, Tapanainen J, 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.
- Platonov PG, Christensen AH, Holmqvist F, et al. Abnormal atrial activation is common in patients with arrhythmogenic right ventricular cardiomyopathy. *J Electrocardiol* 2011;44:237–241.
- Holmqvist F, Platonov Pyotr G, McNitt S, et al. Abnormal P-wave morphology is a predictor of atrial fibrillation development and cardiac death in MADIT II patients. *Ann Noninvasive Electrocardiol* 2010;15:63–72.
- Holmqvist F, Platonov PG, Havmøller R, et al. Signal-averaged P wave analysis for delineation of interatrial conduction: Further validation of the method. *BMC Cardiovasc Disord* 2007;7:29.
- Holmqvist F, Platonov Pyotr G, Carlson J, et al. Variable interatrial conduction illustrated in a hypertrophic cardiomyopathy population. *Ann Noninvasive Electrocardiol* 2007;12:227–236.
- Pelliccia A, Maron BJ, Di Paolo FM, et al. Prevalence and clinical significance of left atrial remodeling in competitive athletes. *J Am Coll Cardiol* 2005;46:690–696.
- D'Ascenzi F, Cameli M, Zacà V, et al. Supernormal diastolic function and role of left atrial myocardial deformation analysis by 2D speckle tracking echocardiography in elite soccer players. *Echocardiography* 2011;28:320–326.
- Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: A report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr* 2005;18:1440–1463.
- Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: A report from the American Society of Echocardiography: Endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010;23:685–713.
- DuBois D, DuBois E. A formula to estimate the approximate surface area if height and weight be known. *Arch Int Med* 1916;17:863–71.
- Mosteller R. Simplified calculation of body-surface area. *N Engl J Med* 1987;3(17):1098.
- Carlson J, Havmøller R, Herreros A, et al. Can orthogonal lead indicators of propensity to atrial fibrillation be accurately assessed from the 12-lead ECG? *Europace* 2005;7:S39–S48.
- Carlson J. Exploration of supraventricular conduction with respect to atrial fibrillation. Methodological aspects on selected techniques. Lund University, Faculty of Medicine Doctoral Dissertation Series 2005:106, KFS i Lund AB, Lund, Sweden, 2005. Available at: lup.lub.lu.se/record/545700/file/545701.pdf [Accessed January 25, 2014].
- Morris JJ, Estes EH, Whalen RE, et al. P-wave analysis in valvular heart disease. *Circulation* 1964;29:242–252.
- Bonow RO, Mann DL, Zipes DP, et al. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 9th ed. Philadelphia: Elsevier, 2012, pp. 137–138.
- Soliman EZ, Alonso A, Misialek JR, et al. Reference ranges of PR duration and P-wave indices in individuals free of cardiovascular disease: The Multi-Ethnic Study of Atherosclerosis (MESA). *J Electrocardiol* 2013;46:702–706.
- Nasir JM, Rubal BJ, Jones SO, et al. The effects of body mass index on surface electrocardiograms in young adults. *J Electrocardiol* 2012;45:646–651.
- Havmøller R, Carlson J, Holmqvist F, et al. Age-related changes in P wave morphology in healthy subjects. *BMC Cardiovasc Disord* 2007;7:22.
- 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.
- Boineau JP, Canavan TE, Schuessler RB, et al. Demonstration of a widely distributed atrial pacemaker complex in the human heart. *Circulation* 1988;77:1221–1237.
- Holmqvist F, Platonov PG, Solomon SD, et al. P-wave morphology is associated with echocardiographic response to cardiac resynchronization therapy in MADIT-CRT patients. *Ann Noninvasive Electrocardiol* 2013;18:510–518.
- Hopkins CB, Barrett Jr ON. Electrocardiographic diagnosis of left atrial enlargement: Role of the P terminal force in lead V1. *J Electrocardiol* 1989;22:359–363.
- Forfang K, Erikssen J. Significance of P wave terminal force in presumably healthy middle-aged men. *Am Heart J* 1978;96:739–743.
- Spodick DH, Ariyaratnam V, Goldberg R. Interatrial block: Correlation with P-terminal force. *Clin Cardiol* 2009;32:181–182.
- Munuswamy K, Alpert MA, Martin RH, Whiting RB, Mechlin NJ. Sensitivity and specificity of commonly used electrocardiographic criteria for left atrial enlargement determined by m-mode echocardiography. *Am J Cardiol* 1984;53:829–832.
- Hazen MS, Marwick TH, Underwood DA. Diagnostic accuracy of the resting electrocardiogram in detection and estimation of left atrial enlargement: An echocardiographic correlation in 551 patients. *Am Heart J* 1991;122:823–828.
- Truong QA, Charipar EM, Ptaszek LM, et al. Usefulness of electrocardiographic parameters as compared with computed tomography measures of left atrial volume enlargement: From the ROMICAT trial. *J Electrocardiol* 2011;44:257–264.
- Hohnloser SH, Ehrlich JR, Steul K, et al. Relationship between clinical and echocardiography-derived parameters and atrial activation as measured by the P-wave signal-averaged electrocardiogram. *Z Kardiol* 2002;91:404–409.
- Birkbeck JP, Wilson DB, Hall MA, et al. P-wave morphology correlation with left atrial volumes assessed by 2-dimensional echocardiography. *J Electrocardiol* 2006;39:225–229.