Association of P-Wave Duration, Dispersion, and Terminal Force in Relation to P-Wave Axis among Outpatients

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Background: While P-wave duration (P-dur) and dispersion (P-disp) could both reflect fractionated and inhomogeneous propagation of sinus cardiac impulses, and may therefore be associated with each other, a clear relationship has not been extensively studied. We studied these markers as well as the significance of P-wave terminal force in lead V1 (PTFV1) in relation to the P-wave axis (P-axis).

Methods: We appraised our previously studied sample of 500 consecutively numbered, otherwise unselected, electrocardiograms (ECGs) of outpatients from the University of Massachusetts, Worcester, Massachusetts, for the foregoing P-wave characteristics. P-disp, defined as the difference of the duration between the widest and narrowest P wave, and the greatest P-dur after a 12-lead ECG search, was measured manually to the nearest 10 ms. PTFV1 was considered positive when ≥ 40 mm² terminal deflection was present on biphasic P waves on lead V₁. Normal P-axis was considered 0° to +75° by manually constructing the mean frontal plane electrical P-axis from standard limb leads.

Results: After excluding those with atrial arrhythmias, paced rhythms, errors in lead placement, P waves with low amplitude or overall technically poor tracing, 428 ECGs formed our final sample. P-dur was strongly associated with P-disp ($P < 0.0001$), but the correlation remained weak ($r = 0.42$). Overall, P-dur was not significantly associated with P-axis but when divided into tertiles and quintiles, the significance was evident within the range of the normal P-axis, particularly 0° to +60° ($P < 0.0001$). In a subanalysis of 380 ECGs that had appreciable biphasic P waves on lead V₁, PTFV1 was noted on 178 (47%) ECGs and was significantly associated with P-dur ($P < 0.0001$), P-disp ($P < 0.0001$), and P-axis ($P = 0.002$). When considering P-axis in tertiles and quintiles, P-dur was greater in patients with a positive PTFV1 and significant within the normal range of the P-axis, especially from 0° to +60°.

Conclusion: P-dur, P-disp, and PTFV1 appear to share a significant tripartite association in relation to the normal P-axis, particularly when P-axis ranges 0° to +60°. Therefore, for optimal clinical assessment, these markers should be evaluated in relation to the normal P-axis.

A.N.E. 2007;12(3):210–215

P-wave duration; P-wave dispersion; P-wave axis; P-terminal force-V1; P-wave axis

There has been increasing interest in the importance of abnormal P waves on the electrocardiogram (ECG) owing largely to the known deleterious clinical associations, particularly with that of atrial tachyarrhythmias.^{1–3} P-wave duration (P-dur) ≥ 110 ms, that is, interatrial block or delay, is exceptionally prevalent ($>40\%$) in general hospital populations^{4,5} and has been consistently shown to be an important noninvasive clinical correlate

of left atrial dilatation⁶ and its electromechanical dysfunction,⁷ myocardial ischemia,⁸ and embolic stroke.⁹ A separate entity, P-wave dispersion (P-disp), which denotes the greatest interlead variability or difference in P-dur, has also been frequently reported with such widened P waves in relation to similar, often comparable conditions.^{10–12} While both electrocardiographic markers could perhaps reflect fractionated and inhomogeneous

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propagation of sinus cardiac impulses,¹³ and may therefore be associated with each other, a clear relationship has not been extensively studied, most important, with reference to the axis of the overall atrial depolarization force. We also investigated the significance of abnormal terminal P-wave negative deflection in lead V₁ (PTFV1), which commonly typifies left atrial abnormality¹⁴ with the foregoing markers to determine if a tripartite association does indeed exist in relation to the P-wave axis (P-axis).

METHODS

The design of our previously published series in which we had investigated 500 consecutively numbered, otherwise unselected, ECGs of outpatients from the University of Massachusetts, Worcester, Massachusetts, has been previously described.¹⁵ In brief, resting 12-lead ECGs were recorded with the patient in the supine position on a Marquette 2000 electrocardiograph (Marquette Electronics Incorporated, Milwaukee, WI) using 25 mm/s and 10 mm/mV standardization. ECGs had been independently evaluated for IAB to the nearest 10 ms using the greatest P-dur in every appropriate lead as measured on a blinded, single-read with a calibrated magnifying graticule by two investigators (M.E.F. and D.H.S.; concordance >95%). P-disp was defined as the difference of the measured duration between the widest and narrowest P wave, and was similarly reported to the nearest 10 ms. The onset of the P wave was defined as the junction between the isoelectric T-P baseline and the beginning of the P deflection while the terminal point, as the junction between the end of the P deflection and the PR segment. PTFV1 was considered positive when ≥ 40 mm² terminal deflection was present on biphasic P waves on lead V₁. The mean frontal plane electrical P-axis was manually constructed from standard limb leads using the triaxial reference frame figure derived from Einthoven's equilateral triangle to de-

termine the orientation of the mean atrial depolarization vector.¹ Positive or negative P-wave voltage in any lead was measured and projected in either mV or mm on the corresponding side of the ECG lead. Normal P-axis was considered 0° to +75° for this investigation and axes were divided into tertiles and quintiles for respective analysis.¹

Data are expressed as mean $\pm$ standard deviation for continuous variables and frequencies for categorical variables. Differences between groups were assessed using

Chi-square statistics for categorical variables and analysis of variance for continuous variables. A P value <0.05 was considered significant. Logistic multivariate regression analysis using significant variables was also performed. Statistical analyses were performed using SPSS version 13.0 statistical software (SPSS Inc., Chicago, IL).

RESULTS

Four hundred twenty-eight patients formed our sample after excluding those with atrial arrhythmias, paced rhythms, errors in lead placement, P waves with low amplitude, or overall technically poor tracing. Ranges for measurements were as follows: 40–170 ms (mean $\pm$ standard deviation, 109.95 $\pm$ 15 ms; mode 120 ms) for P-dur, 10–120 ms (mean $\pm$ standard deviation, 45.5 $\pm$ 19 ms; mode 40 ms) for P-disp, and –30° to 95° (mean $\pm$ standard deviation, 48.55° $\pm$ 21°; mode 60°) for P-axis (Tables 1 and 2). P-dur was strongly associated with P-disp (P < 0.0001) but the correlation, though direct, remained weak (r = 0.42). Overall, P-dur was not significantly associated with P-axis but when divided into tertiles and quintiles, the significance was evident within the range of the normal P-axis, particularly 0° to +60° (P < 0.0001).

From the sample, only 380 ECGs that had appreciable biphasic P waves on lead V₁ and were without motion artifact were further assessed for PTFV1. PTFV1 was present in 178 (47%) patients

Table 1. Baseline Investigated Electrocardiographic Variables of Outpatient Sample (n = 428)

Variable	Range	Mean $\pm$ Standard Deviation	Median	Mode
P-wave duration (ms)	40 to 170	109.95 $\pm$ 15	110	120
P-wave dispersion (ms)	10 to 120	45.5 $\pm$ 19	40	40
P-wave axis (degrees)	–30 to 95	48.55 $\pm$ 21	55	60

*P-wave terminal force (presence or absence) evaluated in 380 patients.

Table 2. Association between Continuous Variables (n = 428)

Variable	P-Wave Duration (ms)	P-Wave Dispersion (ms)	P-Wave Axis (Degrees)
P-wave duration (ms)	N/A	r = 0.42; P < 0.0001	r = -0.09; P = 0.06
P-wave dispersion (ms)	r = 0.42; P < 0.0001	N/A	r = 0.04; P = 0.47
P-wave axis (degrees)	r = -0.09; P = 0.06	r = 0.04; P = 0.47	N/A

and was significantly associated with P-dur (P < 0.0001), P-disp (P < 0.0001), and P-axis (P = 0.002) (Table 3). When considering P-axis in tertiles and quintiles, P-dur was greater in patients with a positive PTFV1 and significant within the normal range of the P-axis, especially when 0° to +60° (Tables 4 and 5). Wider P-disp was similarly associated with positive PTFV1 (Table 6).

DISCUSSION

Prolonged P-dur denotes abnormal conduction between the right and left atria, and is associated with a host of sequelae.^{3,6-9} Atrial function and as a result, the P waves depicted on the ECG are influenced by the autonomic nervous system that produces alterations in atrial size, contraction, heart rate and homogeneity, as well as velocity of sinus impulse propagation. With abnormal conduction such as that in patients with paroxysmal atrial fibrillation (AF), this autonomic control is lost or dampened, resulting in fractionated impulses and has been associated with chemoreflex sensitivity.¹⁶ Moreover, as a sum of several factors including the different intrinsic properties of the interatrial conduction system, the P-dur in different ECG leads may vary, allowing for an increased chance for greater dispersion.

Clinically, increased P-dur and P-disp have been demonstrated in cardiac and noncardiac disease as well as in health. Dogan et al.¹⁰ reported that maximum P-dur and P-disp were predictors for the maintenance of sinus rhythm after cardioversion of AF and that increased P-disp could be used to identify patients at risk of such postprocedural recur-

rence. P-dur of >125 ms and P-disp >25 ms, when measured very early after an acute myocardial infarction, were observed to be independently associated with AF.¹¹ Less surprising therefore was that a higher prevalence of P-disp can be observed among patients with stable coronary artery disease, dilated and hypertrophic cardiomyopathies, and diastolic dysfunction of any cause and severity.¹⁷⁻¹⁹ Aras et al.²⁰ reported that the high risk for the development of AF could be identified by simply measuring maximum P-dur and P-disp values in patients with clinical and subclinical hyperthyroidism. In renal failure patients, hemodialysis ends with significant increase in maximum P-dur and P-disp, which might be responsible for the increased occurrence of AF in these groups of patients.²¹ In two separate investigations, Uyarel et al. found that anxiety in otherwise healthy individuals (P < 0.001)²² and moderate dose of alcohol intake over a short period (P = 0.027)²³ was associated with increased P-dur and P-disp. Despite these clinical associations, however, the true mechanism of P-disp has yet to be precisely defined.

A positive PTFV1 along with prolonged P-dur portends left atrial abnormality, particularly in patients with underlying cardiovascular diseases (sensitivity, 82%; specificity, 40%; positive predictive value, 70%; and negative predictive value, 55%) and its presence indicates the need for further evaluation.¹⁴ Moreover, a positive PTFV1 has been independently associated with ischemic stroke after adjustment for other stroke risk factors (odds ratio, 2.32; 95% confidence interval, 1.29-4.18).²⁴ The higher prevalence of PTFV1 ≥40 mm² in patients with prolonged P-dur and wider P-disp as noted

Table 3. Association between Continuous Variables and P-Wave Terminal Force (n = 380)

Variable	P-Wave Terminal Force		P Value
	Present (n = 178)	Absent (n = 202)	
P-wave duration (mean ± standard deviation; ms)	115.1 ± 14	107.1 ± 14	<0.0001
P-wave dispersion (mean ± standard deviation; ms)	50.3 ± 19	40.9 ± 20	<0.0001
P-wave axis (mean ± standard deviation; degrees)	51.4 ± 20	44.5 ± 22	0.002

Table 4. Correlation between P-Wave Duration and P-Wave Dispersion with Different P-Wave Axes* (n = 428)

Dividing P-Wave Axis into Tertiles (<0°, 0° to +75°, and > +75°)				
P-Wave Axis (Degrees)	P-Wave Duration (Mean ± Standard Deviation; ms)	P-Wave Dispersion (Mean ± Standard Deviation; ms)	R Value	P Value
<0 (n = 6)	100 ± 15	28.3 ± 15	0.702	0.12
0-75 (n = 400)	110.1 ± 15	45.3 ± 20	0.442	<0.0001
>75 (n = 22)	109.6 ± 16	48.5 ± 20	-0.009	0.97
Dividing P-Wave Axis into Quintiles (<0°, 0° to +30°, 31° to +60°, 61° to +90°, and > +90°)				
P-Wave axis (Degrees)	P-Wave Duration (Mean ± Standard Deviation; ms)	P-Wave Dispersion (Mean ± Standard Deviation; ms)	R Value	P Value
<0 (n = 6)	100 ± 15	28.3 ± 15	0.702	0.12
0-30 (n = 94)	112.7 ± 19	46.6 ± 21	0.566	<0.0001
31-60 (n = 233)	109.9 ± 13	44.2 ± 20	0.431	<0.0001
61-90 (n = 93)	107.9 ± 14	47.6 ± 19	0.187	0.07
>90 (n = 2)	110 ± 14	50 ± 42	1	N/A

*Normal P-Wave axis was considered 0° to +75° in our study.

in our study probably reflects the associated delayed impulse conduction through an already abnormal and enlarged left atria. Indeed, a positive PTFV1 could be a more common finding in the presence of an extremely leftward P-axis, parallel to the net direction of electrical forces of the heart. What is, however, most clinically relevant is that within the normal range of the frontal plane P-axis, where the major vectorial forces are not in the direction of such PTFV1, its presence becomes

increasingly significant and may be more indicative of left atrial abnormality. Therefore, similar to that of P-dur and P-disp, assessment of a positive PTFV1 cannot be determined in isolation and would certainly be optimally served when P-axis is taken into account. Moreover, increased P-dur and P-disp as well as a positive PTFV1 within the limits of a normal P-axis range may be in fact pose as stronger clinical predictors of the already known deleterious sequelae, mainly atrial fibrillation, left

Table 5. Association between P-Wave Terminal Force and P-Wave Duration with Different P-Wave Axes* (n = 380)

Dividing P-Wave Axis into Tertiles (<0°, 0° to +75°, and > +75°)			
P-Wave Axis (Mean ± Standard Deviation; Degrees)	P-Wave Duration (Mean ± Standard Deviation; ms)		P Value
	P-Wave Terminal Force Present (n = 178)	P-Wave Terminal Force Absent (n = 202)	
<0 (n = 6)	110 ± 28 (n = 2)	95 ± 6 (n = 4)	0.31
0-75 (n = 355)	115.6 ± 14 (n = 164)	107.2 ± 14 (n = 191)	<0.0001
>75 (n = 19)	108.3 ± 13 (n = 12)	110 ± 24 (n = 7)	0.85
Dividing P-Wave Axis into Quintiles (<0°, 0° to +30°, 31° to +60°, 61° to +90°, and > +90°)			
P-Wave Axis (mean ± Standard Deviation; Degrees)	P-Wave Duration (Mean ± Standard Deviation; ms)		P Value
	With P-Wave Terminal Force Present (n = 178)	Without P-Wave Terminal Force Absent (n = 202)	
<0 (n = 6)	110 ± 28 (n = 2)	95 ± 6 (n = 4)	0.31
0-30 (n = 83)	126.2 ± 19 (n = 29)	106.5 ± 15 (n = 54)	<0.0001
31-60 (n = 212)	114.2 ± 11 (n = 102)	106.8 ± 14 (n = 110)	<0.0001
61-90 (n = 79)	110 ± 13 (n = 45)	110.3 ± 14 (n = 34)	0.93
>90 (n = 0)	N/A	N/A	N/A

*Normal P-Wave axis was considered 0° to +75° in our study.

Table 6. Association between P-Wave Terminal Force and P-Wave Dispersion with Different P-Wave Axes* (n = 380)

Dividing P-Wave Axis into Three Groups (<0°, 0°–75°, and >75°)			
P-Wave Axis (Mean ± Standard Deviation; Degrees)	P-Wave Dispersion (Mean ± Standard Deviation; ms)		P Value
	With P-Wave Terminal Force Present (n = 178)	Without P-Wave Terminal Force Absent (n = 202)	
<0 (n = 6)	45 ± 7 (n = 2)	20 ± 8 (n = 4)	0.02
0–75 (n = 355)	50.5 ± 20 (n = 164)	41.2 ± 19 (n = 191)	<0.0001
>75 (n = 19)	49.2 ± 12 (n = 12)	45.3 ± 29 (n = 7)	0.69
Dividing P-Wave Axis into Five Groups (<0, 0°–30°, 31°–60, 61°–90°, and >90°)			
P-Wave Axis (Mean ± Standard Deviation; Degrees)	P-Wave dispersion (mean ± standard deviation; ms)		P Value
	With P-Wave Terminal Force Present (n = 178)	Without P-Wave Terminal Force Absent (n = 202)	
<0 (n = 6)	45 ± 7 (n = 2)	20 ± 8 (n = 4)	0.02
0–30 (n = 83)	58.6 ± 22 (n = 29)	39.8 ± 18 (n = 54)	<0.0001
31–60 (n = 212)	48.5 ± 20 (n = 102)	40.7 ± 19 (n = 110)	0.004
61–90 (n = 79)	49.3 ± 15 (n = 45)	45.7 ± 25 (n = 34)	0.41
>90 (n = 0)	N/A	N/A	N/A

*Normal P-Wave axis was considered 0° to +75° in our study.

atrial electromechanical dysfunction, and embolic stroke. However, though arguably intuitive, further studies are warranted to conclusively prove a clinical association in this particular context.

LIMITATIONS

P-disp may be misleading in the patients with uniformly wide P waves in all leads. Electrophysiologic studies are needed to shed further insight on the precise association between P-dur and P-disp, especially in such circumstances. We did not include heart rate as a variable in this present study. Nevertheless, our sample consisted of stable outpatients who could be considered "less sick" compared to inpatients where extremes of heart rates may become an issue. Moreover, because change in heart rate could potentially affect P-dur, P-disp, which we have shown in this present assessment to be significantly and directly correlated, may perhaps be affected proportionately. We also did not evaluate the effects of medications and comorbidities on our findings at this time, and these need to be considered in future studies.

CONCLUSION

P-dur, P-disp, and PTFV1 appear to share a significant tripartite association in relation to thenormal P-axis, particularly when P-axis ranges 0° to

+60°. These findings parallel previous hypotheses and studies that had clinically appraised some, if not all of these ECG markers. As such, for optimal assessment, these markers should be evaluated in relation to the normal P-axis, where their presence within a normal P-axis range could perhaps be a clinically stronger predictor of already known deleterious consequences, mainly atrial fibrillation, left atrial electromechanical dysfunction, and embolic stroke.

DISCLOSURE

None of the authors received any funding for this investigation. Any affiliations or financial involvement, within the past 5 years and foreseeable future with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript are completely disclosed. Dr. Spodick receives research support from the University of Massachusetts, Worcester, Massachusetts.

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