The Prognostic Importance of Isolated P-Wave Abnormalities

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ABSTRACT

Background: While certain P-wave morphologies have been associated with abnormal atrial size and either pulmonary or cardiovascular (CV) disease, their relationship to mortality and specific cause of death has not been reported.

Methods: Analyses were performed on the first digitally recorded electrocardiogram (ECG) on 43 903 patients at the Palo Alto Veterans Administration Medical Center since 1987. After appropriate exclusions, 40 020 patients remained. Using computerized algorithms, P-wave amplitude and duration in 12 leads as well as several standardized ECG interpretations were extracted. The main outcome measures were pulmonary and CV mortality.

Results: During a mean follow-up of 6 years there were 3417 CV and 1213 pulmonary deaths. After adjusting for age and heart rate in a Cox regression model, P-wave amplitude in the inferior leads was the strongest predictor of pulmonary death (hazard ratio [HR]: 3.0, 95% confidence interval [CI]: 2.3–3.9, $P < .0001$ for an amplitude >2.5 mm), outperforming all other ECG criteria. The depth of P-wave inversion in leads V_1 or V_2 and P-wave duration were strong predictors of CV death (HR: 1.7, 95% CI: 1.5–2.0, $P < .0001$ for a P-wave inversion deeper than 1 mm), outperforming many previously established ECG predictors of CV death.

Conclusions: P-wave amplitude in the inferior leads is the strongest independent predictor of pulmonary death while P-wave duration and the depth of P-wave inversion in leads V_1 or V_2 significantly predict CV death. These measurements can be obtained easily and should be considered as part of clinical risk stratification.

Introduction

P-wave abnormalities on the resting electrocardiogram (ECG) have been associated with abnormal atrial size and either pulmonary or cardiovascular (CV) disease. Abnormalities of P-wave morphology have a limited sensitivity for echocardiographic findings[1–6] and have been associated with left sided heart disease[7–10] or severe lung disease.[11–14] The only prognostic studies of P-wave abnormalities have been in selected populations after acute myocardial infarction[15] or patients with lung disease.[12,16,17] P-wave changes[18] before and after treatment of chronic obstructive pulmonary disease,[19] status asthmaticus,[20] and congestive heart failure[21] have been described as well as with increasing severity of lung disease[22] and exercise.[23] However, P-wave abnormalities are usually considered to be nonspecific, labile, and not to have prognostic value. Since the prognostic value of P-wave abnormalities has not been determined in a large general medical population, we felt it worthwhile to do so at a major medical center where simple P-wave measurements using computerized 12 lead ECGs were available.

Methods

All ECGs obtained at the Palo Alto Veterans Administration Medical Center between March 1987 and December 2000 were digitally recorded and stored in the MUSE-General Electric ECG management system. Computerized measurements from the ECG as well as several standardized, computerized ECG interpretations were extracted. ECGs exhibiting electronic pacing (n = 309), Wolff-Parkinson-White pattern (n = 44), atrial fibrillation (n = 1341), and those in which the computer algorithm failed to detect the P wave (n = 2189) were excluded from the analyses.

Using computerized algorithms, P-wave amplitude (in microvolt [μ V]) and duration (in millisecond [ms]) in 12 leads were measured. P-wave duration was defined as the longest P-wave duration in any lead. P-wave amplitude in inferior leads was defined as the tallest P wave in leads II, III, or aVF. Depth of P-wave inversion in leads V_1 or V_2 was defined as the maximum deviation of P terminal phase in leads V_1 or V_2 below the PR isoelectric baseline. P terminal force in lead V_1 was defined as amplitude area of the terminal phase of the P wave (the negative component when the P wave was biphasic) in lead V_1 (in mm $\times$ ms). Left ventricular hypertrophy (LVH) was defined as the Romhilt-Estes point score of 4 or greater. In our population, the Romhilt score has outperformed all other LVH criteria including all of those proposed by the Cornell group.[24]

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Standardized computerized ECG criteria as described by the General Electric 12 lead ECG analysis program were utilized for right atrial abnormality, left atrial abnormality (LAA), right axis deviation, left axis deviation, right ventricular hypertrophy, left bundle branch block, right bundle branch block, and intraventricular conduction delay (see MUSE-General Electric/12 lead ECG physician program manual at www.gemedicalsystems.com for further information on the definitions). For instance, LAA was defined in the MUSE ECG system as: absolute, if P-wave amplitude was $< -200\,\mu$ V in leads V_1 or V_2; possible if $< -100\,\mu$ V and P-wave duration was $\geqslant 60$ ms and P terminal force in lead V_1 was $\geqslant 4000\,\mu$ V $\times$ ms. Left atrial abnormality was pronounced if absolute or possible criteria were met.

Both the Social Security Death Index and the California Health Department Service were used to ascertain the vital status of each patient as of December 31, 2000. The main outcome measures were CV and pulmonary mortality. Cardiovascular and pulmonary deaths were defined as per International Statistical Classification of Diseases and Related Health Problems (ICD-9) codes.

Statistical Analysis

The database was imported into NCSS (Number Cruncher Statistical System, Kaysville, UT) software for analysis. Descriptive statistics were used to determine mean values for continuous variables and to test for normality. Bivariate associations between those who died from CV or pulmonary death were tested using χ^2 tests for categorical data and t tests for continuous variables. P values less than. 05 were considered significant.

Cox proportional hazards regression analyses were performed to assess the significance and independence of predictors of outcomes. Analyses were statistically adjusted for potential confounding effects of age and heart rate. This was performed by adding age and heart rate as continuous variables to the Cox regression models. Including the potential confounders and important prognostic variables in the model will allow one to see if the tested variable is independently predictive. For instance, if P-wave abnormalities are more likely due to age and only predictive because those with P-wave abnormalities are older, then with age adjustment, the P-wave abnormality will not be significantly predictive in the model any longer. P-wave measurements as well as other established ECG indicators were considered in the model. Multivariate analyses were repeated for both continuous and dichotomous variables using conventional cut points for P-wave duration (above and below 120 ms), P-wave amplitude (above and below $250\,\mu$ V), and the depth of P-wave inversion in leads V_1 or V_2 (above and below $-100\,\mu$ V). A subset analysis was repeated in outpatients in order to exclude ECGs possibly associated with acute clinical events (eg, acute pulmonary embolism and acute myocardial infarction).

Kaplan-Meier survival curves were performed to display impact on survival, stratifying P-wave amplitude, P-wave duration, and the depth of P-wave inversion in leads V_1 or V_2. For practical clinical utility, these variables were classified as scores for survival plots: the P-wave amplitude score was defined as P-wave amplitude in inferior leads (μ V) of $\leqslant 200 = 1$; $201–250 = 2$; $251–300 = 3$; and $>300 = 4$; the P-wave duration score was defined as P-wave duration (ms) of $\leqslant 120 = 1$; $121–130 = 2$; $131–140 = 3$; and $>140 = 4$; the P-wave inversion score was defined as the depth of P-wave inversion in leads V_1 or V_2 (μ V) of $\geqslant -50 = 1$; -51 to $-100 = 2$; -101 to $-150 = 3$; and $< -150 = 4$; and annual CV and pulmonary mortality rates were calculated. The log-rank test was used to test the differences between strata of each predictor.

Results

Baseline Demographics and ECG Findings

Demographics and ECG findings of the total study cohort (40 020 males, mean age of 56 ± 14 y) classified by CV and pulmonary death status comparing those who died from CV vs pulmonary causes are shown in Table 1. During a mean follow-up of 6.1 ± 3.8 years, there were 3417 CV (annual mortality rate = 1.3%) and 1213 pulmonary deaths (annual mortality rate = 0.6%). Those who died from CV causes were older, had a longer P-wave duration, shorter P-wave amplitude in inferior leads, larger P terminal force in lead V_1, and a deeper P-wave inversion in leads V_1 or V_2 compared to those who died from pulmonary causes.

Cox Analysis

Analysis of variables adjusted for age and heart rate is shown in Table 2. P-wave duration was a significant predictor of both CV and pulmonary death, being directly associated with CV death but inversely associated with pulmonary death. Subjects with a P-wave duration >120 ms had a 45% greater risk of CV death compared to subjects with a P-wave duration $\leqslant$120 ms. Each increment in the P-wave duration score was associated with a 25% increase in CV death. P-wave amplitude in the inferior leads was also a significant predictor of both CV and pulmonary death; being directly associated with pulmonary death but inversely associated with CV death. Subjects with P-wave amplitude $>250\,\mu$ V in the inferior leads had 3.2 times higher risk for pulmonary deaths compared to the subjects with P-wave amplitude $\leqslant 250\,\mu$ V. Each increment in the P-wave amplitude score was associated with a 70% increase in pulmonary death. P-wave inversion in leads V_1 or V_2 deeper than $-100\,\mu$ V was associated with 2.6 and 1.4 times more risk for CV and pulmonary death, respectively. Each increment in the P-wave inversion score was associated with a 56% and 17% increase in CV and pulmonary death, respectively. P terminal force in lead $V_1 > 40$ mm $\times$ ms was a significant

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Table 1. Demographics and ECG Findings of the Total Study Cohort Classified as to Death Status for Comparisons Between Those Who Died From Cardiac vs Pulmonary Causes of Death

Characteristic	Total	CV Death	Pulmonary Death	*P* Value
Number of subjects	40020	3417	1213	. . .
Age (y)	56.2 ± 14.2	66.9 ± 11.2	65.9 ± 9.5	.007
Body mass index (kg/m²)	27.3 ± 5.4	26.8 ± 5.2	24.5 ± 5.0	<.001
Outpatient status	25407 (63.5%)	1756 (51.4%)	526 (43.4%)	<.001
Heart rate (beats per minute)	73.8 ± 15.8	76.8 ± 16.4	83.6 ± 18.9	<.001
P-wave duration (ms)	106.8 ± 14.4	110.3 ± 17.5	104.9 ± 16.5	<.001
P-wave duration >120 ms	4905 (12.3%)	763 (22.3%)	145 (11.9%)	<.001
P terminal force V_1 >40 (mm × ms)	5326 (13.3%)	798 (23.4%)	236 (19.5%)	.005
P-wave amplitude inferior leads (μ V)	117.8 ± 46.3	115.6 ± 53.0	139.5 ± 64.5	<.001
P-wave amplitude inferior leads >250μ V	401 (1.0%)	56 (1.6%)	67 (5.5%)	<.001
Depth of P-wave inversion in leads V^1 or V_2 (μ V)	−40.3 ± 31.2	−54.1 ± 38.6	−50.6 ± 35.0	.006
Depth of P-wave inversion in leads V^1 or V_2 < − 100 μ V	1536 (3.8%)	354 (10.4%)	96 (7.9%)	.01
Left atrial abnormality	1685 (4.2%)	389 (11.4%)	119 (9.8%)	.01
Right atrial abnormality	420 (1%)	58 (1.7%)	69 (5.7%)	<.001

predictor of CV death, but not a predictor of pulmonary death.

Multivariate Comparisons With Other ECG Measurements

All of the significant variables in the univariate analysis were considered for multivariate Cox regression analysis and the results (adjusted for age, heart rate, and body mass index) are shown in Table 3. P-wave amplitude in the inferior leads was the strongest predictor of pulmonary death (hazard ratio [HR]: 3.0, 95% confidence interval [CI]: 2.3–3.9, $P < .0001$ for an amplitude >2.5 mm], outperforming all other ECG findings. Depth of P-wave inversion in leads V_1 or V_2 was the strongest predictor of CV death (HR: 1.7, 95% CI: 1.5–2.0, $P < .0001$; for a P wave inversion deeper than 1 mm), outperforming other P-wave variables as well as significant ECG predictors of CV death such as QRS duration, Q-wave myocardial infarction, QT interval, LVH, and ST-depression. The depth of P-wave inversion in leads V_1 or V_2 was only exceeded as a predictor of CV death by T-wave abnormalities. These multivariate analyses were repeated separately in both the inpatient and outpatient populations with the same or similar results. This finding strongly suggests that the predictive power of P-wave measurements are not limited to acute inpatient settings. "P pulmonale" merely represents an extreme at one end of a large continuum of P-wave amplitudes. Even though the extreme end of the spectrum can be seen in acute settings and in emergency departments, the minimal changes of the P-wave amplitude or duration (outside the extreme ends of the spectrum) in outpatient settings can be suggestive of worsening right or left sided pressures, and thus possibly, progression of lung disease or left sided processes.

Kaplan-Meier Survival Curves

Survival plots for CV death are shown in Figure 1, stratifying P-wave duration and P-wave inversion scores; survival plot for pulmonary death is shown in Figure 2, stratifying the P-wave amplitude score. A significant and quantitative separation was demonstrated for each score ($P < .0001$ by log-rank test). The annual CV mortality rate for P-wave duration ⩽120 ms was 1.2% and increased significantly per score increment, with a 3.4% annual CV mortality rate for P-wave durations >140 ms. The annual CV mortality rate for the depth of P-wave inversion of −50 μ V or more was 0.96% and increased significantly per each score increment, with a 6.3% annual CV mortality rate for P-wave inversions deeper than −150 μ V. The annual pulmonary mortality rate for P-wave amplitude of ⩽200 μ V was 0.49% and increased significantly per score increment, with a 4.4% annual pulmonary mortality rate for P-wave amplitudes taller than 300μ V.

Discussion

Atrial enlargement and the P wave received attention in the first half of the 1900s, and a myriad of criteria evolved for both right and left atrial enlargement. Studies[1,2] seeking

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Table 2. Analysis of Variables Adjusted for Age and Heart Rate

Cause of Death	Cardiovascular			Pulmonary Death		
Variable	Regression Coefficient	Hazard Ratio	*P* Value	Regression Coefficient	Hazard Ratio	*P* Value
P-wave duration (ms)	0.01	1.01	<.0001	−0.01	0.99	<.0001
P-wave duration >120 ms	0.37	1.45	<.0001	−0.32	0.73	.0004
P-wave duration score	0.22	1.25	<.0001	−0.17	0.84	.001
P-wave inversion in leads V_1 or V_2 < − 100 μ V	0.95	2.57	<.0001	0.32	1.38	.003
Depth of P-wave inversion score	0.45	1.56	<.0001	0.16	1.17	.0003
P terminal force V_1 >40 (mm × ms)	0.38	1.47	<.0001	0.13	1.13	.08
P-wave amplitude in inferior leads (μ V)	−0.001	0.999	.01	0.01	1.01	<.0001
P-wave amplitude inferior leads >250 μ V	0.39	1.48	.004	1.16	3.20	<.0001
P-wave amplitude score	0.21	1.24	<.0001	0.53	1.70	<.0001
Left atrial abnormality	0.77	2.15	<.0001	0.44	1.56	<.0001
Right atrial abnormality	0.34	1.41	.01	1.09	2.98	<.0001

to relate electrocardiographic criteria for right atrial abnormality with echocardiographic findings were disappointing, demonstrating low sensitivity. Similarly, studies[3–6] showed a poor relationship between electrocardiographic criteria for LAA and echocardiographic evidence of its presence. The Macruz index[25] (P-wave duration divided by P-R segment) described in 1958 to differentiate right from left atrial enlargement also fell out of favor due to its limited sensitivity.[6] However, there has been a renewed interest in P-wave and atrial abnormalities, rekindled by the recent prominence of atrial fibrillation and its sequelae in clinical practice and the literature. Recent studies have suggested that increased P-wave duration is a precursor for atrial tachyarrhythmias, mainly atrial fibrillation.[10,26–28] Increased P-wave duration is prevalent,[29,20] associated with left atrial electromechanical dysfunction, and a potential risk of embolism.[31,32]

The few studies which have addressed the prognostic value of P-wave changes, were performed in selective and limited populations of patients. Perkiomaki et al[15] studied the independent value of ECG variables in predicting cardiac events after acute myocardial infarction. After adjustment for all risk variables, lateral ST-segment depression and LAA were the only ECG variables that independently predicted cardiac death. Incalzi et al[12] demonstrated the prognostic implications of ECG signs of chronic cor pulmonale in patients with chronic obstructive pulmonary disease. The $S_1S_2S_3$ pattern and a P-wave axis of >90-degrees were significant independent predictors of mortality over 13 years of follow-up. Bossone et al[16] demonstrated in 51 patients with primary pulmonary hypertension that significant predictors of decreased survival included pulmonary vascular resistance, cardiac index, P-wave amplitude in lead II >250 μ V, qR wave in lead V_1, and right ventricular hypertrophy. Lorbar et al[33] recently demonstrated an exceptionally high prevalence of interatrial block (P-wave duration ⩾110 ms) in patients with a probable embolic cerebrovascular event and proposed interatrial block as a new risk factor for stroke.

While previous studies have considered the association of atrial abnormalities on ECG with echocardiographic, clinical status, or prognosis in selected populations of coronary artery disease and pulmonary patients, our study is the first to evaluate the prognostic significance of P-wave measurements with specific cause of death in a general clinical population. These measurements have additional predictive value compared to many commonly used ECG findings and are readily available, widely applicable, noninvasive, easily interpretable, and reproducible for clinical risk stratification.

The practicing physician is often confronted with P-wave abnormalities which are usually considered nonspecific and are widely overlooked.[34,35] The physician might apply our findings to help make decisions regarding further cardiac or pulmonary testing and follow-up, the intensity of risk factor modification, and/or referral to a cardiologist or pulmonologist.

Limitations

All of our patients were male veterans. We do not have the specific reasons for why the ECGs were obtained, but

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Table 3. Multivariate Comparison of P-Wave Measurements With Other ECG Predictors

Cardiovascular Death			
Variable	Regression Coefficient	Hazard Ratio	*P* Value
T-wave abnormality	0.47	1.60	<.0001
Depth of P-wave inversion in leads V_1 or $V_2 < -100\,\mu V$	0.55	1.74	<.0001
Diagnostic Q wave	0.51	1.66	<.0001
Corrected QT interval >450 ms	0.30	1.35	<.0001
Left ventricular hypertrophy	0.38	1.46	<.0001
Intraventricular conduction delay	0.25	1.28	<.0001
P-wave duration >120 ms	0.19	1.21	<.0001
ST-depression	0.19	1.21	<.0001
QRS duration >120 ms	0.34	1.41	.0003
Right axis deviation	0.32	1.38	.01
Left axis deviation	0.13	1.14	.01
Right bundle branch block	−0.31	0.73	.01
P-wave amplitude inferior leads $>250\,\mu V$	0.28	1.32	.04
Pulmonary Death			
Variable	Regression Coefficient	Hazard Ratio	*P* Value
P-wave amplitude inferior leads $>250\,\mu V$	1.11	3.03	<.0001
Right axis deviation	0.55	1.73	<.0001
Left atrial abnormality	0.47	1.59	<.0001
P-wave duration >120 ms	−0.37	0.69	<.0001
Right ventricular hypertrophy	0.90	2.35	.003
Left ventricular hypertrophy	−0.31	0.73	.04

have accounted for those obtained in an acute setting. The ECGs were obtained from inpatients and outpatients and represent findings from a very broad range of patients. This contrasts with previous studies which focused on individuals with specific conditions or those that were considered community epidemiological cohorts. Thus, our sample comes from a setting where the ECG is commonly used as the first assessment tool for possible cardiac or pulmonary disease and is used by physicians in the decision process to determine the need for further testing.

We do not have baseline clinical, echocardiographic, and laboratory data on our patients but can account for those presenting with acute symptoms and those hospitalized. Therefore, we cannot say how helpful P-wave abnormalities would be for screening for early pulmonary or cardiac disease or if they are independent of other information. Retrospective gathering of other information and test results would be highly selective so the independence of the P-wave abnormalities could only be accurately assessed using a prospectively designed study.

Conclusions

P-wave abnormalities are common findings that should not be ignored. P-wave amplitude in the inferior leads is the strongest independent predictor of pulmonary mortality outperforming all of the classical ECG risk indicators. P-wave duration and the depth of P-wave inversion in leads V_1 or V_2 are strong independent predictors of CV death, even stronger predictors than many previously established ECG criteria. These P-wave measurements can be obtained

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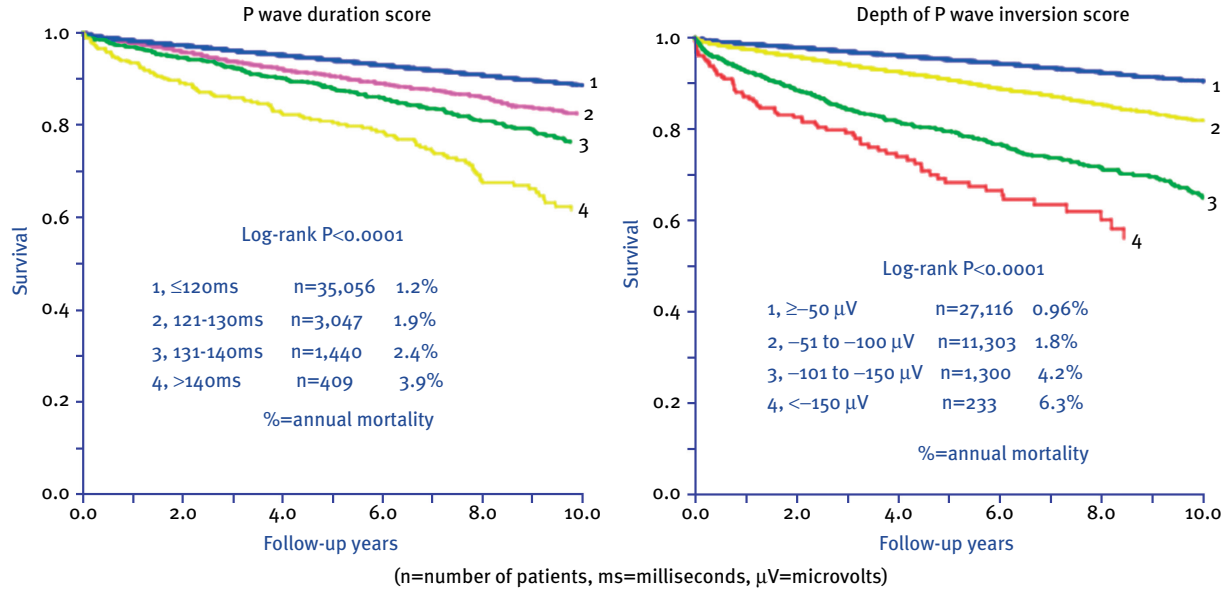

Figure 1. Kaplan-Meier survival plots for P-wave duration and the depth of P-wave inversion scores and CV death. *Abbreviations*: n, number of patients; ms, milliseconds; µ V, microvolts.

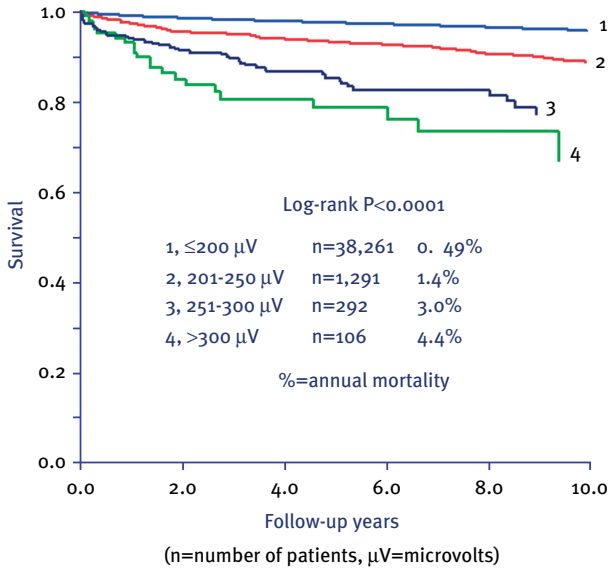

Figure 2. Kaplan-Meier survival plot for P-wave amplitude score and pulmonary death. *Abbreviations*: n, number of patients; µ V, microvolts.

easily and should be considered as part of clinical practice to stratify patients for risk of CV and pulmonary death.

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