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P Wave Dispersion in Patients with Generalised Anxiety Disorder

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Abstract

Introduction: Generalised anxiety disorder (GAD) is a prevalent condition defined by persistent, irrational, and uncontrollable worries regarding various daily activities. While previous research has identified alterations in atrial electrophysiology in conditions such as panic disorder and obsessive-compulsive disorder, P-wave dispersion (Pd) remains largely unexplored in GAD. This study aimed to evaluate Pd parameters—specifically maximum P-wave duration (Pmax) and minimum P-wave duration (Pmin)—in patients with GAD to identify potential cardiac risks associated with chronic anxiety.

Materials and Methods: A case-control study was conducted involving 20 patients diagnosed with GAD via the SCID-5-CV and 20 age- and sex-matched healthy controls. Standard 12-lead surface electrocardiograms (ECGs) were recorded at a paper speed of 50 mm/s. Pmax and Pmin were manually measured by a blinded cardiologist using high-precision callipers, with Pd calculated as the difference between the two values (Pmax-Pmin). Symptom severity was assessed using the Hamilton Anxiety Rating Scale (HARS).

Results: Statistical analysis revealed that Pmax and Pmin values were significantly elevated in patients with GAD compared to healthy controls. Notably, the primary outcome measure—mean P-wave dispersion—was significantly higher in the patient cohort (50.74 ± 6.10 ms) than in the control group (35.32 ± 3.79).

Conclusion: Patients with GAD demonstrate a distinct electrophysiological profile characterised by a global prolongation of atrial conduction parameters and significantly increased P-wave dispersion. These findings suggest that the "mental" worry of GAD carries a measurable "physical" cardiac risk, necessitating closer clinical monitoring of atrial electrophysiology, particularly when prescribing psychotropic medications that may influence cardiac conduction.

Key words: Anxiety disorders; atrial function; case-control study; electrocardiography.

Introduction

Generalised anxiety disorder (GAD) is classified as an anxiety disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), 5th edition (1). It is defined by excessive, uncontrollable, and often irrational worry. Catastrophically negative and dysfunctional thoughts concerning a variety of daily activities are frequently exhibited by patients with GAD. Furthermore, the specific themes of worry are often found to be similar between patients with GAD and healthy controls. In patients with GAD, daily activities are influenced by this excessive worry, as highlighted in the diagnostic criteria. The specific concerns often centre on daily life domains such as family problems, health, interpersonal relationships, employment, finances, or mortality (2). P wave dispersion (Pd) is defined as the difference between the maximum (Pmax) and minimum (Pmin) P wave durations observed on the electrocardiogram (ECG) (3). Also, the maximum P wave duration has been utilised to denote the prolongation of atrial conduction time and the discontinuous propagation of sinus impulses. Previous research has explored P wave dispersion across various anxiety-related conditions, consistently identifying alterations in atrial electrophysiology. Investigations in patients with panic disorder (PD) and hypochondriasis have demonstrated significant increases in both Pmax and Pmin values, leading to a notably higher Pd compared to healthy controls (4,5). Similarly, studies in obsessive-compulsive disorder (OCD) reported significantly higher Pmax and Pd, although no differences were found in Pmin or

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left atrial size in that specific cohort (3). Collectively, these findings suggest that different manifestations of anxiety are associated with increased Pd, potentially elevating the risk for cardiac arrhythmias. It is well-established that worry, fear, and anxiety are associated with the hypothalamic–pituitary–adrenal axis (HPA). These emotions provoke the release of corticotropin-releasing hormone (CRH) from the hypothalamus, which triggers the subsequent production of adrenocorticotropin hormone (ACTH) from the pituitary gland. This cascade stimulates cortisol release from the adrenal cortex. The aforementioned cascade has been shown to be closely related to the autonomic nervous system (ANS) (6). Furthermore, close associations exist between the ANS and heart rate variability (HRV), which has been proposed as a tool to investigate the function of the ANS. It has also been emphasised that the ANS is involved in both the duration of cardiac ventricular repolarisation, by conditioning it to changes in heart rate, and the spatial heterogeneity of repolarisation (7). As evidenced by the knowledge outlined above, a close relationship exists between anxiety, the ANS, and cardiac conduction. To the best of our knowledge, Pd in patients with GAD is a relatively unexplored topic in psychiatric and cardiological literature. Against this backdrop, the primary objective of the present study was to investigate Pd parameters, specifically P(max) and P(min) values, in a cohort of patients diagnosed with GAD, compared to healthy control subjects. This evaluation aimed to ascertain whether the atrial electrophysiological changes observed in other anxiety disorders are also present in this population. The study aims to contribute to the existing body of knowledge by providing preliminary data on the potential relationship between chronic anxiety and atrial conduction. Such findings could emphasise the importance of monitoring atrial electrophysiology when managing GAD patients, especially when considering psychotropic medications that could affect cardiac conduction.

Materials and Methods

Study design and participants: The research was conducted between 1 February 2021 and 1 September 2021. Twenty patients with GAD and twenty healthy controls were recruited for this study. All GAD diagnoses were confirmed using the Structured Clinical Interview for DSM-5 Disorders, Clinical Version (SCID-5-CV), in accordance with DSM-5 criteria (8,9). Healthy controls were recruited from volunteers who had applied to the psychiatric outpatient clinic for a clean bill of health. GAD frequently presents with psychiatric comorbidity. While the study prioritised the recruitment of patients without concurrent conditions, four individuals presented with comorbidities: major depressive disorder (n=1), obsessive-compulsive disorder (n=1), social anxiety disorder (n=1), and specific phobia (n=1; elevator phobia). All participants, including healthy controls, underwent comprehensive physical examinations and routine laboratory evaluations, with no significant abnormalities identified. Specific inclusion and exclusion criteria were applied to all participants. A minimum two-week washout period was required for all vasoactive and psychotropic medications (e.g., antipsychotics and anxiolytics). For those on antidepressant therapy, dosages were required to be stable for at least four weeks prior to enrolment. Exclusion criteria comprised: myocardial infarction within the preceding two months; resting blood pressure exceeding 180/120 mmHg; current or previous congestive heart failure; and pre-existing cardiac conditions, including valvular heart disease, cardiomyopathy, arrhythmias, or the presence of a pacemaker. Furthermore, individuals with severe systemic diseases (such as malignancy or endocrinological disorders), congenital heart disease, or any condition known to affect autonomic function were excluded. Standard 12-lead electrocardiograms (ECGs) were recorded for all GAD patients and healthy controls in a quiet room within the Cardiology Department. To ensure physiological stability, participants rested in the supine position for ten minutes prior to the recording. All ECGs were obtained during morning hours under conditions of spontaneous breathing. The Hamilton Anxiety Rating Scale (HARS) was used to assess the severity of anxiety symptoms in all participants. This 14-item clinician-rated scale measures both psychic anxiety (mental agitation and psychological distress) and somatic anxiety (physical complaints related to anxiety), with each item defined by a series of symptoms. Each item is scored on a 5-point scale ranging from 0 (not present) to 4 (very severe). A total score of 0–17 indicates mild severity, 18–24 indicates mild to moderate severity, and 25–56 indicates moderate to severe anxiety (10,11).

The analysis of P wave dispersion: Standard 12-lead surface ECGs were recorded for all participants in a resting supine position using a Nihon Kohden device (Tokyo, Japan). During the procedure, participants were instructed to remain silent while breathing spontaneously. Recordings were obtained at a paper speed of 50 mm/s with three simultaneous leads. P-wave durations (Pmax and Pmin) were manually measured by a senior cardiologist who was blinded to the participants' clinical diagnoses. Measurements were performed using high-precision calipers and a magnifying lens to ensure the accurate identification of ECG deflections (12,13). To determine P-wave dispersion (Pd), specific ECG parameters were first defined. The onset of the P-wave was identified as the junction between the start of the P-wave deflection and the isoelectric line, while the offset was defined as the point where the P-wave deflection met the isoelectric line. The maximum P-wave duration (Pmax) was recorded from the 12-lead ECG as a marker of prolonged atrial conduction time. Finally, P-wave dispersion was calculated as the difference between the maximum and minimum P-wave durations (Pd = Pmax - Pmin). Figures 1 and 2 show the Pmax and Pmin measurements from a participant's ECG sample.

Table 1: The comparison of participants' sociodemographic data and scale scores.

	Control group (n=20)	**Patient group (n=20)**	**p value**
Age	30.11 ± 5.89	29.12 ± 4.28	0.547[a]
Sex			0.743[b]
Female	12 (60%)	13 (65%)	
Male	8 (40%)	7 (35%)	
Marital status			0.723[b]
Single	6 (30%)	5 (25%)	
Married	14 (70%)	15 (75%)	
Employment status			0.231[c]
Employed	20 (100%)	17 (85%)	
Unemployed)	0	3 (15%)	
Education status			0.106[c]
Primary school and below	0	4 (20%)	
Secondary school and above	20 (100%)	16 (80%)	
Smoking status			0.490[b]
Yes	5 (25%)	7 (35%)	
No	15 (75%)	13 (65%)	
Alcohol status			1.000
No	20 (100%)	20 (100%)	
Hamilton Anxiety Rating Scale	5.13 ± 2.92	19.38 ± 4.10	<0.001[a]

[a]Student t-test [b]Chi square test [c]Fisher's Exact Test

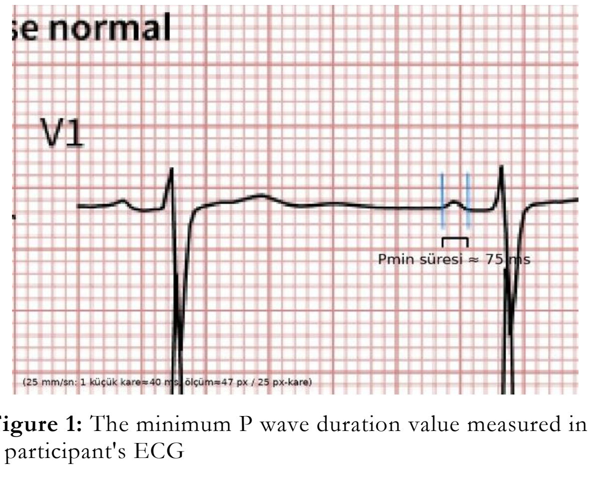

Figure 1: The minimum P wave duration value measured in a participant's ECG

Figure 2: The maximum P wave duration value measured in a participant's ECG

Power analysis: A post-hoc power analysis was performed for the primary outcome variable (P wave dispersion). Given the observed means of 50.74 ms and 35.32 ms, with standard deviations of 6.10 ms and 3.79 ms respectively, and a sample size of 20 per group, the calculated effect size (Cohen's d) was 3.04. This resulted in a statistical power of >0.99 at an alpha level of 0.05, suggesting that despite the relatively small sample size, the study was sufficiently powered to detect the identified differences in atrial conduction.

Ethical approval: Ethical approval for the study was obtained from the Firat University School of Medicine Local Ethics Committee on 14 January 2021 (Approval Number: 2021/01-37). Subsequent to the approval process, written informed consent was formally secured from all participating patients and healthy control subjects.

Statistical analysis: Statistical analyses were conducted using the IBM SPSS Statistics for Windows, Version 22.0 (Armonk, NY: IBM Corp.). The normality of the data distribution was assessed using the Shapiro-Wilk test, alongside an evaluation of skewness and kurtosis values. For the comparison of continuous variables between two independent groups that met the criteria for normal distribution, the Student's t-test was utilised. Categorical data were analysed using the Chi-square test or Fisher's exact test, where appropriate, to determine associations between variables. The relationship between continuous variables was examined using the Pearson correlation coefficient. For all statistical evaluations, a p-value of less than 0.05 was considered to indicate statistical significance.

Results

As illustrated in Table 1, the control and patient groups were well-matched, with no statistically significant differences observed in age, sex distribution, marital status, employment, or education level. Lifestyle factors, including smoking and alcohol consumption, were also comparable between the two cohorts. However, the patient group exhibited significantly higher Hamilton Anxiety Rating Scale (HARS) scores (19.38 ± 4.10) relative to the control group (5.13 ± 2.92; p < 0.001). Comparative analysis of electrocardiogram (ECG) parameters, summarised in Table 2, revealed significant differences between the groups. The patient group demonstrated a significantly longer maximum P wave duration (91.12 ± 7.92 ms vs 66.88 ± 5.80 ms; p < 0.001) and minimum P wave duration (40.38 ± 6.09 ms vs 31.56 ± 4.04 ms; p < 0.001) compared to controls. Consequently, P wave dispersion was significantly higher in the patient cohort than in the control cohort (50.74 ± 6.10 ms vs 35.32 ± 3.79 ms; p < 0.001). The relationships between age, clinical scores, and ECG variables are presented in Table 3. Pearson correlation analysis indicated that neither age nor HARS scores were significantly associated with Pmax, Pmin, or Pd.

Table 2: The comparison of the electrocardiogram findings of the participants.

	Control group (n=20)	Patient group (n=20)	p value
Pmax	66.88 ± 5.80	91.12 ± 7.92	<0.001[a]
Pmin	31.56 ± 4.04	40.38 ± 6.09	<0.001[a]
P wave dispersion	35.32 ± 3.79	50.74 ± 6.10	<0.001[a]

[a]Student t-test **Pmax:** Maximum P wave duration, **Pmin:** Minimum P wave duration

Table 3: The correlation between ECG parameters, age and scale scores

Variables		Age	HARS	Pmax	Pmin	Pd
Age		1				
HARS	r	0.109	1			
	p	0.280				
Pmax (ms)	r	0.127	-0.065	1		
	p	0.208	0.521			
Pmin (ms)	r	0.014	-0.062	-0.574	1	
	p	0.890	0.541	0.001		
Pd (ms)	r	0.119	-0.101	0.964	-0.594	1
	p	0.240	0.320	0.001	0.001	

***Pearson correlation test **Pmax:** Maximum P wave duration, **Pmin:** Minimum P wave duration, **Pd:** P wave dispersion, **HARS:** Hamilton Anxiety Rating Scale, **ms:** millisecond

Discussion

To the best of our knowledge, the present study is the first to examine Pmax, Pmin, and P-wave dispersion in the context of GAD. Our results yield several important observations: both Pmax and Pmin values were significantly higher in the

GAD group than in healthy controls. Most notably, the mean P-wave dispersion was found to be significantly elevated in patients, suggesting a distinct electrophysiological profile in this population. Emotional states such as worry and anxiety activate the HPA axis, beginning with the hypothalamic release of CRH. This induces pituitary secretion of ACTH and the subsequent release of cortisol from the adrenal cortex. In patients with anxiety, this hormonal cascade plays a critical role due to its established influence on autonomic nervous system (ANS) function (6). The link between GAD and increased Pd is fundamentally rooted in the effect ofANS dysregulation on atrial electrophysiology. Chronic anxiety frequently activates the HPA axis, leading to a shift in autonomic balance characterised by sympathetic overactivity and a reduction in vagal tone. This vagal withdrawal removes the stabilising inhibitory effect on the heart, allowing catecholamines to exert a dominant influence on atrial tissue. Such sympathetic overactivity promotes spatial heterogeneity in atrial refractoriness, creating an electrical environment in which different regions of atrial myocardium recover at different rates. When sinus impulses travel through tissue with varying refractory periods, conduction becomes fragmented and non-uniform. Furthermore, sympathetic surges can shorten action potential durations and atrial refractory periods, while also slowing conduction velocity in specific areas. This directly contributes to the difference between Pmax and Pmin. The ANS also modulates the exit of impulses from the sinus node and their subsequent propagation through internodal pathways (7,14,15). Under autonomic stress, the discontinuous propagation of these impulses is exacerbated, contributing to the prolongation of Pmax observed in our GAD cohort. Taken together, these mechanisms imply that persistent mental worry in GAD patients manifests as a physical state of electrical instability in the atria, which could lower the threshold for atrial arrhythmias. Furthermore, the impact of anxiety on healthy populations is well-documented; persistent anxiety appears to disrupt cardiac autonomic balance, potentially increasing the risk of coronary heart disease (12,16). Despite these well-established links, relatively few studies have examined how cardiac conduction changes across different anxiety disorders. Our findings in patients with GAD align with and extend the observations made in other anxiety-related disorders. Although we observed significant increases in Pmax, Pmin and Pd, similar to those seen in panic disorder and hypochondriasis, the clinical implications for GAD are different (4,5). Unlike panic disorder, which is characterised by episodic autonomic surges, GAD involves a state of persistent, uncontrollable worry. The fact that GAD patients exhibit similar Pd elevations suggests that chronic, low-grade sympathetic modulation may impact atrial conduction as much as acute panic states. Furthermore, our results differed slightly from those in the OCD literature, where Pmin remained unchanged (3). In our GAD cohort, however, both Pmax and Pmin were significantly elevated. This global prolongation of atrial conduction parameters in GAD patients may reflect a more pervasive influence on the autonomic nervous system. These electrophysiological changes, likely mediated by the HPA axis and subsequent ANS imbalance, emphasise that the 'mental' worry of GAD carries a measurable 'physical' cardiac risk, even in the absence of acute physical symptoms. While previous research investigated Pd in conditions like panic disorder, obsessive-compulsive disorder, and hypochondriasis, GAD represents a distinct clinical entity characterized by constant, pervasive worry rather than episodic panic or specific obsessions. By specifically isolating GAD, our study provides novel evidence that chronic, 'background' anxiety—even in the absence of acute panic attacks—is sufficient to significantly alter atrial electrophysiology. This finding is superior to existing literature as it extends the known cardiovascular risks of anxiety to the most common anxiety disorder in clinical practice, which had previously been overlooked in this context.

Study limitations: When interpreting the results of the present study, several limitations must be considered to prevent conclusions that exceed its intended purpose. Firstly, although our post-hoc power analysis suggested that the study was sufficiently powered to detect differences in P-wave dispersion, the relatively small sample size remains a primary limitation. Small cohorts are more susceptible to sampling variability, which can lead to effect size inflation — a phenomenon whereby observed effects appear significantly larger than they would in a broader population. The notably large effect size reported here (Cohen’s d = 3.04) should therefore be interpreted with caution. While these results provide a strong preliminary indication of altered atrial electrophysiology in GAD, they may not be generalisable to all GAD patients, particularly those with different demographic profiles or lower symptom severity. Consequently, larger, multicentre studies are required to validate these findings and provide a more precise estimate of the true effect size in the general population. Secondly, a lack of other cardiac conduction parameters, such as HRV, apart from ECG changes, must be acknowledged. Although GAD was the primary focus of this study, the presence of psychiatric comorbidities in four participants constitutes a notable confounding factor. Anxiety and depressive disorders are known to share overlapping neurobiological mechanisms, particularly dysregulation of the hypothalamic–pituitary–adrenal axis and consequent autonomic nervous system imbalance. Accordingly, the observed increases in Pmax, Pmin, and Pd in these individuals may reflect the cumulative effects of multiple psychiatric stressors on atrial electrophysiology rather than GAD-related worry alone. This potential amplification of atrial electrical instability represents a limitation of the study, underscoring the need for larger, more homogeneous cohorts to better delineate the independent contribution of GAD. Lastly, the potential influence of pharmacological treatments must be considered. While we required a minimum two-week washout period for most psychotropics and stable antidepressant dosing for at least four weeks, it is known that selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors modulate autonomic tone, which could impact atrial conduction parameters. Additionally, while we compared basic demographic and lifestyle factors such as smoking and alcohol use, other variables such as body mass index, daily caffeine consumption, physical activity levels and sleep patterns were not systematically assessed. These factors could be sources of residual confounding that may have influenced the observed differences in P-wave parameters between the groups.

Conclusion

Our results indicate that GAD is associated with elevated Pmax, Pmin, and P-wave dispersion values, signifying altered atrial electrophysiology. Consequently, clinical monitoring is warranted in GAD patients, especially when prescribing psychotropic treatments known to influence cardiac conduction pathways. As the findings of the present study are preliminary, several recommendations for future research are warranted. Firstly, subsequent studies should utilise significantly larger sample sizes to enhance statistical power and generalisability. Secondly, future investigations would benefit from including a broader range of cardiac conduction markers, specifically heart rate variability (HRV), in addition to the ECG parameters measured here, to provide a more comprehensive assessment of autonomic functioning. Thirdly, given the high prevalence of psychiatric comorbidity in GAD, future studies should implement stricter controls for concurrent conditions and subclinical depressive symptoms in order to isolate the specific effects of generalised anxiety on atrial electrophysiology. Finally, longitudinal research is essential to evaluate whether successful treatment, whether pharmacological or psychotherapeutic, can reverse the observed increase in P wave dispersion and mitigate long-term cardiac risk.

Ethical approval: Ethical approval for the study was obtained from the Firat University School of Medicine Local Ethics Committee on 14 January 2021 (Approval Number: 2021/01-37). Subsequent to the approval process, written informed consent was formally secured from all participating patients and healthy control subjects.
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