North Clin Istanb 2025;12(5):627–632
doi: 10.14744/nci.2024.13914

Original Article NEONATOLOGY

Is the P-wave dispersion a valuable tool in determining the atrial arrhythmia of newborn babies with sepsis?

Fatma Hilal Yilmaz,[1] Derya Aslan,[2] Cengizhan Ceylan[3]

[1]Department of Neonatology, Ministry of Health Konya Dr. Ali Kemal Belviranlı Obstetrics and Gynecology Hospital, Konya, Turkiye
[2]Department of Pediatric Cardiology, Health Sciences University Konya City Hospital, Konya, Turkiye
[3]Department of Clinical Pharmacy, Selcuk University Faculty of Pharmacy, Konya, Turkiye

ABSTRACT

OBJECTIVE: This study aimed to ensure that newborns receive early diagnosis through P-wave dispersion in terms of possible atrial arrhythmias on the provocative basis of sepsis.

METHODS: In this prospective study twenty term sepsis and twenty preterm sepsis patients were compared with control groups with their own characteristics in terms of P-wave dispersion by electrocardiography.

RESULTS: The P-wave dispersion value in patients with term sepsis was determined to be statistically significant (p=0.03). There was no difference in APW between the patient and control groups in preterms (p<0.05). Heart rate in preterm patients with sepsis was found to be statistically significantly higher than in the control group (p=0.001).

CONCLUSION: Our study revealed a tendency for atrial arrhythmias in term newborn infants with sepsis. These babies should be followed by pediatric cardiology with cardiac monitoring throughout their treatment.

Keywords: Atrial arrhythmias; newborn; P-wave dispersion; sepsis.

Cite this article as: *Yilmaz FH, Aslan D, Ceylan C. Is the P-wave dispersion a valuable tool in determining the atrial arrhythmia of newborn babies with sepsis? North Clin Istanb 2025;12(5):627–632.*

Neonatal sepsis is a clinical syndrome caused by the spesific pathogens in which systemic signs and symptoms of infection are observed in the first month of life [1]. Neonatal sepsis can be categorized into two types: early onset, which manifests as respiratory distress within 72 hours after birth due to perinatal risk factors, and late onset, which is diagnosed after 72 hours and develops secondary to nosocomial risk factors [2]. Despite the developments in the neonatal field, it is still an important cause of morbidity and mortality [3]. The mortality rate due to sepsis can be up to 24.4%, but this rate can be up to 30% in babies born between 25 and 28 weeks, and up to 54% in babies born between 22 and 24 weeks [4]. The lack of specific findings related to sepsis and the similar signs and symptoms of non-infectious clinical conditions frequently encountered in the neonatal period create diagnostic difficulties.

In sepsis, upregulation of pro- and antiinflammatory pathways leads to system-wide release of cytokines, and activated cascades lead to myocardial dysfunction [5]. Hyperinflammation, the combination of myocardial dysfunction and extra fluid volume with resuscitation, leading to the development of stress in the left atrium [6]. These pathological changes of myocardium may lead to rhythm disturbances in patients with sepsis. The existence of various cardiac arrhythmias in septic patients has been demonstrated by various clinical reports and studies [7–9]. Troponin values in newborns are variable and no pathology may be observed on echocardiogra-

Received: March 26, 2024 *Accepted:* September 04, 2024 *Online:* October 31, 2025

Correspondence: Fatma Hilal YILMAZ, MD. Konya Dr. Ali Kemal Belviranlı Kadin Hastaliklari ve Dogum Hastanesi, Yenidogan Klinigi, Konya, Turkiye.
Tel: +90 332 235 42 05 e-mail: f.h.yilmaz@hotmail.com

Istanbul Provincial Directorate of Health - Available online at www.northclinist.com

phy until the decompensated period [10]. Thus, damage to the conduction pathways and/or myocardium can be detected practically and non-invasively by electrocardiography (EKG). P-wave dispersion (PWD) is a measure of the likelihood of developing abnormal heart rhythms, which is determined via a non-invasive method that involves analyzing a surface electrocardiogram. An elevation in PWD is presumed to be linked to an escalation in the likelihood of both first and recurring episodes of atrial arrhythmias. Assessing the occurrence of atrial arrhythmias in newborns with sepsis might be a useful approach.

This study aimed to ensure that newborns, who are already prone to atrial arrhythmias, receive early diagnosis through PWD in terms of possible atrial arrhythmias on the provocative basis of sepsis. This will be the first study designed in this way in the neonatal patient group in the literature to date.

Highlight key points

- P wave dispersion (PWD) is a measure of the likelihood of developing abnormal heart rhythms.
- An increase in PWD is thought to be associated with an increased likelihood of both initial and recurrent episodes of atrial arrhythmia.
- The PWD value was significantly higher in term infants with sepsis. Therefore, these infants are prone to atrial arrhythmia. These infants should be monitored by pediatric cardiology with cardiac monitoring during treatment.

MATERIALS AND METHODS

The study was conducted prospectively from June 2023 to December 2023 in Ministry of Health Konya Dr. Ali Kemal Belviranlı Obstetrics and Gynecology Hospital, Department of Neonatology. 20 preterm and 20-term newborns with sepsis verified by clinical or laboratory testing, whom we monitored in the neonatal intensive care unit of our hospital, and 20 preterm and 20-term babies without sepsis as a control group were included in the study. The demographic and laboratory findings of the patients were recorded in the hospital information system. The patients with congenital cardiac defects, newborns of mothers with systemic lupus erythematosus, those with metabolic illnesses, and those with documented arrhythmias were excluded from the study.

The parents/legal guardians of the children provided informed consent.

Electrocardiogram Analysis

Electrodes were placed in anatomical places following standard protocol, and electrocardiography (ECG) strips were recorded for a duration of 10 seconds using a standard instrument. The 12-lead electrocardiogram (ECG) was obtained using a Cardiofax C machine manufactured by Nihon Kohden in Tokyo, Japan. The electrocardiogram (ECG) was obtained using a paper speed of 25 millimeters per second and an amplitude of 10 millimeters per millivolt, while the patient was in a supine posture. The ECG strips were evaluated in a manner that the pediatric cardiologist, who had over ten years of experience in the area, did not have access to any identifying information.

The initiation of the P wave was determined as the moment when positive waveforms first deviated upwards from the baseline, or when negative waveforms first deviated downwards from the baseline. The return to the starting state was acknowledged as the conclusion of the P wave. The length of the P-wave was calculated using data from all leads. The maximum, minimum, and mean durations of the P-wave, as well as the P-wave dispersion, were determined by subtracting the minimum P-wave duration from the maximum P-wave duration in 12 leads. This calculation was performed using one randomly selected beat in a steady state. Simultaneously, an average of 7–12 beats were calculated for a duration of 10 seconds for each ECG measurement. P-wave lengths and P-wave dispersion were computed using the same approach (P-wave dispersion equals the highest P-wave duration minus the shortest P-wave duration). In order to eliminate daily fluctuations, we collected electrocardiography recordings from all working groups within a consistent time frame of 10–12 hours.

Statistical Analysis

All statistical analyses were performed using SPSS 22.0 (Armonk, New York: IBM Corp.) program. Data were presented as count (n) and standard deviation. An independent sample t-test was performed. A p-value less than 0.05 is considered statistically significant. This study was conducted in accordance with the Declaration of Helsinki.

RESULTS

20 preterm healthy and 20 preterm sick babies, 20 term healthy and 20 term sick babies were included in this study. Table 1 shows demographic data, maternal ages, Agpar 1st and 5th minute scores, and peak heart rates of the patient and control groups.

Table 1. Characteristics of term and preterm patients

Variables	Group	n	Mean±SD	p
Birth week	Preterm sepsis	20	31.15±2.96	>0.05
	Preterm control	20	32.45±2.48	
	Term sepsis	20	39.00±1.41	>0.05
	Term control	20	38.584±.73	
Weight	Preterm sepsis	20	1713.25±558.36	>0.05
	Preterm control	20	1799.00±488.87	
	Term sepsis	20	3240.45±658.35	>0.05
	Term control	20	3273.55±525.72	
Maternal age	Preterm sepsis	20	26.35±6.20	p>0.05
	Preterm control	20	26.50±5.82	
	Term sepsis	22	27.00±6.60	p>0.05
	Term control	20	29.20±6.26	
Apgar 1 score	Preterm sepsis	20	6.60±1.53	**0.043**
	Preterm control	20	7.70±1.78	
	Term sepsis	22	790±1.37	
	Term control	20	8.70±0.57	**0.022**
Apgar 5 score	Preterm sepsis	20	7.50±1.10	**0.008**
	Preterm control	20	8.45±1.05	
	Term sepsis	20	8.72±1.03	>0.05
	Term control	20	9.20±0.41	

SD: Standard deviation.

Table 2. Characteristics of the term and preterm sepsis groups related to sepsis

Variable	Preterm sepsis	Term sepsis
Clinic sepsis	9	15
Klebsiella pneumoniae	6	2
Staphylococcus epidermidis	3	1
Candida	1	0
Staphylococcus haemolyticus	1	1
Streptococcus agalactiae	0	2
Staphylococcus aureus	0	1
C-reactive protein (mg/dl)	49.68±39.36	57.78±47.77
Length of stay (day)	45.05±27.63	16.59±13.93
Platelet count	165700.07±153692.71	176154.55±109020.04
White blood cell count (10^9/L)	12969.47±9509.44	213112.27±33823.57

The term and preterm patient and control groups were similar in terms of gender and mode of delivery.

Details of sepsis in the term and preterm sepsis groups are given in Table 2.

Table 3. Comparison of ECG findings between patients

Variables	Group	n	Mean±SD	p
P-wave dispersion	Preterm control	20	0.01±0.00	>0.05
	Preterm sepsis	20	0.02±0.03	
	Term control	20	0.01±0.00	**0.03**
	Term sepsis	20	0.01±0.00	
P-wave dispersion minumum	Preterm control	20	0.05±0.00	>0.05
	Preterm sepsis	20	0.05±0.00	
	Term control	20	0.07±0.00	>0.05
	Term sepsis	20	0.10±0.14	
P-wave dispersion maximum	Preterm control	20	0.06±0.00	>0.05
	Preterm sepsis	20	0.07±0.00	
	Term control	20	0.08±0.00	>0.05
	Term sepsis	20	0.11±0.14	
Heart rate	Preterm control	20	146.10±19.41	**0.01**
	Preterm sepsis	20	159.70±8.82	
	Term control	20	144.42±14.19	>0.05
	Term sepsis	20	143.30±16.36	

SD: Standard deviation.

When ECG findings (Table 3) of the study group were compared with the control group, it was found that average PWD value of the patients in the preterm control group was 0.013050±0.0033791 seconds. In patients with preterm sepsis, this value was determined to be 0.020409±0.0313531 seconds on average. No statistically significant difference was found between the groups (p>0.05).

The average heart rate of the patients in the preterm control group was determined to be 146.1053±19.41046, and this value was found to be 157.0455±13.43583 in patients with preterm sepsis. There was a statistically significant disparity in heart rates between the two groups (p=0.01).

While the average PWD values of the patients in the term control group were 0.011000±0.0031464 seconds, the average PWD values of the patients in the term sepsis group were 0.013667±0.0046726 seconds. A significant statistical difference was observed between the groups (p=0.03).

The correlation analysis between CRP and PWD did not yield any statistically significant results. (p=0.064).

DISCUSSION

Neonatal sepsis remains a significant contributor to morbidity and mortality and it has become a global health challenge [11]. Delayed treatment and prematurity often lead to adverse outcomes. It is known that sepsis increases the likelihood of neurodevelopmental complications such as cerebral palsy, psychomotor and mental developmental delay, especially in babies with low birth weight [12]. In addition, overdose antibiotic use can lead to the presence of multidrug-resistant organisms and candida infections. Sepsis can result in the failure of several organs. One of the primary causes of this failure is the impairment of the heart's function, known as cardiac dysfunction [13]. In experimental animal models of sepsis, the heart's production of ATP is predominantly achieved by the oxidation of fatty acids and glucose. However, both of these processes are significantly reduced [14]. On the other hand, impaired β-adrenergic signaling that leads to reduced cardiac contractility is also present in sepsis [15].

It is widely recognized that infants are susceptible to life-threatening irregular heart rhythms, which can hap-

pen in babies with a healthy heart or in those with cardiac abnormalities. The incidence of neonatal arrhythmia is reported to be 1%–5% in all neonates [16]. Turner et al. [17] retrospective study spanning two decades revealed an arrhythmia rate of 0.02% in 662,698 live births. However, Badrawi et al. [18] determined this rate as 8.5% in benign arrhythmias and 1.5% in malignant arrhythmias in patients in the neonatal intensive care unit. These data prove the increased tendency for arrhythmias in sick babies. Previous studies have shown that atrial arrhythmias, especially premature atrial contractions, can be seen with a frequency of up to 51% even among normal births [19]. Therefore, it is possible to say that newborns are more prone to atrial arrhythmias. Previous studies have reported that increased PWD is a strong indicator of the likelihood of atrial fibrillation [20–22]. In our study investigating sepsis and atrial arrhythmia in newborns, we found that the PWD value was significantly higher in term babies with sepsis. Nevertheless, we did not observe any supraventricular arrhythmia when administering therapy to these individuals. Similar to our study, Özdemir et al. [23] found that PWD was significantly higher in the sepsis group compared to the control group in pediatric intensive care unit patients with sepsis. Similar to the results of our study, they did not detect supraventricular arrhythmia in these patients. However, Walkey et al. [8] found that the risk of developing arterial fibrillation in adults with sepsis who were treated in the hospital was 6 times higher than in those without sepsis. Even in a study, the atrial fibrillation caused by sepsis rate was between 2–26%, and in cases of septic shock, this rate could increase up to 40% [24–26].

We did not detect a correlation between CRP, an important indicator of inflammation, and APW in our term patients with sepsis. In our study, we did not find a connection between the severity of sepsis and the tendency to atrial arrhythmia. While Özdemir et al. [23] did not find a correlation between crp and apw in their study, they showed a positive correlation with Tp-e interval and Tp-e/QTc ratio. They associated this with ventricular tachyarrhythmias based on myocardial fibrosis.

Neonates in the neonatal intensive unit are susceptible to sepsis, especially preterm neonates and low birth weight neonates [27]. In our study, we did not find any difference in PWD between the sepsis and control groups in preterms, whose hemodynamics and physiology are different from term babies. We attributed this difference in babies with preterm sepsis to lack of maturation in every system of their body. However, heart rate was found to be higher in these babies with sepsis. The gold standard for diagnosis is the presence of a positive blood culture. But, the initiation of treatment is not dependent on it since the results are often delayed. So timely treatment of antibiotics will decrease the length and intensity of the illness. Deviation from normal heart rate patterns is linked to the onset of sepsis and might be crucial in promptly identifying and treating high-risk infants. In sepsis, the adrenergic system is activated, and overproduction of catecholamines leads to tachycardia [28]. There are even studies showing that early recognition of sepsis by observing heart rate variability with the use of the Hemoaccess Reliable Outflow device reduces mortality in newborns [29, 30].

The study has some limitations. Increasing the sample size would enhance the accuracy of our result interpretation. Evaluation of ECG parameters indicating ventricular arrhythmias would provide a stronger chance of predicting arrhythmias in patients with sepsis.

Conclusion

Our study revealed a tendency for atrial arrhythmias in term newborn infants with sepsis. These babies should be followed by pediatric cardiology with cardiac monitoring throughout their treatment.

Ethics Committee Approval: The Selcuk University Clinical Research Ethics Committee granted approval for this study (date: 19.12.2023, number: 2023/592).

Informed Consent: Written informed consents were obtained from patients who participated in this study.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

Use of AI for Writing Assistance: The authors declared that artificial intelligence was not used in the study.

Authorship Contributions: Concept – FHY, DA; Design – FHY; Supervision – FHY; Materials – FHY, DA; Data collection and/or processing – FHY, CC, DA; Analysis and/or interpretation – FHY, CC, DA; Literature review – FHY, CC, DA; Writing – FHY, CC, DA; Critical review – FHY.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Kim F, Polin RA, Hooven TA. Neonatal sepsis. BMJ 2020;371. [Crossref]
2. Hincu MA, Zonda GI, Stanciu GD, Nemescu D, Paduraru L. Relevance of biomarkers currently in use or research for practical diagnosis approach of neonatal early-onset sepsis. Children 2020;7:309. [Crossref]

3. Stoll BJ, Hansen NI, Bell EF, Walsh MC, Carlo WA, Shankaran S, et al. Trends in care practices, morbidity, and mortality of extremely preterm neonates, 1993-2012. JAMA 2015;314:1039-51. [Crossref]
4. Sharma D, Farahbakhsh N, Shastri S, Sharma P. Biomarkers for diagnosis of neonatal sepsis: a literature review. J Matern Fetal Neonatal Med 2018;31:1646-59. [Crossref]
5. Chousterman BG, Swirski FK, Weber GF. Cytokine storm and sepsis disease pathogenesis. Semin Immunopathol 2017;39:517-28. [Crossref]
6. Romero-Bermejo FJ, Ruiz-Bailen M, Gil-Cebrian J, Huertos-Ranchal MJ. Sepsis-induced cardiomyopathy. Current Cardiol Rev 2011;7:163-83. [Crossref]
7. Walkey AJ, Evans SR, Winter MR, Benjamin EJ. Practice patterns and outcomes of treatments for atrial fibrillation during sepsis: a propensity-matched cohort study. Chest 2016;149:74-83. [Crossref]
8. Walkey AJ, Wiener RS, Ghobrial JM, Curtis LH, Benjamin EJ. Incident stroke and mortality associated with new-onset atrial fibrillation in patients hospitalized with severe sepsis. JAMA 2011;306:2248-54. [Crossref]
9. Leng Y, Li Y, Wang J, Deng P, Wang W, Wu J, et al. Sepsis as an independent risk factor in atrial fibrillation and cardioembolic stroke. Front Endocrinol 2023;14. [Crossref]
10. Karlén J, Karlsson M, Eliasson H, Bonamy AE, Halvorsen CP. Cardiac troponin T in healthy full-term infants. Pediatr Cardiol 2019;40:1645-54. [Crossref]
11. Alzahrani AK. Cardiac function affection in infants with neonatal sepsis. J Clin Trials 2017;7. [Crossref]
12. Cai S, Thompson DK, Anderson PJ, Yang JY-M. Short-and long-term neurodevelopmental outcomes of very preterm infants with neonatal sepsis: a systematic review and meta-analysis. Children 2019;6:131. [Crossref]
13. Zaky A, Deem S, Bendjelid K, Treggiari MM. Characterization of cardiac dysfunction in sepsis: an ongoing challenge. Shock 2014;41:12-24. [Crossref]
14. Drosatos K, Khan RS, Trent CM, Jiang H, Son NH, Blaner WS, et al. Peroxisome proliferator-activated receptor-γ activation prevents sepsis-related cardiac dysfunction and mortality in mice. Circ Heart Fail 2013;6:550-62. [Crossref]
15. de Montmollin E, Aboab J, Mansart A, Annane D. Bench-to-bedside review: β-adrenergic modulation in sepsis. Crit Care 2009;13:230. [Crossref]
16. Kundak AA, Dilli D, Karagöl B, Karadag N, Zenciroğlu A, Okumuş N et al. Non benign neonatal arrhythmias observed in a tertiary neonatal intensive care unit. Indian J Pediatr2013;80:555-9. [Crossref]
17. Turner CJ, Wren C. The epidemiology of arrhythmia in infants: a population-based study. Jof Paediatr Child Health 2013;49:278-81. [Crossref]
18. Badrawi N, Hegazy RA, Tokovic E, Lotfy W, Mahmoud F, Aly H. Arrhythmia in the neonatal intensive care unit. Pediatr Cardiol 2009;30:325-30. [Crossref]
19. Ban JE. Neonatal arrhythmias: diagnosis, treatment, and clinical outcome. Korean J Pediatr 2017;60:344-352. [Crossref]
20. Chen SC, Su HM, Huang JC, Chang K, Tsai YC, Chen LI, Chang JM, Hwang SJ, Chen HC. Association of P-Wave Dispersion with Overall and Cardiovascular Mortality in Hemodialysis Patients. Am J Nephrol 2015;42:198-205. [Crossref]
21. Kocaoglu C, Sert A, Aypar E, Oran B, Odabas D, Arslan D, et al. P-wave dispersion in children with acute rheumatic fever. Pediatr Cardiol 2012;33:90-4. [Crossref]
22. Turhan H, Yetkin E, Atak R, Altinok T, Senen K, Ileri M, et al. Increased P-wave duration and P-wave dispersion in patients with aortic stenosis. Annals of Noninvasive Electrocardiology 2003;8:18-21. [Crossref]
23. Ozdemir R, Isguder R, Kucuk M, Karadeniz C, Ceylan G, Katipoglu N, et al. A valuable tool in predicting poor outcome due to sepsis in pediatric intensive care unit: Tp-e/QT ratio. J Trop Pediatric 2016;62:377-84. [Crossref]
24. Paroxysmal atrial fibrillation in critically ill patients with sepsis. J Intensive Care Med 2008;23:178-83. [Crossref]
25. Klein Klouwenberg PM, Frencken JF, Kuipers S, Ong DS, Peelen LM, van Vught LA, et al. Incidence, predictors, and outcomes of new-onset atrial fibrillation in critically ill patients with sepsis. A cohort study. Am J Respir Crit Care Med 2017;195:205-11. [Crossref]
26. Meierhenrich R, Steinhilber E, Eggermann C, Weiss M, Voglic S, Bögelein D, et al. Incidence and prognostic impact of new-onset atrial fibrillation in patients with septic shock: a prospective observational study. Critical Care 2010;14:1-8. [Crossref]
27. Wynn JL, Wong HR, Shanley TP, Bizzarro MJ, Saiman L, Polin RA. Time for a neonatal-specific consensus definition for sepsis. Pediatr Crit Care Med 2014;15:523-8. [Crossref]
28. Suzuki T, Suzuki Y, Okuda J, Kurazumi T, Suhara T, Ueda T, et al. Sepsis-induced cardiac dysfunction and β-adrenergic blockade therapy for sepsis. J Intensive Care 2017;5:1-10. [Crossref]
29. Sullivan BA, McClure C, Hicks J, Lake DE, Moorman JR, Fairchild KD. Early heart rate characteristics predict death and morbidities in preterm infants. J Pediatr 2016;174:57-62. [Crossref]
30. Fairchild KD, Aschner JL. HeRO monitoring to reduce mortality in NICU patients. Res Repin Neonatol 2012:65-76. [Crossref]