ORIGINAL RESEARCH ARTICLE

The Relationship Between Sleep Disorders and Congenital Heart Disease in Children

Erling D Kaunang[1], Praevilia M Salendu[2], Angelina F Wulur[3*]

[1,2,3]Department of Pediatric, Prof. R. D. Kandou General Hospital, Manado, Indonesia

DOI: 10.55489/njcm.161220255897

ABSTRACT

Background: Congenital heart disease (CHD) is the most prevalent congenital abnormality in humans. Children with CHD may have poor sleep quality owing to hypoxia, cyanosis, and circulatory irregularities. However, there is insufficient data accessible, notably in Indonesia. The purpose of this study was to evaluate the association between sleep problems and congenital heart disease in children.

Methods: A cross-sectional study was conducted from November 2024 to March 2025 at Prof. Dr R. D. Kandou General Hospital. Children aged 1 to <18 years with echocardiographically confirmed CHD were assessed using actigraphy and validated questionnaires. Sleep metrics measured were sleep efficiency, total sleep time, sleep latency, and waking after sleep onset (WASO). Bivariate and multivariate analysis yielded significant results ($p < 0.05$).

Results: A total of 35 children with CHD were included (median age 3 years; 60% female). Sleep disturbances were highly prevalent, with 94% of children exhibiting at least one abnormal parameter. Multivariate analysis showed that female sex was independently associated with shorter total sleep time ($\beta = -2.14$ hours; $p < 0.05$; 95%CI = -3.18 - (-1.09)), and older age was associated with fewer total awakenings ($\beta = -0.89$; $p = 0.01$; 95%CI = -1.50 - (-0.28)).

Conclusion: Sleep difficulties are quite common in children with CHD, especially among females, underweight youngsters, and those with cyanotic abnormalities.

Keywords: Actigraphy, Congenital Heart Disease, Cross-Sectional Study, Indonesia, Pediatric Cardiology, Sleep Disorder

ARTICLE INFO

Financial Support: None declared

Conflict of Interest: The authors have declared that no conflict of interests exists.

Received: 12-08-2025, **Accepted**: 01-11-2025, **Published**: 01-12-2025

***Correspondence:** Angelina Fransisca Wulur (Email: angelina94wulur@yahoo.com)

How to cite this article: Kaunang ED, Salendu PM, Wulur AF. The Relationship Between Sleep Disorders and Congenital Heart Disease in Children. Natl J Community Med 2025;16(12):1196-1202. DOI: 10.55489/njcm.161220255897

www.njcmindia.com | pISSN: 0976-3325 | eISSN: 2229-6816 | Published by Medsci Publications

INTRODUCTION

Congenital heart disease (CHD) affects roughly one out of every 125 babies and is the most common congenital abnormality in humans, causing considerable morbidity and death globally.[1] Approximately one-third of infants with CHD have complicated abnormalities, which are a combination of excessive volume and pressure that may compromise systemic and pulmonary circulation.[2] Although CHD is the primary cause of morbidity and death in childhood, more than 90% of children with CHD now survive into adulthood because to considerable developments in disease detection and improvements in medical and surgical care.[3]

CHD may have an impact on a child's sleep quality. According to its mechanism, there is an interruption in the flow of oxygen intake and distribution to the foetus after birth. Congenital heart disease may lead to a reduction in cardiac output and heart failure. Furthermore, systemic hypoxia and cyanosis, induced by the mixing of oxygenated and deoxygenated blood via the systemic circulation, often occur in individuals with CHD.[4] The occurrence of hypoxemia can increase peripheral chemoreflex sensitivity and trigger chronic sympathetic nerve activation, which can lead to sleep-disordered breathing (SDB) and subsequently cause sleep disruption, increased nighttime blood pressure, and sympathetic nerve activation.[5,6] This cycle worsens autonomic tone imbalance and can worsen the sleep quality of children with CHD. Sleep disorders in children with CHD can occur due to various causes, including respiratory problems, physical discomfort, anxiety, and stress related to their health condition. Chronic sleep disorders in children can affect physical, cognitive, and emotional development, as well as reduce overall quality of life.[7] We hypothesised that cyanotic CHD and malnutrition are associated with poorer sleep quality.

To yet, epidemiological data describing the link between CHD and sleep disturbances in children are quite scarce. In a small prospective study, 79% of infants with CHD evaluated for heart surgery were found to have comorbid sleep-disordered breathing.[7] There was also limited evidence on sleep quality in Indonesian children with CHD measured using actigraphy. Therefore, this research aims to examine the incidence and determinants of sleep disruptions among children with CHD using actigraphy. This research assessed sleep efficiency, sleep latency, total sleep duration, wake after sleep initiation, and total waking time in paediatric patients with CHD.

METHODOLOGY

This cross-sectional research was undertaken in the inpatient ward of Prof. Dr. R. D. Kandou General Hospital from November 2024 to March 2025.

Sample Size: The analytical correlation formula was α = 0.05 (two-tailed; $Z_{1-\alpha/2}$=1.96), power = 80% (Z_{β} =0.84), and an estimated correlation coefficient (r) of 0.4 based on previous studies. This yielded a minimum required sample of 30 participants. The assumed correlation coefficient (r = 0.4) was based on prior pediatric sleep research reporting moderate correlations between sleep parameters and behavioural or clinical outcomes.[8] This value represents a moderate effect size according to Cohen's classification. By using r = 0.4, the sample size was designed to guarantee adequate statistical power (80%) to discover a modest connection, so balancing the risk of Type I and Type II errors in this investigation, which included 35 participants. A sequential sampling technique was utilised to recruit all eligible individuals.

Over the course of the study, from November 2024 to March 2025, a total of 90 children were registered in the inpatient ward of Prof. Dr R. D. Kandou General Hospital. Of these, 75 children were assessed for eligibility to participate in the study. Out of the 75 youngsters tested, 35 were included. A total of 40 children were excluded for not meeting the inclusion criteria or other relevant reasons for exclusion. There were no missing data among the included participants, and therefore, the final data analysed consisted of 35 children, with complete and valid data for further analysis.

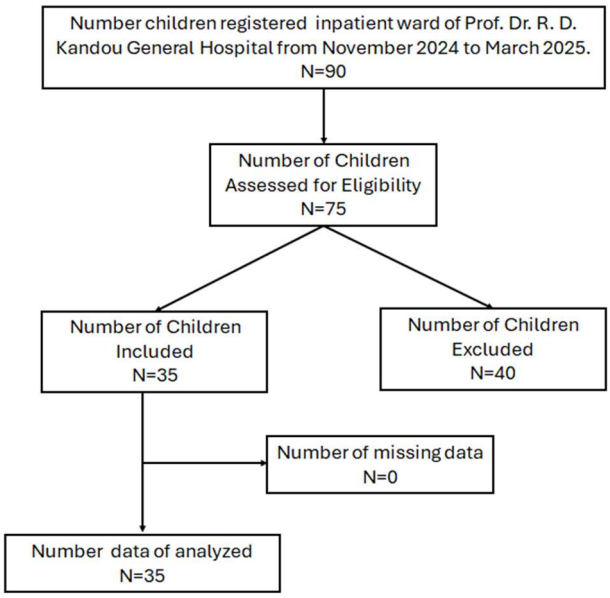

Figure 1: Participant flow diagram depicting the selecting procedure for children

Each participant's data was thoroughly reviewed to ensure completeness. In cases where missing data were identified, whether in actigraphy measurements or clinical data (e.g., age, gender, CHD type, or nutritional status), those participants were excluded from the analysis. To maintain the minimum required sample size of 35, new participants were enrolled to replace any excluded samples, ensuring the

final analysis included a complete dataset without compromising the integrity of the study. This approach ensured that missing data did not affect the overall sample size or the study's power. However, all 35 participants had complete actigraphy recordings and clinical data, and no missing data were reported in this study.

Study Population: This study included all children with congenital heart disease aged one year 1 year to <18 years Meanwhile, the exclusion criteria for this study include: 1) Children with other significant medical conditions besides congenital heart disease that may affect sleep (e.g., heart failure, neurological disorders, respiratory disorders (asthma, cystic fibrosis, interstitial lung disease), or obesity), 2) Children with previously diagnosed sleep disorders, and 3) Children who cannot use the actigraph device (device size is not suitable, have wounds on the limbs, or are allergic to the actigraph strap material).

Variables and Measurement: This study evaluated patient demographic parameters, including age, gender, nutritional status, type of CHD, size of CHD defect, duration of CHD diagnosis, as well as sleep disturbance parameters, including sleep efficiency, total sleep time, wake after sleep onset, and sleep latency. Demographic data were obtained from medical records, while sleep disorder data were obtained from actigraphy measurements that were placed on the wrist. This study has received ethical clearance from Prof. Dr R. D. Kandou General Hospital Ethics Commission with number No. 003/EC/KEPK-KANDOU/I/2025. All methods followed the ethical norms specified in the Declaration of Helsinki. All children participating in the research provided informed permission via their parents or legal guardians. The informed consent form included thorough information on the study's objectives, methods, possible hazards, and voluntary participation. All participants were informed that their information would be kept private and that they may withdraw from the research at any moment with no ill effects.

Actigraphy measures determine sleep efficiency, which is the entire interval between sleeping and bedtime (cutoff <85%). Total sleep time was defined as the duration between sleep onset and final awakening recorded each day. Wake after sleep onset represented the mean period of wakefulness occurring between episodes of sleep, with a cutoff value of more than 40 minutes. Sleep latency was defined as the interval from lying down in bed to the onset of sleep, with a cutoff value exceeding 20 minutes. Actigraphy monitoring was conducted for 7 consecutive nights on each participant to evaluate their sleep quality. Each child wore an actigraphy device on their wrist at night, from 8 p.m. to 7 a.m., to ensure representative data on their sleep patterns during the monitoring period.

Nutritional status is determined based on height-for-weight (H/W), which is then plotted using the WHO curve for children under five years old, and the CDC curve for children over five years old. A value below -2 is categorized as malnutrition, while a value of -2 or above is categorized as non-malnutrition.

Data Analysis: The SPSS program was used for data analysis, which included descriptive statistics to characterize the demographic features of patients, including age, gender, nutritional status, and duration of CHD diagnosis. This was followed by bivariate analysis between age, gender, type of CHD, and size of CHD defect with sleep quality assessed for numerical data, use the Independent T-Test or Mann-Whitney, while for categorical data, use the Chi-square or Fisher's exact test. Linear regression was employed to conduct a multivariate study of variables related to sleep quality. Data were considered significant if the p-value was <0.05.

Model diagnostics were conducted to assess the adequacy of the regression analyses. Normality of residuals and homoscedasticity were verified graphically. Although the Variance Inflation Factor (VIF) was not formally calculated, the included predictors (age, sex, nutritional status, and defect size) were chosen based on their clinical distinctiveness and lack of strong pairwise correlations (Pearson's r <0.6), suggesting a low risk of multicollinearity. Variable selection for the multivariate model followed both clinical relevance and univariate significance threshold (p <0.25) to prevent overfitting, given the limited sample size (n = 35). All analyses were done using SPSS ver 22 with significant data if the p-value was <0.05.

Ethical Approval: This study has been approved by the Ethics Committee of Prof. R. D. Kandou General Hospital, Manado, North Sulawesi, with ethical clearance number (003/EC/KEPK-KANDOU/I/2025).

RESULTS

A total of 35 children out of 40 eligible children were enrolled in this study, excluding 5 children due to incomplete data. Female participants slightly outnumbered males, with a ratio of 60:40 (Table 1). The median age was three years, with the majority of youngsters ranging from one to slightly more than six. Nearly half of the participants were aged 1-2 years, while older age groups were less represented. Nineteen children (54%) were underweighted, and 17 (49%) had short stature. This high proportion of low weight and height reflects the prevalence of malnutrition, observed in 54% of the sample. Most CHD diagnoses had been established about two years earlier. Acyanotic CHD was the most common type, found in 74% of cases. The median defect size was 4 mm, ranging from 3 mm to 7 mm.

Table 2 shows the distribution of sleep quality parameters in the study. These consist of sleep latency, sleep efficiency, total sleep time, waking after sleep, and total awakenings. The sleep quality parameter was gathered through actigraphy monitoring of each participant in seven consecutive nights of valid actigraphy data, which were averaged for analysis.

Table 1: Characteristics of the subjects

Characteristics	Participants (%)
Age (year)	3.0 (1.0; 6.5)*
1-2	17 (48.6)
3-5	8 (22.9)
6-12	4 (11.4)
13-17	6 (17.1)
Gender	
Male	14 (40.0)
Female	21 (60.0)
Body weight (kg)	11.5 (7.8; 19.0)*
Body weight category	
Normal	16 (45.7)
Underweight	19 (54.3)
Body height (cm)	94.0 (72.8; 121.0)*
Body height category	
Normal	18 (51.4)
Short	17 (48.6)
Nutritional status	
Non-Malnutrition	16 (45.7)
Malnutrition	19 (54.3)
Type of CHD	
Acyanotic	26 (74.3)
Cyanotic	9 (25.7)
CHD defect size (mm)	4.0 (3.0; 7.0)*
Length of time diagnosed with CHD (years)	2.0 (1.0; 3.0)*

Note: Q1 = first quartile, Q3 = third quartile
*Values are in Median (Q1; Q3)

The median sleep efficiency was 74.4% (IQR 58.6-82.3), indicating moderate sleep fragmentation. The median total sleep time was 5.0 hours (IQR 4.0-6.0), substantially below the recommended duration for children, while the median wake-after-sleep onset (WASO) of 63.0 minutes suggested frequent nocturnal awakenings. In total, 94% of children exhibited at least one abnormal sleep parameter, confirming the high prevalence of sleep disturbances among the study cohort.

Table 3 presents the relationship between CHD characteristics and sleep quality parameters measured by actigraphy. For clarity, the analysis focuses on variables included in the multivariate model. Age, sex, nutritional status, CHD type, and duration since diagnosis were identified as potential factors influencing sleep quality. However, only sex and age showed significant associations.

Female sex was associated with a significantly shorter total sleep time (β = -2.14 hours; 95% CI -3.18 to -1.09; p <0.05), while older age correlated with fewer total awakenings (β = -0.89; 95% CI -1.50 to -0.28; p = 0.01). Malnutrition showed a borderline association with longer total sleep duration (β = 1.30; 95% CI 0.04 to 2.56; p = 0.05) (Table 3).

Table 2: Comparing sleep quality in children with cyanotic and acyanotic CHD

Sleep Quality Parameter	All subjects (N = 35)	Acyanotic (n = 26)	Cyanotic (n = 9)	P*
Sleep Latency (minutes) (Median (Q_1-Q_3))	0.0 (0.0 - 14.5)	0.0 (0.0 - 14.8)	0.0 (0.0 - 11.0)	0.950
≤20 minutes (n (%))	27 (77.1)	20 (76.9)	7 (78.8)	0.999
>20 minutes (n (%))	8 (22.9)	6 (23.1)	2 (22.2)	
Sleep Efficiency (%), (Mean (SD))	74.4 (14.1)	74.3 (15.8)	74.8 (7.8)	0.928
≥ 85 (n (%))	10 (28.6)	9 (34.6)	1 (11.1)	0.364
< 85 (n (%))	25 (71.4)	17 (65.4)	8 (88.9)	
Total Sleep Time (hours) (Median (Q_1-Q_3))	5.0 (4.0 - 6.0)	5.0 (4.0 - 6.0)	5.0 (5.0 - 6.0)	0.861
Sufficient (n (%))	2 (5.7)	2 (7.7)	0 (0)	0.999
Insufficient (n (%))	33 (94.3)	24 (92.3)	9 (100)	
Wake After Sleep Onset (Median (Q_1-Q_3))	63.0 (38.5 - 134.0)	58.0 (38.2 - 157.2)	63.0 (52.0 - 101.0)	0.999
≤40 (n (%))	10 (28.6)	8 (30.8)	2 (22.2)	0.977
>40 (n (%))	25 (71.4)	18 (69.2)	7 (77.8)	
Total Awakening, (Mean (SD))	20.5 (9.6)	20.2 (9.3)	21.3 (10.7)	0.770
Sleep disturbance (3)† (n (%))	27 (77.1)	19 (73.1)	8 (88.9)	
Sleep disturbance (4)‡ (n (%))	33 (94.3)	24 (92.3)	9 (100)	

Note: Q1 = first quartile, Q3 = third quartile. *p-values derived from the Independent T-Test or Mann-Whitney test for numerical variables and the Chi-square test or Fisher's Exact test for categorical variables. SD = standard deviation. The classification of 'sleep disturbance: (3)† is based on three actigraphy parameters-sleep latency > 20 minutes, sleep efficiency < 85%, and wake after sleep onset > 40 minutes. (4)‡ Based on the criterion of age-appropriate sleep duration. NS: not significant (p-value >0.99)

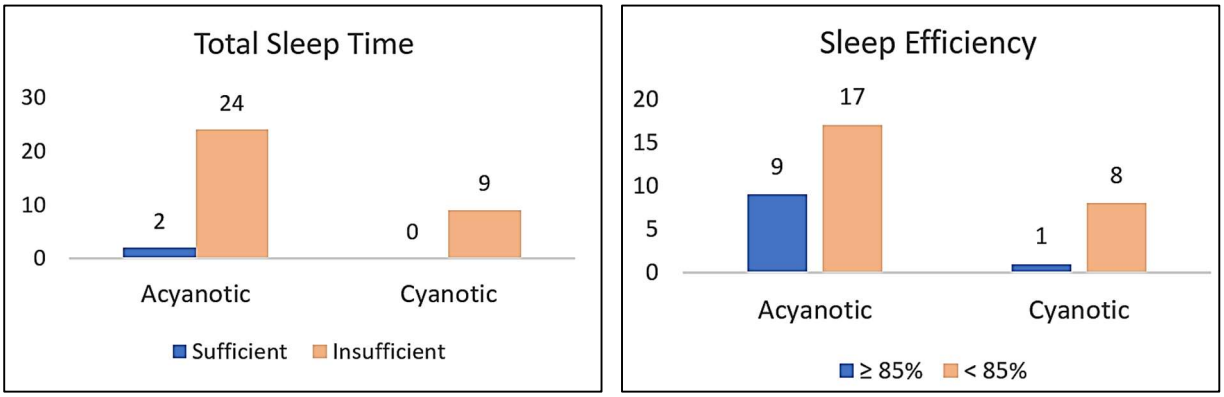

Figure 1: Total Sleep Time and Sleep Efficiency by CHD Type

Table 3: Linear regression analysis of sleep quality using actigraphic parameters

Variable	Univariate		Multivariate	
	β (95% CI)	p	β (95% CI)	p
Sleep Latency				
Female vs male	13.44 (-5.42 - 32.30)	0.16	17.02 (-3.06 - 37.10)	0.11
Age	0.05 (-1.74 - 1.85)	0.95	-1.44 (-3.52 - 0.65)	0.19
Length of time diagnosed with CHD	2.09 (-0.71 - 4.89)	0.14	2.27 (-0.72 - 5.25)	0.15
Short stature	-8.96 (-32.58 - 14.65)	0.45	-17.99 (-43.62 - 7.64)	0.18
Sleep Efficiency				
Female vs male	-6.40 (-16.15 - 3.35)	0.19	-8.25 (-18.31 - 1.81)	0.12
Age	0.54 (-0.36 - 1.44)	0.23	0.87 (-0.08 - 1.82)	0.09
Malnutrition	5.08 (-6.47 - 16.62)	0.38	9.07 (-3.25 - 21.38)	0.16
Total Sleep Time				
Female vs male	-1.43 (-2.61 - (-0.25))	**0.02**	-2.14 (-3.18 - (-1.09))	**< 0.05**
Age	0.01 (-0.11 - 0.13)	0.85	0.07 (-0.03 - 0.17)	0.15
Size of CHD	-0.07 (-0.28 - 0.13)	0.47	-0.14 (-0.30 - 0.02)	0.09
Malnutrition	1.37 (-0.05 - 2.79)	0.06	1.30 (0.04 - 2.56)	0.05
Wake After Sleep Onset				
Female vs male	30.66 (-18.92 - 80.24)	0.22	40.29 (-9.61 - 90.18)	0.12
Length of time diagnosed with CHD	-5.81 (-13.08 - 1.46)	0.11	-6.82 (-13.95 - 0.32)	0.07
Total Awakening				
Age	-0.53 (-1.13 - 0.07)	0.08	-0.89 (-1.50 - (-0.28))	**0.01**
Short stature	-1.79 (-10.10 - 6.53)	0.67	-7.47 (-15.49 - 0.56)	0.08

Note: CI: confidence interval. SD = standard deviation. CHD = congenital heart disease

DISCUSSION

Congenital heart disease (CHD) is commonly detected during the neonatal period, particularly when symptoms like cyanosis or congestive heart failure manifest early. In this study, the median age at diagnosis was 3.0 years, consistent with previous findings showing that 86.7% of CHD cases are identified within the first year of life.[9] Amelia P et al also reported that 48.1% of CHD patients were aged 0-2 years, aligning with the current study, where most cases (49%) occurred at 1-2 years.[10] Gender distribution in CHD varies by defect type. Sixty per cent subjects of this study were female. Previous study found higher atrial septal defect (ASD) prevalence in females and ventricular septal defect (VSD) in males.[11]

Cyanotic CHD is more often associated with growth impairment due to chronic hypoxemia and increased basal metabolism. Up to 55% of CHD patients may be malnourished, adversely affecting prognosis.[12] In cyanotic cases, children were more likely to be severely underweight and stunted compared to those with acyanotic defects.[10] Fifty-four per cent of subjects had nutritional issues, supporting previous reports of increased energy expenditure in CHD children.[13] Acyanotic CHD was more frequent (74%) than cyanotic CHD (26%), which is consistent with previous investigations.[10,14] Defect size also influences clinical outcomes. The median defect size was 4.0 mm (IQR: 3.0-7.0 mm). Small acyanotic defects may close spontaneously, while larger ones (>5 mm) pose risks of heart failure and pulmonary hypertension.[15] Given that growth and nutritional problems are common in CHD, another underrecognized issue is sleep disturbance, which may further affect overall development and psychosocial well-being.

This study found that 94% (33/35) of children with CHD experienced sleep disturbances, a notably high prevalence that should be interpreted with caution. This may partly reflect measurement artefacts from actigraphy or environmental influences such as hospital noise, light exposure, or nighttime clinical monitoring rather than true sleep pathology. Combs D et al. reported a 57% rate of sleep disorders, mainly obstructive sleep apnea (OSA), among children with CHD.[16] Sleep latency in this study had a median of 0.0 minutes (IQR 0.0-14.5), which appears physiologically implausible. This likely reflects actigraphy's tendency to misclassify motionless wakefulness as sleep onset, particularly in hospitalised or fatigued children. Previous research indicates that short latency may represent either efficient sleep initiation or compensatory hypersomnolence due to chronic fatigue or hypoxemia.[17]

Sleep efficiency averaged 74.4 ± 14.1%, below the 85% threshold for good sleep quality. This indicates fragmented sleep in CHD patients, potentially caused by persistent hypoxia or severe cardiac pathology. Reduced efficiency is consistent with persistent hypoxia, autonomic imbalance, or sleep-disrupting medications. Dai et al. also observed decreased sleep efficiency in CHD children, particularly cyanotic types.[18] The mean total sleep time was 5.0 hours (IQR 4-6), significantly lower than the recommended 9-12 hours for children aged 4-12. Previous studies by Sadhwani recorded an average of 12.4 hours of total sleep in CHD patients.[19] Median Wake After Sleep Onset (WASO) was 63.0 minutes (range: 38.5-134.0), indicating frequent nighttime awakenings.[20]

Physiological and psychosocial mechanisms contribute to sleep disorders in CHD. Hypoxemia, autonomic imbalance, cardiac workload, and medications like diuretics or beta-blockers interfere with sleep.[21-25] Beyond these physiological causes, psychosocial stressors such as anxiety, depression, and caregiver

burden are also important but were not measured in this study. Hamdani AR et al. reported elevated anxiety and depressive symptoms in CHD children.[26] The mean number of total awakenings was 20.5 ± 9.6 per night, indicating highly fragmented sleep. This negatively affects development. Caissie et al. confirmed more awakenings in CHD children, which correlated with poorer adaptive functioning.[27]

Although female participants appeared to have higher odds of sleep disturbances (OR = 2.14, p <0.05), this association should be interpreted cautiously. The small sample size and lack of multivariable adjustment may overestimate the strength of this relationship. This is consistent with research demonstrating increased anxiety and emotional difficulties in females with CHD, increasing their likelihood of sleep disturbances.[26] Emotional vulnerability and psychological distress may explain the greater prevalence in females. It was found that older age was associated with nearly one fewer total awakening (p=0.01), indicating that the relationship between age and sleep disorders is statistically significant. This aligns with previous research by Dai et al., who found that younger children with PJB more frequently experience sleep fragmentation due to autonomic nervous system instability, making them more susceptible to changes in heart rate and blood pressure during sleep.[18]

Given the significant frequency of sleep disruptions in children with CHD found in this research, routine screening for sleep problems should be considered as part of comprehensive CHD management. Early detection through actigraphy or structured questionnaires can enable timely intervention, potentially improving both neurodevelopmental and cardiovascular outcomes.

Strengths and Limitations

The strengths of this study include the use of actigraphy, a novel and objective method for assessing sleep disturbances in children with CHD, which provides continuous and accurate data. Additionally, the study offers valuable local insights, as there is limited data on this topic from Indonesia. However, the study has several limitations. It lacks a control group, which limits the ability to compare the results to a healthy pediatric population. The tiny sample size may limit the generalisability of the results. Furthermore, data on oxygenation levels and medication use, which could help clarify the physiological factors affecting sleep, were not collected. The inpatient setting of the study may not fully reflect the sleep disturbances experienced by children in a community-based setting, limiting the broader applicability of the results.

Conclusion

Sleep disturbances were highly prevalent among children with congenital heart disease, with female sex, malnutrition, and cyanotic CHD showing associations with poorer sleep quality. These findings suggest that routine screening for sleep problems should be integrated into pediatric cardiology practice. Early identification and management of sleep disturbances may help improve neurobehavioral outcomes, emotional well-being, and overall QOL in children with CHD. Future research with larger, longitudinal cohorts incorporating both physiological (e.g., oxygenation profiles, medication use) and psychosocial assessments (e.g., anxiety, depression, caregiver stress) is warranted to clarify underlying mechanisms and guide targeted interventions.

Acknowledgement: The writers like to convey their profound thanks to the Department of Child Health, Faculty of Medicine, Sam Ratulangi University, and Prof. R. D. Kandou General Hospital, Manado, North Sulawesi, for their invaluable support in facilitating this research. We also extend our appreciation to the pediatric cardiology and pediatric ward staff for their assistance in patient recruitment and data collection. Special thanks are given to the participating children and their families for their cooperation and willingness to be part of this study.

Individual Authors' Contributions: EDK contributed to the study design, data collection, and statistical analysis. **PMS** was involved in statistical analysis, data interpretation, manuscript preparation, and literature search. **AFW** participated in data collection, statistical analysis, manuscript preparation, and literature search.

Availability of Data: The datasets during the current study are available from the corresponding author upon reasonable request.

Declaration of No use of generative AI tools: This manuscript was prepared and finalised entirely by the authors, without the use of any automated or algorithm-based content generation tools.

References

1. Clausen H, Norén E, Valtonen S, Koivu A, Sairanen M, Liuba P. Evaluation of Circulating Cardiovascular Biomarker Levels for Early Detection of Congenital Heart Disease in Newborns in Sweden. JAMA Netw Open 2020;3(12):e2027561. DOI: https://doi.org/10.1001/jamanetworkopen.2020.27561
2. Nakano SJ, Miyamoto SD, Price JF, Rossano JW, Cabrera AG. Pediatric Heart Failure: An Evolving Public Health Concern. J Pediatr 2020;218:217-221. DOI: https://doi.org/10.1016/j.jpeds.2019.09.049 PMid:31740144 PMCid:PMC7662928
3. Yasuhara J, Garg V. Genetics of congenital heart disease: a narrative review of recent advances and clinical implications. Transl Pediatr 2021;10(9):2366-2386. DOI: https://doi.org/10.21037/tp-21-297 PMid:34733677 PMCid:PMC8506053
4. Ykeda DS, Lorenzi-Filho G, Lopes AAB, Alves RSC. Sleep in Infants with Congenital Heart Disease. Clinics 2009;64(12):1205-1210. DOI: https://doi.org/10.1590/S1807-59322009001200011 PMid:20037709 PMCid:PMC2797590
5. Schlatterer SD, Govindan RB, Murnick J, Barnett SD, Lopez C, Donofrio MT, et al. In infants with congenital heart disease, au-

tonomic dysfunction is associated with pre-operative brain injury. Pediatr Res 2022;91(7):1723-1729. DOI: https://doi.org/10.1038/s41390-021-01931-7 PMid:34963700

6. Baker-Smith CM, Isaiah A, Melendres MC, Mahgerefteh J, Lasso-Pirot A, Mayo S, et al. Sleep-Disordered Breathing and Cardiovascular Disease in Children and Adolescents: A Scientific Statement From the American Heart Association. J Am Heart Assoc. 2021 Sep 21;10(18):e022427. DOI: https://doi.org/10.1161/JAHA.121.022427 PMid:34404224
7. Combs D, Skrepnek G, Seckeler MD, Barber BJ, Morgan WJ, Parthasarathy S. Sleep-Disordered Breathing is Associated With Increased Mortality in Hospitalized Infants With Congenital Heart Disease. Journal of Clinical Sleep Medicine 2018;14(09):1551-1558. DOI: https://doi.org/10.5664/jcsm.7334 PMid:30176962 PMCid:PMC6134246
8. Kim H, Kim JH, Yi JH, Kim JY, Solmi M, Cortese S, et al. Correlations between sleep problems, core symptoms, and behavioral problems in children and adolescents with autism spectrum disorder: a systematic review and meta-analysis. Eur Child Adolesc Psychiatry 2024;33(5):1539-1549. DOI: https://doi.org/10.1007/s00787-023-02253-1 PMid:37477722
9. Al-Fahham MM, Ali YA. Pattern of congenital heart disease among Egyptian children: a 3-year retrospective study. The Egyptian Heart Journal 2021;73(1):11. DOI: https://doi.org/10.1186/s43044-021-00133-0 PMid:33512632
10. Amelia P, Yosephine AG, Tobing TC, Savira M, Viandy V, Inglin M. Association between type of congenital heart disease with child growth and development status: A cross-sectional study in Medan, Indonesia. Narra J 2023;3(3):e414-e414. DOI: https://doi.org/10.52225/narra.v3i3.414 PMid:38450335.
11. Alaani STA, Alkubaisy FS, Merie AA. Epidemiological and clinical aspects of congenital heart disease in Fallujah Maternity and Children Hospital, Iraq. Iraq Medical Journal 2023;7(2):46-49. DOI: https://doi.org/10.22317/imj.v7i2.1240
12. Yuruk E, Çetinkaya Ş. Growth and Development of Children with Congenital Heart Disease: Nutrition. Türkiye Sağlık Bilimleri ve Araştırmaları Dergisi 2023;6(3):53-66. DOI: https://doi.org/10.51536/tusbad.1221255
13. Abdur Rahman M, Ketut Alit Utamayasa I, Hidayat T, Irawan R, Elizabeth R. Anthropometric Profile of Children with Cyanotic and Noncyanotic Congenital Heart Disease. Media Gizi Indonesia. 2020;15(1):1-6. DOI: https://doi.org/10.20473/mgi.v15i1.1-6
14. Sherzad AG, Zalmai AS, Zafarzai I, Zazai MK, Zeng Q. Assessing the Frequency of Congenital Heart Diseases Among Children in Eastern Afghanistan. Pediatric Health Med Ther 2024;15:299-309. DOI: https://doi.org/10.2147/PHMT.S481934 PMid:39371077 PMCid:PMC11453131
15. Chowdhury MNI, Ullah MA, Hossain MB, Islam SMS. Children With Suspected Congenital Heart Disease - A Review. J Biosci (Rajshari) 2023;31(2):87-101. DOI: https://doi.org/10.3329/jbs.v31i2.74149
16. Combs D, Partha M, Hsu CH, Edgin J, Seckeler M, Klewer S, et al. 0484 Trouble Sleeping Predicts Future Decreased Quality of Life in Young Children with Fontan Circulation. Sleep 2022;45(Supplement_1):A214-A214. DOI: https://doi.org/10.1093/sleep/zsac079.481
17. Gaina A, Sekine M, Kanayama H, Sengoku K, Yamagami T, Kagamimori S. Short-long sleep latency and associated factors in Japanese junior high school children. Sleep Biol Rhythms 2005;3(3):162-165. DOI: https://doi.org/10.1111/j.1479-8425.2005.00185.x
18. Dai WS, Xie WP, Liu JF, Chen Q, Cao H. The Effect of Family-centered Care Model on Sleep Quality and Medication Compliance of Children With Simple Congenital Heart Disease After Transcatheter Intervention. Research Square 2021. Preprint. DOI: https://doi.org/10.21203/rs.3.rs-786722/v1
19. Sadhwani A, Butler S, Rofeberg V, Espinosa K, Wood L, Cassidy AR, et al. Sleep Patterns in Young Children with Congenital Heart Disease. J Pediatr 2023;252:198-203.e2. DOI: https://doi.org/10.1016/j.jpeds.2022.08.031 PMid:36029823
20. Combs D, Fernandez V, barber brent j, Morgan WJ, Hsu CH, Andrews J, et al. Abstract 14771: Obstructive Sleep Apnea is Associated With Cardiac Dysfunction in Children With Congenital Heart Disease. Circulation 2020;142(Suppl_3):A14771. DOI: https://doi.org/10.1161/circ.142.suppl_3.14771
21. Dudoignon B, Bokov P, Couque N, Denjoy I, Matrot B, Delclaux C. Neurogenic hypertension characterizes children with congenital central hypoventilation syndrome and is aggravated by alveolar hypoventilation during sleep. J Hypertens 2023;41(8):1339-1346. DOI: https://doi.org/10.1097/HJH.0000000000003475 PMid:37260279
22. Hamad AKS. Sleep Disturbance and the Risk of Cardiovascular Diseases. Journal of the Bahrain Medical Society 2023;35(1):57-63. DOI: https://doi.org/10.26715/jbms.35_1_7 PMid:26918091
23. Buchhorn R. Pathophysiology, Treatment and Long-Term Consequences of Heart Failure in Infancy. Journal of Biomedical Research & Environmental Sciences 2022;3(4):436-444. DOI: https://doi.org/10.37871/jbres1462
24. Damkjaer M, Urhoj SK, Tan J, Briggs G, Loane M, Given JE, et al. Prescription of cardiovascular medication in children with congenital heart defects across six European Regions from 2000 to 2014: data from the EUROlinkCAT population-based cohort study. BMJ Open 2022;12(4):e057400. DOI: https://doi.org/10.1136/bmjopen-2021-057400 PMid:35450908 PMCid:PMC9024225
25. Loomba RS, Rausa J, Dorsey V, Bronicki RA, Villarreal EG, Flores S. The impact of medical interventions on admission characteristics in children with congenital heart disease and cardiomyopathy. Cardiol Young 2021;31(3):406-413. DOI: https://doi.org/10.1017/S1047951120004175
26. Hamdani AR, Khawar R, Mughal AR. Gender Differences in Externalizing and Internalizing Behaviors Among Children and Adolescents with Congenital Heart Disease. Pakistan Journal of Social Research 2022;04(03):523-529. Available from: https://pjsr.com.pk/wp-content/uploads/2022/10/55.-Vol.-4.-Issue-3.-September-2022-Hamdani-Khawar-Mughal-Gender-Differences-in-externalizing-1.pdf
27. Caissie DM, Dollimount A, Tomczak CR, Erlandson MC, Pockett C, Adamko DJ, Olver TD, Switzer H, Wright KD. Adaptive Functioning and Sleep Quality: Associations in Young Children with Congenital Heart Disease. Pediatr Cardiol. 2025 Aug;46(6):1514-1522. DOI: https://doi.org/10.1007/s00246-024-03565-y PMid:38942985