SANAMED
2025; 20(2): 157–167
ISSN-1452-662X

DOI: 10.5937/sanamed0-57055
UDK: 616.8-009.832-053.2; 616.12-073-053.2
ID: 174824969
Original article

CARDIOLOGICAL DIAGNOSTIC APPROACH TO CHILDREN WITH CONSCIOUSNESS DISORDERS

Mišanović Verica,[1] Viteškić Selma,[2] Begić Nedim,[1] Mulać Ahmed,[1] Dizdar Selma,[1] Anić Duško,[1] Terzić Sabina,[1] Kovačević Prstojević Jelena,[3] Čengić Adisa,[1] Begić Zijo,[1] Užičanin Sajra,[1] Međedović Edin,[4] Selimović Amina[1]

[1] University Clinical Center Sarajevo, Pediatric Clinic, Sarajevo, Bosnia and Herzegovina
[2] Primary Health Care Center of Sarajevo Canton, Sarajevo, Bosnia and Herzegovina
[3] University Clinical Center Sarajevo, Clinic of Urology, Sarajevo, Bosnia and Herzegovina
[4] University Clinical Center Sarajevo, Clinic of Gynecology and Obstetrics, Sarajevo, Bosnia and Herzegovina

Primljen/Received: 25. 02. 2025. Prihvaćen/Accepted: 12. 07. 2025. Published online first 18. 07. 2025.

Abstract: Introduction: Syncope is one of the most common reasons for seeking medical attention in the pediatric population. The underlying etiology ranges from benign causes to potentially life-threatening conditions.

Objective: This cross-sectional retrospective study aimed to cardiologically evaluate children presenting with syncope and identify potential predictive parameters for cardiogenic syncope, the most dangerous type of syncope.

Patients and Methods: Data from 100 children aged 6 to 18 years who presented with syncope were retrospectively collected from medical records at the Pediatric Clinic of the Clinical Center of the University of Sarajevo between January 1, 2021, and December 31, 2022. Binary logistic regression was used to examine the predictive significance of the studied parameters.

Results: Of the 100 children with documented syncope, 71.0% were girls, with the peak incidence of syncope episodes occurring at age 15. There were no statistically significant differences in height, weight, or BMI between boys and girls. The most common cardiac diagnosis was sinus arrhythmia, while headache was the most frequent non-cardiac symptom. Seventy-three percent of patients experienced more than one syncope episode, with the highest percentage occurring at school. Prodromal symptoms were present in 87% of cases, whereas palpitations and chest pain prior to syncope were reported in 10% and 12% of cases, respectively. Among the 49 patients with abnormal ECG findings, 29% had sinus arrhythmia and 25% had incomplete right bundle branch block. The most common echocardiographic finding was mild pulmonary valve regurgitation, which is considered a physiological variant. Of all studied parameters, only EEG demonstrated significant predictive value for cardiogenic syncope ($p = 0.035$, $EXP(B) = 2.99$).

Conclusion: EEG findings have predictive significance for cardiogenic syncope in children. A borderline EEG increases the odds of cardiogenic syncope by approximately threefold.

Keywords: syncope, pediatric population, EEG, echocardiography, predictive parameters, clinical characteristics, electrocardiography.

INTRODUCTION

With the advancement of modern medicine, there has been significant progress in diagnosing and understanding the pathophysiological mechanisms underlying disorders of consciousness in adults. However, diagnosing and managing disorders of consciousness in children continue to pose challenges for clinicians and researchers (1, 2). This difficulty is partly due to the limited ability of the pediatric population to report on their consciousness (3 ,4).

Consciousness can be divided into two domains. The first domain is "wakefulness," referring to the level of consciousness or alertness. This component can be somewhat quantified using well-designed and universal methods that grade the level of consciousness. The second domain of consciousness, which is more resistant to quantification, is "awareness," referring to the content of consciousness. There is no objective way to quantify this component of consciousness. Disorders of

these two domains of consciousness are heterogeneous and numerous, with qualitative disorders falling more within the domain of psychology and psychiatry.

Syncope represents a transient loss of consciousness due to cerebral hypoperfusion, which can be benign but also indicative of more serious health problems. Syncope is a common problem in pediatrics. According to data from some hospitals, syncope is the most common paroxysmal non-epileptic event causing changes in consciousness in children (5).

The minimum duration of sudden cerebral blood flow reduction that causes syncope is 6 to 8 seconds. Syncope occurs when blood flow to the brain is compromised, resulting in symptoms such as nausea, dizziness, changes in visual fields, and ultimately loss of consciousness. Circulation does not necessarily need to be compromised for syncope to occur; sometimes the problem is a lack of oxygen or glucose in the blood.

Causes of inadequate blood flow to the brain can be different, and the most practical classification of syncope is based on etiology: neurological, orthostatic, and cardiovascular. In the diagnosis of syncope, it is essential to start with a well-constructed medical history, both personal and family. The physical examination that follows this initial step is perhaps the most important part of the diagnostic process as it can help exclude potentially life-threatening conditions. In addition, blood tests are necessary, paying attention to parameters related to red blood cells, electrolytes, and blood glucose levels (2).

An ECG (Electrocardiogram) is a necessary step in diagnostics and treating patients presenting with syncope, and in cases of suspected cardiac etiology of these syncope episodes, it is essential to continue with tests including echocardiography, continuous monitoring of cardiac rhythm, and possibly even cardiac enzymes. Ultimately, the tilt table test is indicated if patients present with recurrent episodes of syncope in the absence of a clear cardiac reason, in suspected vasovagal syncope, and orthostatic hypotensive syncope.

Although a wide range of diagnostic methods and tests are available for the diagnostic treatment of syncope, up to 45% of patients are discharged without a diagnosis. This speaks to the complexity of syncope, which requires multimodal approaches to treatment (6).

Neurological syncope is also known as neurocardiogenic or reflex syncope, and it includes vasovagal and situational syncope. Both occur due to inadequate cardiovascular reflexes that can lead to hypotension, bradycardia, or reduced cardiac output. The most common form of syncope in pediatric patients is vasovagal syncope. It is more common in children over 10 years old, with a predominance of females. It occurs after increased activation of the parasympathetic nervous system over the sympathetic nervous system, usually after exposure to a trigger. These triggers can be emotional stress, phobias, or trauma. Conditions that favor the occurrence of this response to a trigger are hunger and exhaustion. Before the onset of vasovagal syncope, children experience prodromal symptoms such as abdominal pain, dizziness, flushes of heat, or tunnel vision. Situational syncope occurs by a similar mechanism. The trigger is reduced preload, which causes stimulation of cardiac mechanoreceptors and subsequent activation of the parasympathetic nervous system. This happens in certain situations such as sneezing, coughing, micturition, defecation, after exercise, or lifting heavy weights. The diagnosis of neurological syncope is clinical and is based on the exclusion of other causes. Treatment is supportive, providing guidance and advice to prevent its occurrence. It is recommended to increase fluid and salt intake and assume a supine position in case of the onset of prodromal symptoms (2).

Orthostatic syncope occurs due to inadequate functioning of blood redistribution mechanisms and subsequent blood pressure drop upon changing from a sitting or supine position to a standing position. Normally, upon such a change in position, there is blood redistribution to the legs and splanchnic circulation. This causes decreased preload, and consequently, stroke volume, which activates a series of reflexes that increase sympathetic activity and normalize blood pressure. In cases where reflex mechanisms are compromised or hypovolemia is present, orthostatic syncope will occur. The diagnosis of orthostatic syncope is based on measuring orthostatic vital signs. There are discrepancies in defining abnormal vital signs that will be considered “positive.” However, the most accepted parameters for the diagnosis of orthostatic hypotension are a drop in systolic blood pressure of at least 20 mmHg or diastolic blood pressure of 10 mm Hg, measured within 3 minutes of changing from supine to standing position. The most common cause of orthostatic syncope in children is hypovolemia. In children, changes in blood pressure do not have a significant impact because they are not as pronounced. Instead, an increase in heart rate of over 20 beats per minute upon changing from supine to standing position is sensitive enough to detect hypovolemia. The diagnosis is made through a series of tests aimed at excluding other causes. A complete blood count may be done (to detect anemia), thyroid hormone levels (to detect thyrotoxicosis), or an ECG (to exclude cardiac arrhythmias). Finally, the tilt-table test is performed to confirm intolerance to postural changes (7).

The treatment is supportive and largely overlaps with the treatment of neurologically induced syncope.

Sufficient hydration and salt intake, education, and regular physical activity represent first-line treatments.

Cardiac syncope can be divided into two groups: obstructive and arrhythmogenic. Obstructive syncope corresponds to structural causes, while arrhythmogenic syncope corresponds to non-structural causes, as described in two studies (8).

Obstructive or structural causes can arise from intrinsic and extrinsic reasons. Intrinsic causes involve congenital heart diseases, aortic stenosis, or hypertrophic cardiomyopathy. Extrinsic causes act on the heart and include pulmonary hypertension or embolism.

Arrhythmogenic or non-structural syncope arises from disturbances in cardiac rhythm.

The most common structural causes of cardiovascular syncope are ischemic cardiomyopathy due to congenital anomalies of the coronary arteries, aortic stenosis, non-ischemic dilated cardiomyopathy, and hypertrophic obstructive cardiomyopathy.

It is not necessary for the heart to be morphologically altered for syncope to occur. Arrhythmias, defined as deviations from the normal rhythm of cardiac activity, can be divided into tachyarrhythmias and bradyarrhythmias, as well as a separate group of congenital channelopathies (8, 9).

There are numerous studies investigating these phenomena in children, but this is not the case in Bosnia and Herzegovina. Careful assessment and diagnostic evaluation of children presenting with this type of consciousness disorder can reveal much and potentially provide answers to questions crucial for further treatment of these children.

Cardiological evaluation is an essential aspect of conducting diagnostic assessments of children with consciousness disorders. Cardiologically induced syncope represents the most dangerous type of syncope, indicating some form of cardiac issue. This study recognizes this fact and is designed to evaluate the presence of possible cardiac or other diseases and to investigate the presence of predictors that could aid in diagnosing cardiologically induced syncope. This study has general and specific objectives.

The general objective is to evaluate cardiac and other findings in children with consciousness disorders aged 6 to 18 years.

Aims of the paper:

1. To examine the sociodemographic characteristics of children aged 6 to 18 years with consciousness disorders.

2. To individually assess predictors for syncope in children who have experienced consciousness disorders.

3. To determine which predictor is most dominant in consciousness disorders among children.

PATIENTS AND METHODS

This is a cross-sectional retrospective study aimed at evaluating the medical histories, anamnesis, and other results of diagnostic tests of children presenting with syncope episodes. Data were collected from the medical records of patients hospitalized at the Pediatric Clinic of the University Clinical Center in Sarajevo from January 2021 to December 2022.

Among all hospitalized children, subjects younger than 6 years old and those without complete findings and measured parameters relevant to this study were excluded. One hundred patients who met all criteria were included in the analysis.

In this study, we evaluated the medical histories, anamnesis, and results of various diagnostic tests in children presenting with syncope episodes. We collected demographic information including age, gender, weight, height, and body mass index (BMI), as these factors may influence the incidence and underlying causes of syncope. The medical history of each participant was examined carefully, with particular attention to family history of consciousness disorders, heart diseases, and sudden death. Comorbidities such as epilepsy or neurological disorders, which could potentially contribute to syncope, were also recorded.

Clinical parameters included blood pressure measurements (both systolic and diastolic), heart rate, and cardiac auscultation to identify any abnormalities, such as innocent systolic murmurs commonly found in children.

We included a variety of diagnostic tests to help determine the cause of syncope. These tests included:

1. Anamnestic data.

2. Blood pressure.

3. Heart rate.

4. ECG (Electrocardiogram) – The ECG was used to assess for conditions like sinus arrhythmia, incomplete right bundle branch block, and other abnormal findings that could indicate arrhythmias or other cardiac abnormalities.

5. Echocardiography– Performed to evaluate pulmonary valve regurgitation, which is a normal finding in children, as well as to screen for any structural heart abnormalities that could contribute to syncope episodes.

6. Tilt Table Testing – Used to assess the presence of vasovagal syncope and evaluate whether the syncope episodes had a cardiac origin.

7. Holter Monitoring – Involved continuous monitoring of the heart's electrical activity to detect ar-

rhythmias or other abnormal heart rhythms that could be linked to syncope.

8. EEG (Electroencephalogram) – Used to detect signs of cerebral hypoperfusion, which may suggest that the syncope is related to a neurological origin. EEG results were also assessed for borderline readings, which were found to be predictive of syncope related to cardiac disorders.

9. Blood Tests – If applicable, blood tests were conducted to rule out any metabolic or electrolyte imbalances that could contribute to the occurrence of syncope.

10. Each of these parameters was carefully evaluated to determine the underlying cause of syncope in our sample. The data were collected and analyzed to help differentiate between **cardiac** and **non-cardiac** causes of syncope in children, with particular emphasis on factors such as arrhythmias, structural heart diseases, and neurological factors.

Statistical Analysis

Upon completion of the study, statistical data processing was conducted. The statistical analysis of the obtained data was performed using SPSS software for Windows (version 19.0, SPSS Inc., Chicago, Illinois, USA) and Microsoft Excel (version 11, Microsoft Corporation, Redmond, WA, USA).

To determine the distribution of continuous variables, the Kolmogorov-Smirnov test was used (for samples larger than 50 subjects). For variables without statistically significant deviations from a normal distribution, mean values were presented as arithmetic means and standard deviations (SD). Parametric tests (one-sample t-test and paired t-test) were applied to compare these variables to reference values and repeated measurements, as well as to analyze trends in changes.

Variables showing statistically significant deviations from a normal distribution were presented using the median and interquartile range (25th–75th percentile). Non-parametric tests (Wilcoxon rank-sum test, Friedman test for repeated measurements) were used to compare these variables to reference values, repeated measurements, and trends in changes.

Logistic regression was employed to examine how independent predictor variables in the study influenced the outcome, specifically whether syncope was cardiologically induced or not.

A significance level of $\alpha = 0.05$ was used to determine statistical significance. Decisions regarding acceptance or rejection of hypotheses in the respective tests were based on the p-value (if $p \geq \alpha$, the hypothesis was accepted; if $p < \alpha$, the hypothesis was rejected).

RESULTS

In this sample, the girls had a mean age of 14.0 ± 2.7 years, while the boys had a mean age of 13.5 ± 2.9 years. There was no statistically significant difference in age between the groups ($p = 0.390$).

The average height of girls in this sample was 160.95 ± 11.8 cm, and the average height of boys was 161.24 ± 18.67 cm. There was no statistically significant difference in height between girls and boys ($p = 0.920$).The average weight of girls in this sample was 54.65 ± 14.78 kg, and the average weight of boys was 56.40 ± 19.15 kg. There was no statistically significant difference in weight between girls and boys ($p = 0.625$).The average BMI for girls in this sample was 20.76 ± 3.95 kg/m², and for boys, it was 21.01 ± 4.04 kg/m². There was no statistically significant dif-

Table 1. *Sexual structure of the sample*

	Frequency	Percentage	Cumulative Percentage
Female	71	71.0	71.0
Male	29	29.0	100.0
Total	100	100.0	

Table 2. *Average values of BMI, height and weight among the subjects*

	Sex	n	Mean value	Standard deviation (SD)	Standard error (SE)	p
Height (cm)	Female	71	160.95	11.786	1.399	0.920
	Male	29	161.24	18.671	3.467	
Weight (kg)	Female	71	54.65	14.786	1.755	0.625
	Male	29	56.40	19.158	3.557	
BMI (kg/m²)	Female	71	20.766	3.9583	0.4698	0.782
	Male	29	21.010	4.0480	0.7517	

Table 3. *Family history of consciousness disorders, heart diseases, and sudden death*

Family history	Outcome	Number (n)	Percentage (%)
Positive family history of the consciousness disorders	No	98	98.0
	Yes	2	2.0
Positive family history of heart disorders	No	83	83.0
	Yes	17	17.0
Positive family history of sudden death	No	98	98.0
	Yes	2	2.0

Table 4. *Age when the first syncope occurred*

	N	Min	Max	Mean value	Standard deviation (SD)
Age when the first syncope occurred	100	2	18	13.00	3.301

Table 5. *Blood pressure and heart rate values in the examined sample*

Variable		Systolic BP		Diastolic BP		Heart rate		
	Value	Number (n)	Percentage %	Number (n)	Percentage%	Value	Number (n)	Percentage %
	Low	25	25.0	23	23.0	Low	5	5.0
	Normal	73	73.0	64	64.0	Normal	67	67.0
	High	2	2.0	13	13.0	High	28	28.0

ference between the BMI values of girls and boys (p = 0.782).

A positive family history of consciousness disorders was present in 2.0% of the children, a positive family history of heart diseases in 17.0%, and a positive family history of sudden death in 2.0% (Table 3).

In addition to cardiological conditions, non-cardiological diseases were also examined. No non-cardiological disease was recorded in 45 children (45.0%), meaning that more than half (55.0%) of the children with documented syncope had at least one non-cardiological condition.

A total of 40 different non-cardiological diseases were recorded among the patients in this sample. The data show that the most common non-cardiological symptom among children with consciousness disorders was headache.

The earliest recorded age at which the first consciousness disorder occurred in these patients was 2 years, and the latest age was 18 years. The average age at which patients experienced a consciousness disorder was 13.0 ± 3.30 years (Table 4, Figure 1).

Normal systolic and diastolic blood pressure was observed in 73.0% and 64.0% of the subjects, respectively. Decreased systolic blood pressure was recorded

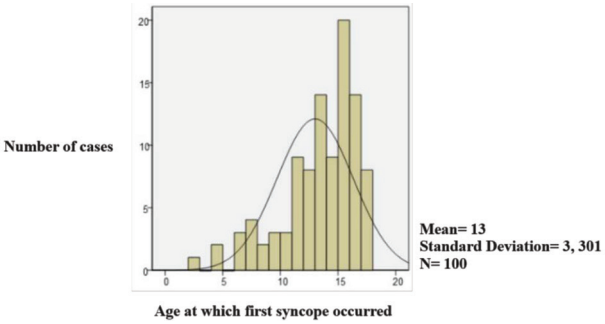

Figure 1. *Age distribution of subjects with consciousness disorders*

in 25.0% of the subjects, and decreased diastolic blood pressure in 23.0%. Elevated systolic blood pressure was noted in 2.0% of the children, while elevated diastolic blood pressure was seen in 13.0%.Normal heart rate was recorded in 67.0% of the subjects. Decreased heart rate was observed in 5.0%, and increased heart rate in 28.0% of the children (Table 5).

The average values of systolic blood pressure were 113.49 ± 13.42 mmHg (Figure 2).

The average values of diastolic blood pressure were 67.52 ± 10.18 mmHg.

The average heart rate was 94.66 ± 17.36 beats per minute (Figure 4).

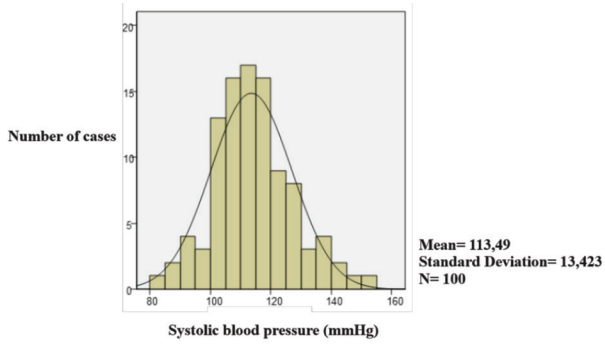

Figure 2. *Distribution of the systolic blood pressure value*

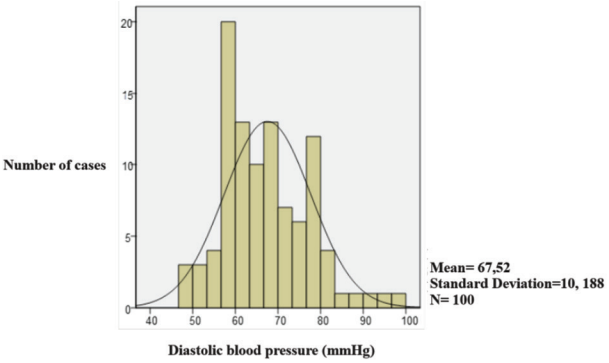

Figure 3. *Distribution of the diastolic blood pressure*

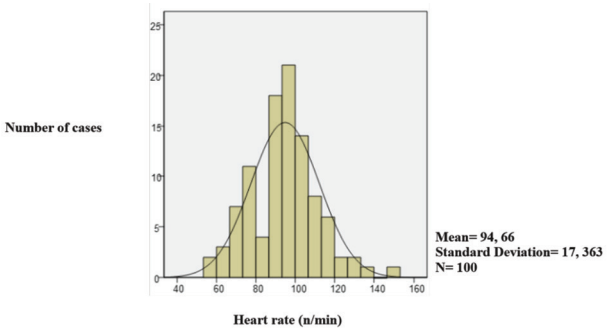

Figure 4. *Distribution of the heart rate*

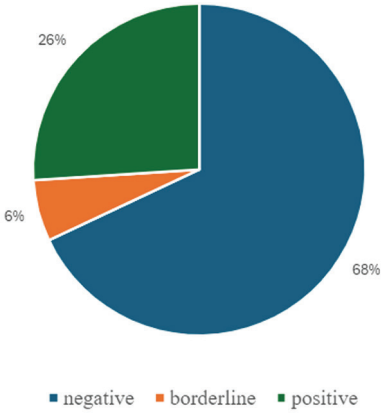

Figure 5. *Tilt table test*

Table 6. *The impact of sex, age, height, weight, and BMI on cardiologically caused syncope*

	B	S.E.	Wald	df	p.	Exp(B)
Sex	-0.378	0.474	0.638	1	0.425	0.685
Age	0.174	0.128	1.869	1	0.172	1.190
Height (cm)	-0.110	0.077	2.040	1	0.153	0.896
Weight (kg)	0.086	0.111	0.593	1	0.441	1.089
BMI (kg/m²)	-0.214	0.294	0.533	1	0.465	0.807
Constant	15.951	11.841	1.814	1	0.178	8459691.848

Table 7. *The impact of positive family history of consciousness disorders, heart diseases, and sudden death on cardiologically caused syncope*

		B	S.E.	Wald	df	p	Exp(B)
Step 1(a)	**Positive family history of consciousness disorders**	20.392	28420.722	0.000	1	0.999	718.511
	Positive family history of heart disorders	0.544	0.640	0.722	1	0.395	1.723
	Positive family history of sudden death	-0.811	1.537	0.279	1	0.598	0.444
	Constant	0.267	0.221	1.449	1	0.229	1.306

Out of 100 subjects, the tilt table test was normal in 68 (68.0%), borderline in 6 (6.0%), and positive in 26 (26.0%) (Figure 5).

Using binary logistic regression, we examined, in the following tables, the impact of independent predictors—variables in the study—on the outcome, specifically whether syncope was cardiologically caused or not.

The predictive significance of sex, age, height, weight, and BMI on whether syncope was cardio-

Table 8. *The impact of the presence of cardiological and non-cardiological diseases on cardiologically caused syncope*

		B	S.E.	Wald	df	p	Exp(B)
Step 1(a)	**Cardiac diseases**	0.517	0.520	0.988	1	0.320	1.676
	Non-Cardiac diseases	-0.764	0.422	3.275	1	0.070	0.466
	Constant	0.689	0.337	4.173	1	0.041	1.993

Table 9. *The Impact of the age of first syncope, number of syncopes, palpitations, chest pain, other prodromal symptoms, and the situation in which it occurred on cardiologically caused syncope*

	B	S.E.	Wald	df	p	Exp(B)
Age of the first syncope	-0.080	0.070	1.330	1	0.249	0.923
Number of syncopes	0.858	0.500	2.950	1	0.086	2.359
Palpitations before syncope	1.285	1.288	0.996	1	0.318	3.615
Pain chest before syncope	-1.699	1.193	2.029	1	0.154	0.183
Prodromal symptoms before syncope	-0.883	0.701	1.586	1	0.208	0.414
Situation in which syncope occurred	-0.059	0.057	1.046	1	0.306	0.943
Constant	1.954	1.242	2.473	1	0.116	7.055

Table 10. *The impact of systolic blood pressure, diastolic blood pressure, and heart rate on cardiologically caused syncope*

	B	S.E.	Wald	df	Sig.	Exp(B)
Systolic BP	-0.007	0.020	0.126	1	0.722	0.993
Diastolic BP	-0.032	0.027	1.430	1	0.232	0.968
Heart rate	-0.017	0.012	1.828	1	0.176	0.984
Constant	4.938	2.279	4.693	1	0.030	139.502

logically caused (yes/no) was examined using binary logistic regression. None of these predictors showed a significant impact, with $p > 0.05$ (Table 6).

The predictive significance of a positive family history of consciousness disorders, heart diseases, and sudden death on whether syncope was cardiologically caused (yes/no) was examined using binary logistic regression. None of these predictors showed a significant impact, with $p > 0.05$ (Table 7).

The predictive significance of the presence of cardiological and non-cardiological diseases on whether syncope is cardiologically caused (yes/no) was examined using binary logistic regression. None of these predictors showed a significant impact, with $p > 0.05$ (Table 8).

The predictive significance of the age of first syncope, number of syncopes, palpitations, chest pain, other prodromal symptoms, and the situation in which it occurred on whether syncope is cardiologically caused (yes/no) was examined using binary logistic regression. Ninety patients had palpitations before syncope, and eighty-seven patients had prodromal symptoms. None of the listed predictors had a significant impact, with $p > 0.05$ (Table 9).

The most prevalent cardiological condition was cardiac arrhythmia, observed in 11 children, accounting for 11.0% of the total number of children in the sample and 50.0% of those with any cardiological disease. The other causes were stenocardia in 2 patients and congenital heart diseases (after surgical corrections) in 5 patients.

Regarding echocardiography, 3 patients had trivial tricuspid regurgitation, 2 had trivial mitral regurgitation, and 12 patients had mild regurgitation of the pulmonary valve, which is considered a physiological finding. Additionally, 1 patient had a surgically corrected atrioventricularseptal defect (AVSD), 1 had a tumorous mass in the right ventricle, 1 had mitral valve prolapse, and 1 had an atrial septal defect (ASD).

The predictive significance of systolic blood pressure, diastolic blood pressure, and heart rate on whether syncope is cardiologically caused was examined using binary logistic regression. None of the listed predictors had a significant impact, with $p > 0.05$ (Table 10).

The predictive significance of the tilt table test and Holter ECG on whether syncope is cardiologically caused (yes/no) was examined using binary logistic regression. Neither of these predictors showed a significant impact, with $p > 0.05$ (Table 11).

Table 11. *The impact of Tilt table test and holter ECG on cardiologically caused syncope*

	B	S.E.	Wald	df	Sig.	Exp(B)
Tilt table test	0.108	0.238	0.204	1	0.651	1.114
Holter ECG	18.291	10524.227	0.000	1	0.999	990.929
Constant	0.232	0.247	0.879	1	0.348	1.261

Table 12. *The predictive significance of EEG findings on cardiologically caused syncope*

	B	S.E.	p	Exp(B)	95,0% C.I.forEXP(B)	
					Lower	Upper
EEG	1.096	0.520	0.035	2.991	1.079	8.297
Constant	0.108	0.233	0.642	1.114		

Table 13. *The relationship between EEG and cardiologically caused syncope*

			EEG		Total
			Normal	Borderline	
Cardiac syncope	**no**	Number	35	6	41
		%	85.4	14.6	100,0
	yes	Number	39	20	59
		%	66.1	33.9	100.0
Total		Number	74	26	100
		%	74.0	26.0	100.0

The significance of EEG findings (normal or borderline) on whether syncope is cardiologically caused (yes/no) was examined using binary logistic regression. EEG was found to be a significant predictor with $p = 0.035$ and $EXP(B) = 2.99$ (Table 12).

For each borderline EEG reading, the odds of syncope being cardiologically caused are approximately three times higher in our sample. In the broader population, this odds ratio ranges from 1 to 8 times.

A "borderline EEG" in children refers to an electroencephalogram (EEG) result that shows subtle, non-specific, or mildly abnormal findings.

The chi-square test of independence showed an association between EEG and syncope with $X^2 = 4.66$, $p = 0.031$. Among the 41 subjects who did not have cardiologically caused syncope, 35 (85%) had a negative EEG and 6 (14.6%) had a positive EEG. Among the 59 subjects with cardiologically caused syncope, 20 (33.9%) had a borderline EEG and 39 (66.1%) had a negative EEG.

DISCUSSION

Syncope is a common issue in pediatric populations and has been extensively studied over the years. According to data from several studies (10), approximately 20% of boys and 40% of girls experience syncope episodes by the age of 18. Our study supports these findings, with 71.0% of syncope patients being female and 29.0% male, showing a higher prevalence of syncope in females.

Two other studies (11, 12) also report a similar gender distribution in syncope prevalence. Another study (13) indicates a higher incidence of syncope among female adolescents compared to males (31.72% vs. 26.25%, $P < 0.05$). Our results align with these, with 71.0% of patients being female. The peak incidence at age 15 further corroborates previous research done by other authors (14).

Despite exploring potential predictors such as gender, age, weight, height, and BMI, our logistic regression analysis found no statistically significant impact on syncope of cardiac origin. However, BMI has been noted in other studies (15) as a potential risk factor for vasovagal syncope. Family history of cardiac diseases did not predict syncope in our study, contrasting with earlier research (16) which reported a positive correlation in 17% of cases. Most participants in our study did not have cardiac conditions (78.0%), with arrhythmias being the most common among those who did.

Our findings differ from previous studies (17, 18), which reported lower rates of cardiac involvement in pediatric syncope. Prodromal symptoms such as dizziness were common but were not predictive of cardiac syncope in our sample. Cardiac auscultation was mostly normal (71.0%), with innocent systolic murmurs widely observed. Blood pressure readings were normal in most cases (73.0% systolic, 64.0% diastolic), consistent with the known association of low blood pressure with vasovagal syncope. A large portion of our participants (78.0%) had no cardiac abnormali-

ties, with arrhythmias being the most frequent finding among those with cardiac conditions.

However, our results diverge from those of Nandini et al, who observed a higher rate of structural heart diseases as the main cause of syncope in children while arrhythmias were less prevalent. This discrepancy might be due to differences in study populations or diagnostic criteria (19).

Arrhythmias in children are increasingly recognized and differ significantly from those in adults (20, 21). In our study, ECG and echocardiogram abnormalities were found in 49.0% and 19.0% of cases, respectively, with sinus arrhythmia and incomplete right bundle branch block common on ECG (both normal pediatric findings), and mild pulmonary valve regurgitation, a normal echocardiographic finding. Tilt table testing and Holter ECG did not predict cardiac syncope in our sample, consistent with the low percentage of abnormal Holter results (4.0%).

Interestingly, EEG showed predictive value for cardiac-related syncope in our study, contrasting with its limited diagnostic value reported elsewhere. EEG findings consistent with cerebral hypoperfusion were seen across syncope types. Despite this, the diagnostic significance of EEG varies among syncope etiologies.

The peak incidence observed at age 15 in our study further aligns with findings by Park et al. which report syncope as being more frequent in adolescents, particularly between 12 and 16 years of age (22).

Among the 100 children studied, 78.0% had no cardiovascular disease, while 22.0% did. Arrhythmias were the most common heart disease in our cohort. A particularly noteworthy finding in our study is the significant predictive value of EEG in diagnosing cardiac syncope, a topic not widely explored previously. While EEG generally has limited value in diagnosing syncope without neurological symptoms, our data suggest that borderline EEG results are strongly associated with cardiac-origin syncope. This contrasts with Dantas et al. (2012), who found EEG useful mainly when neurological involvement was evident. In our population, the odds of cardiac syncope were approximately three times higher in children with borderline EEG findings (23).

CONCLUSION

The study identified syncope episodes peaking at age 15 among girls, with arrhythmia as the predominant cardiac condition. Despite non-cardiac ailments such as headaches being prevalent, most participants had normal cardiovascular parameters,including blood pressure and heart rate. The findings underscore the importance of considering both cardiac and non-cardiac factors in diagnosing and managing syncope in adolescents. Additionally, EEG emerged as a valuable predictor for identifying syncope related to cardiac disorders in children and adolescents aged 6 to 18. These insights highlight the complex nature of syncope etiology and the need for comprehensive diagnostic approaches in clinical practice.

This may reflect the developing brain's greater susceptibility to cerebral hypoperfusion, which can produce transient EEG changes such as generalized slowing or borderline abnormalities. Such findings highlight the importance of considering age-specific physiological responses when interpreting EEG in the context of syncope.

Abbreviations

BMI – Body Mass Index
ECG – Electrocardiogram
ECHO – Echocardiogram
EEG – Electroencephalogram
SD – Standard Deviation
SE – Standard Error
BP – Blood Pressure
AVSD – Atrioventricular septal defect
ASD – Atrial septal defect

Authors' contributions: MV – Supervision and conceptualization of study; **VS**- Writing - review and editing, data collection; Writing - original draft; **BN**-Writing - review and editing; **MA**- Writing - review and editing; **DS**–Conceptualisation; **AD**–Conceptualisation; **TS**–Conceptualisation; **KPJ**–Conceptualisation; **ČA**–Conceptualisation; **BZ** – Involvement in cardiological part of research; **US** – Involvement in neuropediatric pat of research; **ME**– Supervision; **SA** – Supervision.

The author takes full responsibility for the accuracy and integrity of the content, as well as the validity of institutional affiliations. The publisher remains neutral regarding jurisdictional claims in institutional affiliations.

Authorship Statement

The authors have made substantial contributions to all phases of the study and are able to take public responsibility for the content and results presented in the manuscript.

Conflict of Interests: The authors declare no conflicts of interest related to this article.

Funding: This research received no external funding.

Note: Artificial intelligence was not utilized as a tool in this study.

Sažetak

KARDIOLOŠKI DIJAGNOSTIĆKI PRISTUP DECI S POREMEĆAJIMA SVESTI

Mišanović Verica,[1] Viteškić Selma,[2] Begić Nedim,[1] Mulać Ahmed,[1] Dizdar Selma,[1] Anić Duško,[1] Terzić Sabina,[1] Kovačević Prstojević Jelena,[3] Čengić Adisa,[1] Begić Zijo,[1] Užičanin Sajra,[1] Međedović Edin,[4] Selimović Amina[1]

[1] Univerzitetski klinički centar Sarajevo, Pedijatrijska klinika, Sarajevo, Bosna i Hercegovina
[2] Dom zdravlja Kantona Sarajevo, Sarajevo, Bosna i Hercegovina
[3] Univerzitetski klinički centar Sarajevo, Klinika za urologiju, Sarajevo, Bosna i Hercegovina
[4] Univerzitetski klinički centar Sarajevo, Klinika za ginekologiju i akušerstvo, Sarajevo, Bosna i Hercegovina

Uvod: Sinkopa je jedan od najčešćih razloga traženja medicinske pomoći u pedijatrijskoj populaciji. Etiologija može varirati od benigne do potencijalno opasne po život.

Cilj: Cilj ove retrospektivne studije preseka bio je kardiološka evaluacija dece sa sinkopom i otkrivanje potencijalnih prediktornih parametara za kardiološki uzrokovanu sinkopu, koja je najopasniji oblik sinkope.

Učesnici i metode: Podaci o 100 dece uzrasta od 6 do 18 godina koji su se javili sa sinkopom prikupljeni su iz medicinske dokumentacije Pedijatrijske klinike Kliničkog centra Univerziteta u Sarajevu u periodu od 01. 01. 2021. do 31. 12. 2022. Binarnom logističkom regresijom ispitana je prediktivna značajnost parametara koje je ovo istraživanje obuhvatilo.

Rezultati: Od 100 dece sa zabeleženom sinkopom, 71,0% su bile devojčice, a najveći broj epizoda sinkope zabeležen je u dobi od 15 godina. Nije bilo statistički značajne razlike u visini, težini i BMI vrednostima između dečaka i devojčica. Najčešće kardiološko oboljenje bila je sinusna aritmija, dok je najčešći nekardiološki simptom bila glavobolja.Više od jedne epizode sinkope imalo je 73,0% dece, a najveći broj epizoda dogodio se u školi. Prodromalni simptomi su bili prisutni kod 87,0% ispitanika, dok su palpitacije i bol u prsima pre sinkope zabeležene kod 10,0% i 12,0% dece. Od 49 ispitanika koji nisu imali uredan EKG nalaz, 29,0% imalo je sinusnu aritmiju, a 25,0% nepotpuni blok desne grane. Najčešći EHO nalaz kod pacijenata bila je blaga regurgitacija pulmonalne valvule, što se smatra fiziološkim nalazom.Nijedan od ispitivanih parametara nije imao prediktivni značaj za kardiološki uzrokovanu sinkopu osim EEG-a, koji se pokazao kao značajan prediktor ($p = 0,035$, EXP(B) = 2,99).

Zaključak: EEG ima prediktivni značaj za kardiološki uzrokovanu sinkopu, pri čemu je verovatnoća da je sinkopa kardiološkog porekla otprilike tri puta veća kod deteta sa graničnim EEG nalazom.

Ključne reči: Sinkopa, pedijatrijska populacija, EEG, ehokardiografija, prediktivni parametri, kliničke karakteristike, elektrokardiografija.

REFERENCES

1. Zeman A. What in the world is consciousness? Prog Brain Res. 2005; 150: 1-10. doi: 10.1016/S0079-6123(05)50001-3.

2. Grossman SA, Badireddy M. Syncope. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK442006/.

3. Singhi P, Saini AG. Syncope in pediatric practice. Indian J Pediatr. 2018; 85(8): 636-40. doi:10.1007/s12098-017-2488-9.

4. McLeod KA. Syncope in childhood. Arch Dis Child. 2003; 88(4): 350-3. doi: 10.1136/adc.88.4.350.

5. Lee M, Sanz LRD, Barra A, Wolff A, Nieminen JO, Boly M, et al. Quantifying arousal and awareness in altered states of consciousness using interpretable deep learning. Nat Commun. 2022; 13(1): 1064. doi: 10.1038/s41467-022-28451-0.

6. Ungar A, Sgobino P, Russo V, Vitale E, Sutton R, Melissano D, et al. Diagnosis of neurally mediated syncope at initial evaluation and with tilt table testing compared with that revealed by prolonged ECG monitoring. An analysis from the Third International Study on Syncope of Uncertain Etiology (ISSUE-3). Heart. 2013; 99(24): 1825-31. doi: 10.1136/heartjnl-2013-304399.

7. Jones PK, Shaw BH, Raj SR. Orthostatic hypotension: managing a difficult problem. Expert Rev Cardiovasc Ther. 2015; 13(11): 1263-76. doi: 10.1586/14779072.2015.1095090.

8. Smith PE. If it‘s not epilepsy.. J Neurol Neurosurg Psychiatry. 2001; 70(Suppl 2): II9-14. doi: 10.1136/jnnp.70.suppl_2.ii9.

9. Brignole M, Moya A, de Lange FJ, Deharo JC, Elliott PM, Fanciulli A, et al 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J. 2018; 39(21): 1883-948. doi: 10.1093/eurheartj/ehy037.

10. DiMario FJ Jr, Wheeler Castillo CS. Clinical categorization of childhood syncope. J Child Neurol. 2011; 26(5): 548-51. doi: 10.1177/0883073810384864.

11. Deveau AP, Sheldon R, Maxey C, Ritchie D, Doucette S, Parkash R. Sex differences in vasovagal syncope: a post hoc analysis of the prevention of syncope trials (POST) I and II. Can J Cardiol. 2020; 36(1): 79-83. doi: 10.1016/j.cjca.2019.10.008.

12. Friedman KG, Alexander ME. Chest pain and syncope in children: a practical approach to the diagnosis of cardiac

disease. J Pediatr. 2013; 163(3): 896-901.e1-3. doi: 10.1016/j.jpeds.2013.05.001.

13. Hu E, Liu X, Chen Q, Wang C. Investigation on the incidence of syncope in children and adolescents aged 2-18 years in Changsha. Front Pediatr. 2021; 9: 638394. doi: 10.3389/fped.2021.638394.

14. Ganzeboom KS, Mairuhu G, Reitsma JB, Linzer M, Wieling W, van Dijk N. Lifetime cumulative incidence of syncope in the general population: a study of 549 Dutch subjects aged 35-60 years. J Cardiovasc Electrophysiol. 2006; 17(11): 1172-6. doi: 10.1111/j.1540-8167.2006.00595.x.

15. Salari N, Karimi Z, Hemmati M, Mohammadi A, Shohaimi S, Mohammadi M. Global prevalence of vasovagal syncope: a systematic review and meta-analysis. Glob Epidemiol. 2024; 7: 100136. doi: 10.1016/j.gloepi.2024.100136.

16. Imes CC, Lewis FM. Family history of cardiovascular disease, perceived cardiovascular disease risk, and health-related behavior: a review of the literature. J Cardiovasc Nurs. 2014; 29(2): 108-29. doi: 10.1097/JCN.0b013e31827db5eb.

17. Yeom JS, Woo HO. Pediatric syncope: pearls and pitfalls in history taking. Clin Exp Pediatr. 2023; 66(3): 88-97. doi: 10.3345/cep.2022.00451.

18. Castro W, Skarin R, Roscelli JD. Orthostatic heart rate and arterial blood pressure changes in normovolemic children. Pediatr Emerg Care. 1985; 1(3): 123-7. doi: 10.1097/00006565-198509000-00004.

19. Madan N, Carvalho KS. Neurological complications of cardiac disease. Semin Pediatr Neurol. 2017; 24(1): 3-13. doi: 10.1016/j.spen.2017.01.001.

20. Sheldon RS, Grubb BP 2nd, Olshansky B, Shen WK, Calkins H, Brignole M, et al. 2015 heart rhythm society expert consensus statement on the diagnosis and treatment of postural tachycardia syndrome, inappropriate sinus tachycardia, and vasovagal syncope. Heart Rhythm. 2015; 12(6): e41-63. doi: 10.1016/j.hrthm.2015.03.029.

21. Begic Z, Begic N, Granov N, Vila H, Berberovic-Hukeljic B, Begic E, et al. Pacemaker implantation in the pediatric population: Bosnian and Herzegovinian experience. Heart and Mind. 2025; 7(2): 111-4. doi: 10.4103/hm.hm_12_22.

22. Park J, Jang SY, Yim HR, On YK, Huh J, Shin DH, et al. Gender difference in patients with recurrent neurally mediated syncope. Yonsei Med J. 2010; 51(4): 499-503. doi: 10.3349/ymj.2010.51.4.499.

23. Dantas FG, Cavalcanti AP, Rodrigues Maciel BD, Ribeiro CD, NapyCharara GC, Lopes JM, et al. The role of EEG in patients with syncope. J Clin Neurophysiol. 2012; 29(1): 55-7. doi: 10.1097/WNP.0b013e318246b589.

Correspondence to/Autor za korespondenciju
Nedim Begić, MD, MA
Clinical Center University of Sarajevo, Pediatric Clinic, Department of Pediatric Gastroenterohepatology and Nutrition
Orcid ID: 0000-0001-5964-2305
e-mail: nedim_begic91@hotmail.com

ORCID ID:
Verica Mišanović – 0000-0002-8800-5003
Nedim Begić – 0000-0001-5964-2305
Ahmed Mulać – 0009-0002-7897-3999
DuškoAnić – 0009-0004-3803-6479
Sabina Terzić – 0000-0003-4917-0142
Zijo Begić – 0000-0002-1863-5755
Edin Međedović – 0000-0003-2357-9580
Amina Selimović – 0000-0002-4914-5468

***How to cite this article**:* Mišanović V, Viteškić S, Begić N, Mulać A, Dizdar S, Anić D,Terzić S, Kovačević Prstojević J, Čengić A, Begić Z, Užičanin S, Međedović E, Selimović A. Cardiological diagnostic approach to children with consciousness disorders. Sanamed. 2025; 20(2): 157-167. doi: 10.5937/sanamed0-57055.