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Syncope in Childhood

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Abstract. The records of 108 children, ages 2 to 19 years (mean age 11.3 years), who were referred to the pediatric neurology and pediatric cardiology clinics for syncope, were reviewed. Sixty-six cases were identified retrospectively, and 42 prospectively. Syncope was defined as transient and complete loss of consciousness with no etiology determined at the time of presentation. The mean follow-up was 2.0 years. In 27 cases (25%), an etiology for syncope was found, including migraines in 12 cases (11%), seizures in 9 cases (8%), and cardiac arrhythmias in 6 cases (6%). All other cases were classified as vasovagal (neurocardiogenic). The past medical history, family history, clinical features of each syncopal episode, and diagnostic tests of each subject were correlated to final diagnosis. No clinical or historical features reliably distinguished children with vasovagal syncope from those with other etiologies. Children referred for the evaluation of syncope have a significant incidence of serious but treatable disorders, which should be actively sought.

Key words: Syncope — Arrhythmia — Seizure — Migraine

Syncope is a common event in childhood. Retrospective surveys of college-age students have shown that 47% had fainted in the past [11]. At least 15% of children experience syncope before the end of adolescence [13]. However, relatively few children are brought to medical attention for syncope. Syncope is responsible for around 1 in 2000 visits to a pediatric emergency room [12]. Most episodes of syncope are due to neurally mediated hypotension or bradycardia [14]. The terms *vasovagal syncope*, *vasodepressor syncope*, and more recently *neurocardiogenic syncope* have been applied to these events [18]. However, many studies have reported potentially dangerous causes of syncope in children [3, 20]. Even vasovagal syncope has been reported to require intervention on occasion [15]. The diagnosis and treatment of syncope in adults has been extensively investigated [7]. However, when a child is brought to medical attention with a history of syncope, the appropriate diagnostic evaluation is less clear.

Methods

The present study reviews the etiologies of syncope in children who presented to our hospital over a 7-year period. Eligible subjects for this study were children who presented with the diagnosis of syncope. For the retrospective arm, the patients were identified using the databases of the pediatric neurology and pediatric cardiology divisions. These databases contain the referring diagnosis of all patients referred to these divisions. The records of all patients referred by the pediatrician or emergency department physician to these divisions for syncope between July 1983 and July 1988 were then reviewed. The prospective study, in which referrals by pediatricians or emergency room physicians to pediatric neurology or pediatric cardiology for syncope were included as the cases occurred, began in July 1988 and ended in September 1990. It was the practice of our emergency department during that time period routinely to refer children who presented with an episode of loss of consciousness for further evaluation to either the pediatric neurology or the pediatric cardiology clinic.

Syncope was defined as any episode in which there was complete and transient loss of consciousness. For both the retrospective and prospective investigations, the criteria for admission to the study were: (1) the patient presented with syncope, (2) no definite etiology was known at the time of the referral, (3) the patient was younger than 19 years of age. Patients with a past history of seizures or arrhythmias were excluded as were those who presented with clear-cut seizures or arrhythmias. In addition, the patient was excluded if the record did not document a complete loss of consciousness.

A standardized data summary was used for each patient. All charts were reviewed and all summaries were coded by a single author (M.L.M.). Abnormal laboratory results were reviewed by all authors.

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Although authors M.L.M., C.A.W., and S.S. were involved in the care of some of the patients, all decisions regarding diagnosis and treatment were made prior to the collection of data.

The etiology was considered a seizure either when an electroencephalogram revealed clear epileptiform discharges or the child subsequently had unequivocal clinical seizures that had an onset similar to the syncopal episode. Nonepileptiform abnormalities on the electroencephalogram were not considered sufficient grounds to classify the syncopal episode as being seizure-related.

An arrhythmia was diagnosed by Holter monitor, stress test, transtelephonic monitor, or invasive electrophysiologic study. Electrocardiograms were performed, but an electrocardiogram demonstrating an arrhythmia in the emergency room would have excluded the patient by virtue of a diagnosis being made prior to referral. An echocardiogram, cardiac catheterization, cardiac magnetic resonance imaging, and/or cardiac biopsy were performed as indicated.

Children with a throbbing, vascular headache immediately prior to the syncopal episode, who also had a history of vascular headaches, were considered to have migraine-related syncope. A vascular headache following a syncopal episode was not sufficient grounds to make this diagnosis, as this is a nonspecific finding, unless other criteria are met. Most of these children did not meet the criteria for basilar artery migraine, which would include, in addition to the syncope, some brain stem findings such as tinnitus, diplopia, visual loss, and true vertigo [9, 16].

The diagnosis of vasovagal syncope was made as a diagnosis of exclusion. All patients who did not have another diagnosis were categorized as having vasovagal syncope.

Data Analysis

The standard data summary included details of the syncopal episode, including trigger factors, duration and associated symptomatology, a history of any prior events, a history of any preexisting cardiac or neurological abnormalities, and a family history of syncope, arrhythmias, migraines, or seizures. A summary of the laboratory tests and results was also recorded on the data sheets. Data were analyzed using standard statistical methods [21]. The descriptive statistics included the relative incidence of different etiologies, associated symptoms, and age distribution. Chi-square statistics and *t*-tests were used. In comparing crude proportions and frequencies in different subgroups, the significances of the resulting 2 × 2 tables were calculated using Fischer's exact test. All *p*-values were computed using 2-tailed distributions using a 0.05 level for significance.

Results

Population Characteristics

The study population consisted of 108 children, 66 evaluated retrospectively, and 42 prospectively. There were 59 girls and 49 boys. The mean age at the time of the first episode was 11.5 years. Children with seizures and arrhythmias were slightly younger (9.9 years and 9.7 years, respectively), and children with migraines were slightly older (13.0 years). All groups except the arrhythmia group had a slight but statistically insignificant female preponderance.

Table 1. Distribution of patients by etiology

	Number	Female	Mean age (years)
Vasovagal	81 (75%)	55%	11.0
Migraine	12 (11%)	58%	13.0
Arrhythmia	6 (6%)	33%	9.7
Seizure	9 (8%)	60%	9.9
Total	108 (100%)	55%	11.5

Etiology of Syncope

The causes of syncope are summarized in Table 1. No nonarrhythmic cardiac, metabolic, pharmacologic, psychogenic, orthostatic, or situational (e.g., micturition, defecation, tussive, deglutition) etiology was found. The majority of patients (75%) were diagnosed as having vasovagal syncope. Twelve children (11%) were classified as having migraine-associated syncope. One patient fainted with his first vascular headache. Atypical seizures and arrhythmias were surprisingly common, representing 8% and 6% of the study population, respectively. The arrhythmias included atrial flutter in a 2-year-old girl with a normal heart; presumed *torsades de pointes* in a 16-year-old deaf boy and a 12-year-old girl, both of whom had long QT syndrome; catecholamine-induced ventricular tachycardia in a 12-year-old boy with a strong family history of sudden death, normal QTc interval, and conduction system disease on autopsy; ventricular tachycardia in a 6-year-old boy with endocardial fibroelastosis on cardiac biopsy; and supraventricular tachycardia in a 13-year-old girl with Wolff–Parkinson–White syndrome (Table 2).

Symptomatology and History

All children in the study had at least one episode of complete loss of consciousness, for this was a selection criterion. Nearly half of all episodes were reported as lasting less than 1 min and 83% of all episodes less than 5 min. Vasovagal syncope more often lasted less than 1 min (49%) vs all others (25%), ($p < 0.05$), but brief and prolonged loss of consciousness was found in all groups (Table 3).

Syncopal episodes were preceded by a prodrome in 73 (68%) cases including lightheadedness in 61%, dizziness in 23%, and vertigo in 12%. There were no clear differences in the proportion of children with these symptoms in each category.

During the syncopal episodes, eye rolling occurred in 14% and movements such as twitching and jerking in 20%. Eye rolling occurred in a third and associated movements in 44% of patients whose syncope was caused by a seizure. However, eye rolling occurred in

Table 2. Arrhythmia-induced syncope

Patient	Age (years)	Sex	Arrhythmia	Diagnosed by	Cardiac anomaly	Clinical status
1	2	F	Atrial flutter	EPS	Normal	Controlled with digoxin
2	16	M	Torsades de pointes (?)	Holter[a]	LQTS (Nl ECG)	Controlled with atenolol
3	12	F	Torsades de pointes (?)	FH of LQTS	LQTS (Nl ECG)	Controlled with propranolol
4	12	M	Ventricular tachycardia	Stress test[b]	Conduction system disease (autopsy)	Died suddenly
5	6	M	Ventricular tachycardia	Stress test	EFE (biopsy)	Controlled with atenolol
6	13	F	Supraventricular tachycardia	TTM	WPW (N1 ECG)	Ablation, asymptomatic

ECG, electrocardiogram; EFE, endocardial fibroelastosis; EPS, electrophysiology study; FH, family history; LQTS, long QT syndrome; Nl, normal; TTM, transtelephonic monitoring; WPW, Wolff–Parkinson–White syndrome.
[a] Deafness in patient and sister.
[b] FH of sudden death, seizures.

Table 3. Duration of syncopal episode

	Length (min)			
	<1	1–5	5–20	Unknown
Vasovagal	37	24	14	6
Migraine	3	5	2	2
Arrhythmia	0	5	1	0
Seizure	3	5	0	1
Total	43	39	17	9

15% and associated movements in 15% of other syncopal episodes and were thus not reliable distinguishing factors.

Of note is that a history of exertion preceding the syncopal episode was obtained in three of the six children in the cardiac arrhythmia group (Table 2, patients 4, 5, 6), but in only one of the 102 other children ($p < 0.001$). Three of the children with an arrhythmia had significant family histories (Table 2, patients 2, 3, 4). A family history of seizures was present in 13 cases, including 10 with vasovagal syncope, one with cardiac arrythmia (Table 2, patient 4), one with migraine, and only one with seizures.

A vascular headache preceding the syncopal episode occurred in the 12 children with migraine-related syncope. Migraine headaches were also a common previous complaint in patients with vasovagal syncope (41%) even though none of these patients had syncope preceded by a vascular headache.

Laboratory Studies

Seventy-six children had electroencephalograms performed. Nonspecific abnormalities were found in one child in both the vasovagal and arrhythmia (Table 2, patient 3) groups. Six of the nine children with seizures had epileptiform EEG patterns. The remainder of this group had subsequent unequivocal seizures that were similar in onset to their syncopal episode and a diagnosis of seizures was made without a specific electroencephalographic abnormality.

Electrocardiograms were performed on 98 children, but since an arrhythmia present on an initial electrocardiogram would have excluded the child from the study, no analysis can be made.

Twenty-nine Holter monitor studies and five stress tests were performed. Holter monitoring was diagnostic in one case, stress testing in two, transtelephonic monitoring in one, and electrophysiologic testing in one.

Prognosis

Recurrent syncope occurred in 53 of 79 (67%) children with vasovagal syncope and 25 (96%) of those with syncope due to other causes ($p < 0.005$; Table 4). Forty-four (42%) children experienced three or more episodes of syncope including 38% of those with vasovagal syncope and 54% of those with syncope due to other causes ($p = 0.46$).

The patient with catecholamine-induced ventricular tachycardia discontinued his medication and died suddenly while running. The patient with Wolff–Parkinson–White syndrome is asymptomatic after ablation of the Kent bundle. The other four patients in the group with arrhythmias are asymptomatic with medication (Table 3). The remainder of the patients have had no significant morbidity from their illness, although about one-third continue to faint.

Table 4. Recurrent syncope

	Number of episodes in a patient			
	1 (Number of patients)	2 (Number of patients)	>2 (Number of patients)	Unknown (Number of patients)
Vasovagal	26	23	30	2
Migraine	1	6	5	0
Arrhythmia	2	1	3	0
Seizure	0	4	4	1
Total	29	34	42	3

Discussion

The majority of children who present with syncope to a pediatric emergency room will have a benign etiology. However, a significant minority will have a more serious and potentially treatable cause of syncope, such as a seizure or cardiac arrhythmia. The population in this study was screened by pediatricians and emergency department physicians, which may have caused the percentage of arrhythmias and seizures to be artificially high. Nevertheless, the findings are of concern. Our results are similar, with regard to seizures and migraine syncope, to those results of a study of children presenting to the emergency department with syncope [12], but the results differ with regard to the incidence of arrhythmias.

In our experience, the diagnosis of an arrhythmia often required extensive evaluation with repeated testing over a long period of time. Arrhythmia-induced syncope was diagnosed if an electrocardiogram, Holter monitor (Table 2, patient 2), stress test (Table 2, patients 4, 5), transtelephonic monitor (Table 2, patient 6), or electrophysiologic study (Table 2, patient 1) demonstrated a significant arrhythmia.

The practice of measuring the QTc interval on an electrocardiogram is particularly important in patients with syncope [4, 5]. However, both patients in this study with long QT syndrome had a normal QTc interval on initial electrocardiogram. The diagnosis made was based on family history and Holter monitoring. In addition, preexcitation was not initially evident on the electrocardiogram of the patient with Wolff–Parkinson–White syndrome. Supraventricular tachycardia was recorded by transtelephonic monitoring in this patient.

Stress testing induced ventricular tachycardia in the patient with endocardial fibroelastosis and in the patient with a family history of sudden death and seizures. The only death in this study occurred in this last patient. (All family members had normal QTc intervals.)

The only patient who underwent an electrophysiology study was a two-year-old girl with a normal electrocardiogram, echocardiogram, and Holter monitoring. She was considered too young for stress testing. During two syncopal events, her mother failed to activate the transtelephonic monitor. Programmed electrical stimulation induced atrial flutter with hypotension. No further episodes occurred after digoxin was started.

Since cardiac arrhythmias may be life-threatening, and are often treatable or even curable today, a thorough search for them is warranted. However, no specific algorhythm for cardiac evaluation can be formulated from this study.

A history of eye rolling and of associated movements was present in 15% of those patients without seizures. A convulsive component to syncopal episodes is not uncommon and by itself cannot be used to make the diagnosis of seizures [2, 6, 19]. The complaint of headache just prior to syncope does not generally occur with etiologies other than migraine [5, 17]. However, headache (including migraine) in general was common in children with vasovagal syncope and seizures.

In this study, vasovagal syncope was made as a diagnosis of exclusion. Since not all children underwent full diagnostic evaluations for seizures or arrhythmias, it is possible that their actual numbers were higher. When this study was done, there was no widely used test for vasovagal syncope. Head-up tilt table testing is now available as a means of provoking episodes of neurocardiogenic (vasovagal) syncope in predisposed persons, thereby enabling the establishment of a diagnosis [1, 8, 14]. Potential problems with head-up tilt-table testing include reproducibility, specificity, and sensitivity, compounded by the absence of a uniform protocol in the literature. Nevertheless, a positive test does indicate the potential vasovagal nature of a syncopal episode.

Conclusions

Syncope in childhood is common and usually has benign causes. In some children, however, it may be the presenting symptom of seizures or cardiac arrhythmias. It appears that in many cases vasovagal syncope cannot be distinguished reliably by history, physical examination, or laboratory tests available in the emergency department, from syncope with more dangerous causes. Therefore, we recommend that children who present to the emergency department with syncope be referred for further work-up, particularly if it is not their first episode.

A child or adolescent with recurrent syncope should be thoroughly evaluated for risk factors for sudden death with a complete history, physical examination and electrocardiogram. Syncope risk factors include cardiac disease, exercise association, history of seizures, and a family history of sudden death, syncope, seizures, arrhythmias, or deafness. A history of exercise-associated syncope could indicate hypertrophic cardiomyopathy, anomalous origin of the left coronary from the right sinus

of Valsalva, aortic stenosis, long QT syndrome, or catecholamine sensitive ventricular tachycardia [10]. Family history might suggest long QT syndrome, hypertrophic cardiomyopathy, or Wolff–Parkinson–White syndrome. Physical examination will uncover many associated structural heart diseases, such as aortic stenosis and cyanotic congenital heart disease. The electrocardiogram is useful to screen for long QT syndrome and Wolff–Parkinson–White syndrome, although it was normal in the patients with these diagnoses in this study. It is also useful in suggesting structural disorders such as hypertrophic cardiomyopathy, which might not be detectable on physical examination if it is nonobstructive.

If any risk factors, especially cardiac, are found, more extensive evaluation should be undertaken, including echocardiography, Holter monitoring, transtelephonic monitoring, stress testing, and even cardiac catheterization and electrophysiology study in selected cases. Since head-up tilt testing was not employed in this study, the data cannot be used to comment on its indications. However, even if the test is positive in a patient with risk factors for sudden death, it is still imperative to rule out cardiac pathology.

Further studies are needed to define the relative proportions of pathologic substrates in less selected populations and to better define the indications for more extensive diagnostic evaluations. Cardiac causes of syncope other than vasovagal, although uncommon, represent potentially fatal but treatable disorders and, therefore, deserve particular attention.

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