Original Article

Value of history taking in children and adolescents with cardiac syncope

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Abstract *Aim:* This study was designed to investigate the value of history taking in identifying children with cardiac syncope, and to improve diagnostic efficiency and accuracy in children with cardiac syncope. *Methods and Results:* We compared the characteristics of a group of children and adolescents with cardiac syncope at the Pediatric Syncope Unit of five hospitals in China with those with typical vasovagal syncope. We included a cohort of 275 patients in Pediatric Syncope Unit. A cardiac cause of syncope was established in 31 patients, autonomic-mediated reflex syncope in 214, non-syncopal attacks in 15, and in the remaining 15 the cause of syncope remained unexplained. Cardiac syncope was triggered by exercise, whereas vasovagal syncope by prolonged standing, warm-crowded place, and fear or pain emotion. Syncopal spells occurred at various positions in cardiac syncope. Children who had prodromal symptoms with cardiac syncope were significantly fewer than those with vasovagal syncope. Most children with cardiac syncope had history of abnormal electrocardiogram findings when compared with children suffering from vasovagal syncope. On multivariable analysis, history of abnormal electrocardiogram findings and exercise-triggered syncope were independent predictors of cardiac syncope. *Conclusion:* Children and adolescents with a history of abnormal electrocardiogram findings and exercise-related syncope spells were at high risk for cardiac syncope.

S YNCOPE IS A COMMON PROBLEM IN CHILDHOOD. About 15% children under the age of 18 years have had at least one syncopal episode.[1–3] Syncope in children and adolescents can be caused by a variety of underlying diseases. According to our study and other research, the most common cause of syncope is vasovagal syncope.[4–6] Other causes include orthostatic hypotension, cardiac and postural orthostatic tachycardia syndrome, and epileptic seizure or psychogenic pseudosyncope. Cardiac syncope represents 2–6% of paediatric cases.[1,6,7] The causes of cardiac syncope are mainly arrhythmia and structural heart diseases. The prognosis of cardiac syncope and vasovagal syncope is different, but the two kinds of syncope in clinical practice are similar. Therefore, it is important to distinguish cardiac syncope and vasovagal syncope. History taking plays a key role in the initial evaluation of syncope.[8] In adult patients, a point score based on clinical history predicting arrhythmias for patients

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with unexplained syncope in the emergency department has developed. For example, Sarasin et al[9] found that abnormal electrocardiogram, a history of congestive heart failure, and age greater than 65 years were predictors of arrhythmic syncope. Rosso et al also developed a diagnostic point score to discriminate cardiac syncope from syncope of other causes. They found that abnormal electrocardiogram and/or heart disease, palpitations before syncope during effort or in supine position, absence of autonomic prodromes, and absence of predisposing and/or precipitating factors were the predictors of cardiac syncope.[10] However, this score is of limited value when evaluated in the children and adolescents with syncope because, as our previous study indicated, the causes of syncope in children and adolescents differed from those of adult patients.[11] The better understanding of clinical characteristics of cardiac syncope in children and adolescents is an important issue to improve the diagnosis of syncope in children. The present study was, therefore, undertaken to identify clinical features of patients with cardiac syncope and explore the useful history findings predictive of the cause of syncope.

Methods

Study subjects

The study was prospective and included consecutive patients less than or equal to 18 years of age with suspected syncope admitted to the Pediatric Syncope Unit of five hospitals in Beijing, Shanghai, Changsha, and Wuhan in China from October, 2006 to June, 2010. This investigation was sponsored by the Pediatric Syncope and Related Disorders Clinical Study Group.[6] All patients were affected by transient loss of consciousness, which, on initial evaluation, was attributed to a syncopal condition or a syncopal condition not to be excluded. Patients with symptoms that were clearly compatible with seizure disorders, vertigo, coma, or shock were not included. All patients underwent a standardised initial evaluation consisting of history taking, physical examination, orthostatic blood pressures, and standard electrocardiogram. According to the initial evaluation, patients were classified into three groups: those with a cause of syncope strongly suspected according to disease-specific diagnostic criteria; those with a specific entity causing syncope suspected by suggestive signs or symptoms that required confirmation by selected diagnostic procedures – such as echocardiography for suggested hypertrophic cardiomyopathy; and those with the cause of syncope undetermined. When the results of the initial evaluation were inconclusive, further clinical and laboratory investigations in patients for

whom a cause of syncope was strongly suspected were guided by a diagnostic hypothesis, which was based on the abnormalities found in the initial clinical assessment.

Diagnostic criteria

Diagnostic criteria of the causes of syncope were developed before recruitment on the basis of the best evidence-based recommendations, and assignment of a cause was based on strict adherence to these criteria. In particular, mechanical cardiac syncope was diagnosed in the presence of outflow obstruction of heart disease, such as hypertrophic cardiomyopathy or idiopathic pulmonary hypertension.[6,8,10] Arrhythmic syncope was diagnosed on the basis of a standard electrocardiogram, electrocardiogram monitoring, 24-hour Holter recordings, or during the electrophysiological study. A diagnosis of arrhythmic syncope was considered with electrocardiogram findings – with 12-lead electrocardiogram and Holter recorder – of sinus pause greater than or equal to 3 seconds, sinus bradycardia less than or equal to 45 beats per minute, Mobits 2 or advanced 2:1 block, complete atrioventricular block, non-sustained ventricular tachycardia greater than 5 beats per minute, congenital long-QT syndrome with prolonged QT interval corrected for heart rate by Bazett's formula greater than 480 milliseconds, and episodic ventricular tachycardia, particularly torsade de pointes. Diagnostic results of electrophysiologic studies included prolonged corrected sinus-node recovery time indicative of sinus-node disease, prolonged H–V interval, and superventricular tachycardia greater than or equal to 180 beats per minute.[6,10,12]

A diagnosis of vasovagal syncope was made[6,12,13] if a trigger – such as prolonged standing, mental stress, and pain – was associated with prodromal signs and symptoms – such as nausea, light-headedness, and diaphoresis called typical vasovagal – or if a less typical history was confirmed by a positive table tilt test and/or information from follow-up, and no other cause of syncope was found after additional testing and follow-up – called tilt-induced syncope.

Owing to the fact that typical vasovagal syncope patients were diagnosed only on the basis of history, we used typical vasovagal syncope patients as control group to find clinical characteristics of cardiac syncope in children and adolescents.

Postural orthostatic tachycardia syndrome is defined as the development of orthostatic intolerance symptoms accompanied by a heart rate increase of at least 30 beats per minute, or a rate that exceeds 120 beats per minute, which occurs within the first 10 minutes of standing or head-up tilt test, in the absence of other chronic debilitating disorders, prolonged bed rest, or medications that impair autonomic reflexes.[6]

Orthostatic hypotension was defined as a persistent decrease of more than 20 millimetres of mercury of systolic blood pressure or decrease of more than 10 millimetres of mercury of diastolic blood pressure within 3 minutes after the start of head-up tilt test or standing.[6]

Situational syncope was diagnosed if syncope occurred during or immediately after micturition, defecation, coughing, or swallowing.[6]

Syncope questionnaire

All patients completed a 118-item questionnaire[12–14] developed with formative input from Calkins et al.[13] It included age, gender, duration of disease, attack frequency, predisposing factors, prodromal symptoms of syncope, syncope duration, body position at the onset of syncope, any associated symptoms, the duration of the loss of consciousness, a previous history of syncope related to heart disease, and family history of sudden death or syncope. A standard 12-lead electrocardiogram was obtained on all patients. The variables investigated are reported in Table 2.

This study was approved by the ethics committee of all participating hospitals, and for each child informed parental consent and the child's agreement were obtained.

Statistical analysis

Continuous variables were summarised as mean scores and standard deviations from the mean. For the purpose of comparing the distribution of continuous variables, a t-test or a non-parametric alternative, Mann–Whitney, was used where appropriate. The χ^2 method or Fisher test was used to compare categorical variables, as appropriate. Logistic regression analysis was used to find out the independent predictors for cardiac syncope. SPSS software (version 13.0) was used to analyse the data (p less than 0.05 was defined as the level of significance).

Results

The diagnosis of cardiac syncope in children

During the recruitment period, data from 275 patients were collected. At the end of the diagnostic evaluation, vasovagal syncope was established in 132 (48.0%), including 55 cases with typical vasovagal syncope and 77 with tilt-induced vasovagal syncope. Postural orthostatic tachycardia syndrome was diagnosed in 67 (24.4%), situational syncope in 8 (2.9%), orthostatic hypotension in 7 (2.5%), cardiac syncope in 31 (11.2%) including 22 patients caused by arrhythmias (8.0%) and 9 caused by mechanical cardiac syncope (3.2%), and the cause of syncope remained unexplained in 15 (Table 1). In 15 (5.5%)

Table 1. Causes of syncope (n = 275).

Causes of syncope	Number (%)
Cardiac	31 (11.2)
Mechanical	9 (3.2)
Bradyarrhythmic	10 (3.6)
Tachyarrhythmic	12 (4.4)
Autonomic-mediated	214 (77.8)
Typical vasovagal	55 (20.0)
Tilt-induced	77 (28.0)
Postural orthostatic tachycardia syndrome	67 (24.4)
Orthostatic hypotension	7 (2.5)
Situational	8 (2.9)
Non-syncopal attacks	15 (5.5)
Unexplained	15 (5.5)

patients, non-syncopal cause of loss of consciousness was confirmed by investigations. Electroencephalography, brain computed tomographic scan, and magnetic resonance imaging were applied in the case of suspected epilepsy or migraine; laboratory blood tests were performed in the case of suspected hypoglycaemia and severe anaemia, and psychiatric consultation in the case of psychogenic pseudosyncope.

Table 2 shows the diagnosis, history of electrocardiogram performance, and diagnostic tests needed to confirm the diagnosis of 31 cases of cardiac syncope. Of the 31 cases of cardiac syncope, case 1 and case 24 – congenital long QT syndrome – had a family history of sudden death; 5 patients had a previous history of heart disease, of which case 8 had a history of myocarditis, case 9 had a history of surgery of total anomalous pulmonary venous return, cases 11 and 28 had a history of ventricular septal defect repair, and case 22 had a history of surgical correction for transposition of the great arteries.

Univariate analysis of clinical features of cardiac syncope compared with typical vasovagal syncope in children and adolescents (Table 3)

Using univariate analysis, children with cardiac syncope were younger than those with vasovagal syncope in terms of age at the first syncope. The mean age of children with cardiac syncope was 8.5 years old, whereas it was 11.6 years old in children with vasovagal syncope.

Most cardiac syncope was triggered by exercise, whereas most vasovagal syncope was triggered by prolonged standing, fear or pain emotion, or warm-crowded place. Syncopal spells in supine position were common in cardiac syncope, but the spells of the majority of children with vasovagal syncope occurred while standing (96.4%). Only 2 out of 55 children with vasovagal syncope had syncopal episodes while in the supine position. The number of children with cardiac syncope who had prodromal

Table 2. The diagnosis and diagnostic tests needed to confirm the diagnosis of 31 cases of cardiac syncope.

No.	Sex	Age	Diagnosis	ECG performance	Diagnostic tests
1	M	3 years and 10 months	Congenital LQTS	QT interval prolongation	ECG, Holt
2	F	12 years	Congenital LQTS	QT interval prolongation	ECG, Holt
3	M	5 years	Congenital LQTS	QT interval prolongation	ECG, Holt
4	M	2 years and 8 months	SSS	Type II second-degree sinoatrial block	ECG, Holt
5	F	6 years and 6 months	SSS	Atrial premature contraction	ECG, Holt, EPS
6	M	14 years	SSS	Sinus arrest	ECG, Holt
7	F	7 years and 7 months	PVT	VT, ventricular premature contraction	ECG, Holt
8	M	15 years	CAVB	CAVB	ECG
9	M	7 years and 7 months	SSS	Atrial tachycardia	ECG, Holt
10	F	5 years and 3 months	HCM	RVH	Echo
11	F	7 years	CAVB	CAVB	ECG
12	F	11 years and 10 months	IPAH	RVH, ST–T changes	Echo, Angiog
13	F	12 years	IPAH	RVH, ST–T changes	Echo, Angiog
14	F	4 years and 11 months	IPAH	RVH, ST–T changes	Echo
15	F	16 years	Congenital LQTS	QT interval prolongation	ECG, Holt
16	F	7 years and 6 months	HCM	RVH, ST–T changes	Echo
17	M	9 years	AF	AF	ECG, Holt
18	M	8 years	PSVT	Sinus rhythm	EPS
19	F	11 years and 9 months	PSVT	WPW syndrome	ECG, Holt
20	M	6 years and 1 months	SSS	Junctions verapamil arrhythmia, sinus arrest	ECG, Holt
21	F	11 years	SSS	Sinus bradycardia	ECG, EPS
22	M	4 years and 6 months	Poor pacemaker working	Cardiac arrest	ECG
23	F	18 years	SSS	Normal	Holt, EPS
24	F	12 years	Congenital LQTS	QT interval prolongation	ECG, Holt
25	M	5 years	AT	Premature atrial contraction	ECG, Holt
26	M	9 years	HCM	CVH	Echo
27	M	3 years and 5 months	IPAH	RVH	Echo
28	M	3 years and 4 months	AF	AF	ECG, Holt
29	M	13 years	HCM	RVH	Echo
30	F	3 years and 3 months	IPAH	CVH	Echo, Angiog
31	F	9 years and 4 months	Congenital LQTS	QT interval prolongation	ECG, Holt

AF = atrial fibrillation; Angiog = pulmonary arterial angiography; AT = atrial tachycardia; CAVB = complete atrioventricular block; CVH = combined ventricular hypertrophy; ECG = electrocardiogram; Echo = echocardiography; EPS = electrophysiological study; F = female; HCM = hypertrophic cardiomyopathy; Holt = Holter electrocardiogram monitoring; IPAH = idiopathic pulmonary arterial hypertension; LQTS = long QT syndrome; M = male; PSVT = paroxysmal supraventricular tachycardia; PVT = paroxysmal ventricular tachycardia; RVH = right ventricular hypertrophy; SSS = sick sinus syndrome; VT = ventricular tachycardia

symptoms was significantly less than that in children with vasovagal syncope. Only 51.6% of children had prodromal symptoms with cardiac syncope, whereas 87.3% of children with vasovagal syncope had prodromal symptoms. The frequency of dizziness and chest discomfort in children with cardiac syncope was significantly lower than that of children with vasovagal syncope. Other prodromal symptoms such as palpitations, headaches, and gastrointestinal symptoms occurred equally in children with cardiac syncope and vasovagal syncope.

In witnessed syncopal spells, the duration was less in cardiac syncope than in vasovagal syncope patients (1.8 versus 4.8 minutes, p less than 0.001).

More associated symptoms occurred in children with cardiac syncope than in those with vasovagal syncope. In particular, syncope with incontinence was seen more often in children with cardiac syncope than in children with vasovagal syncope, but there was no significant difference in the

frequency of physical injury between cardiac syncope and vasovagal syncope.

Family history of sudden death or syncope in both groups of children was less, but only three children – two with cardiac syncope and one with vasovagal syncope – were found to have positive family histories. In 31 cases of cardiac syncope, 5 had previous history of heart diseases, but only 2 of 55 cases of vasovagal syncope had previous history of heart disease (p less than 0.05). Children with cardiac syncope had a history of abnormal electrocardiogram more frequently than those with vasovagal syncope (93.5% versus 9.1%, p less than 0.001).

Logistic analysis of clinical features in children with cardiac syncope and vasovagal syncope

Multivariate logistic regression analysis showed that history of electrocardiogram abnormalities and exercise-triggered syncope were independent

Table 3. Predictors of cardiac syncope on univariate analysis in children and adolescents with syncope.

Clinical feature	Cardiac syncope (n = 31)	Vasovagal syncope (n = 55)	p-value
Age	8.5 ± 4.2	11.6 ± 2.6	0.001
Sex (male/female)	15/16	21/34	0.357
Course of disease (month)	19.4 ± 22.4	22.3 ± 14.2	0.566
Number of episodes	7.8 ± 17.6	5.0 ± 3.5	0.376
Predisposing factors	20/31 (64.5%)	43/55 (78.2%)	0.169
Persistent standing	1/31 (3.2%)	21/55 (38.2%)	0.002
Warm and crowded place	0	10/55 (18.2%)	
Fear–pain emotion	0	10/55 (18.2%)	
Exercise	19/31 (61.3%)	8/55 (14.5%)	0.000
Position			
Standing	22/31 (71.0%)	53/55 (96.4%)	0.001
Supine	8/31 (25.8%)	2/55 (3.6%)	0.002
Various position	6/31 (19.4%)	2/55 (3.6%)	0.016
With prodromal symptoms	16/31 (51.6%)	48/55 (87.3%)	0.000
Dizziness	1/31 (3.1%)	33/55 (60.0%)	0.000
Headache	2/31 (6.5%)	13/55 (23.6%)	0.044
Chest discomfort	3/31 (9.7%)	17/55 (30.9%)	0.025
Palpitations	6/31 (19.4%)	9/55 (15.5%)	0.645
Sweating	5/31 (16.1%)	7/55 (12.7%)	0.662
Pale	9/31 (29.0%)	11/55 (20.0%)	0.314
Nausea, vomiting	4/31 (12.9%)	15/55 (27.3%)	0.123
Blurred vision	4/31 (12.9%)	19/55 (34.5%)	0.130
Fatigue	1/31 (3.1%)	5/55 (9.1%)	0.473
Duration of the loss of consciousness (min)	1.8 ± 3.4	4.8 ± 3.8	0.000
≤1 min	17/31 (54.8%)	13/55 (23.6%)	
1–5 min	13/31 (41.9%)	14/55 (25.5%)	
>5 min	1/31 (3.1%)	28/55 (50.9%)	
Accompanying symptoms	17/23 (54.8%)	11/55 (20.0%)	0.001
Physical injury	2/31 (6.5%)	7/55 (12.7%)	0.905
Convulsion	4/23 (12.9%)	3/55 (5.5%)	0.251
Urine or faecal incontinence	8/31 (25.8%)	1/55 (1.8%)	0.001
Family history of sudden death or syncope	2/31 (6.5%)	1/55 (1.8%)	0.294
History of heart disease	5/31 (16.1%)	2/55 (3.6%)	0.042
Standard ECG abnormalities	29/31 (93.5%)	5/55 (9.1%)	0.000

ECG = electrocardiogram

predictors of cardiac syncope. Their odds ratio values and 95% confidence intervals were 180.670 and 19.9842–1633.574 (p less than 0.001), and 18.145, 1.840–178.892 (p less than 0.05), respectively. The sensitivity of history of abnormal electrocardiogram for predicting cardiac syncope was 93.5% and specificity 90.9%. The sensitivity of exercise-triggered syncope spell for predicting cardiac syncope was 61.3% and specificity 85.5%.

Discussion

Syncope is common in children, although the majority of paediatric syncope is benign. Vasovagal syncope is the most common cause of syncope in children. However, under some rare conditions, syncope may be a symptom of a potentially fatal heart disease.[3,5–7,15] Therefore, in clinical practice, children presenting with syncope present a diagnostic dilemma. For the purpose of excluding heart disease as a cause in children, expenses on medical resource utilisation, associated with syncope management, are enormous.[7,9,15,16] Many potential diagnostic approaches to this symptom complex remain difficult, expensive, and often unrewarding. History taking plays a key role in the initial evaluations of syncope.[8] Colman et al compared the characteristics of patients with prolonged QT syndrome with vasovagal syncope who were younger than 40 years of age, presenting to the emergency department. They found that a family history for syncope and sudden cardiac death, palpitation as a symptom, supine syncope, syncope associated with exercise, and emotion stress placed patients at higher risk for long QT syndrome. These findings should alert physicians to the potentially life-threatening illness of long QT syndrome. However, their study[17] did not include other causes of cardiac syncope. In previous studies, clinical variables have been found, which are predictive of cardiac syncope, and developed a diagnostic point score to discriminate cardiac syncope from syncope of other

causes. However, their study results were applicable only to adult patients.[9,10] The causes of syncope in children and adolescents differ from those of adult patients.[11] If we find the characteristics that may be shown as cardiac syncope based on a history of patients, the further diagnosis and treatment of syncope will be greatly improved. Therefore, the study was designed to compare characteristics of the history of the children who were diagnosed with cardiac syncope with that of the children with vasovagal syncope to find out the features that were important to the diagnosis of cardiac syncope.

Through the univariate analysis of the study, we found that the children with cardiac syncope were younger than those with vasovagal syncope, because vasovagal syncope mostly occurred in adolescents.[11] Most of the syncopal episodes in children with cardiac syncope were triggered by exercise. This result was consistent with previous studies in adults, and was consistent with the features of cardiac syncope noted in most of the syncope guidelines we reviewed.[8–10,12,18,19] Syncopal spells in children with vasovagal syncope were prevalent in the upright position, whereas cardiac syncope attacks could occur in the supine position, which, again, was consistent with the features of cardiac syncope that was noted in reviewed syncope guidelines.[8,19] Prodromal symptoms of syncope were relatively rare in cardiac syncope, when compared with vasovagal syncope, in children. The symptoms of autonomic nervous activation such as diaphoresis, nausea, vomiting, blurred vision, and pallor occurred similarly in both groups. There was activation of the autonomic nervous system in both types of syncopal attacks, which was similar to the results of Alboni's study.[12] Results of most studies and syncope guidelines indicated that the presence of palpitation could be used as one of the key clinical features in cardiac syncope.[8,10,19] Colman et al[17] also fund that palpitation, as a symptom, increased the risk of long QT syndromes. However, this study revealed that children with both types of syncope had a similar incidence of palpitation. The reason for such inconsistency was, mainly, due to the fact that children and caregivers did not clearly understand the meaning of palpitation. Therefore, this is why palpitations are not reliable prodromal symptoms in childhood cardiac syncope.

Syncope, accompanied with incontinence, was more common in children with cardiac syncope, when compared with children with vasovagal syncope. This, however, was inconsistent with Alboni's research.[12] Recently, Costanino et al[20] found an increased likelihood of adverse events within 10 days, in patients with syncope and accompanied by physical injury. Our study did not find that children with cardiac syncope had higher risk of injury, when compared with children with vasovagal syncope. Family history of sudden death was considered to be a feature of cardiac syncope, but this study did not find the similar results, perhaps because of fewer cases of cardiac syncope. It was known that the leading causes of cardiac syncope in children were mostly congenital and hereditary diseases, such as congenital long QT syndromes and hypertrophic cardiomyopathy.[7,8,19] Therefore, prospectively, children with a family history of sudden death should be carefully and thoroughly evaluated. The children who had a previous history of heart disease suffered from cardiac syncope more often than those with vasovagal syncope, which was consistent with a large number of research studies. Most cardiac syncope patients had a history of abnormalities on a standard 12-lead electrocardiogram, which can be easily obtained, and can establish the diagnosis of some cardiac anomalies, such as long QT syndromes and third-degree atrioventricular block. In addition, mechanical heart diseases can be manifested on electrocardiogram, such as hypertrophic cardiomyopathy and idiopathic pulmonary arterial hypertension. In our study, we found that the prevalence of history of abnormal electrocardiogram findings in children with cardiac syncope was higher than that with vasovagal syncope, and it predicted cardiac syncope in children and adolescents with high sensitivity and specificity. Most researchers and the syncope guidelines of European Society of Cardiology and American Heart Association believe that electrocardiogram examination is necessary and is the most basic examination for patients with syncope.[6–8,16,19] However, it should be noticed that the electrocardiogram findings of 2 cases in 31 cases of children with cardiac syncope were normal. Therefore, for patients with syncope with normal electrocardiogram findings, further evaluation is warranted, if there is other evidence of cardiac syncope, such as syncope during effort.

Through multivariable factors analysis, we found that the independent factors predicting for the cardiac syncope were history of abnormal electrocardiogram findings and exercise-triggered syncope, with the odds ratio value of 180.670 and 18.145, respectively. The sensitivity and specificity of prediction of a history of abnormal electrocardiogram were all above 90%. The sensitivity of exercise-triggered syncope for predicting cardiac syncope was 61.3%, and the specificity reached 85.5%. Therefore, for children with these two characteristics of syncope, the possibility of cardiac syncope should be strongly considered. Further examination for heart disease should be carried out, including Holter electrocardiogram monitoring, echocardiography, and cardiac electrophysiological examination, in order to determine the cause of syncope in children.

Our study still has limitations. First, in syncope no diagnostic "gold standard" has been determined and diagnosis often remains presumptive. Although the diagnostic criteria adhered strictly to the recommendations of guidelines, another competitive diagnosis cannot be excluded in some cases. Second, we did not develop a risk score based on clinical and history of electrocardiogram factors predicting cardiac syncope in children and adolescents. This would decrease the value of this study; however, this is the objective of a continuing study.

In conclusion, through our study, compared with vasovagal syncope in children, the patients with cardiac syncope have cardinal clinical features, especially history of abnormal electrocardiogram and exercise-triggered syncope. For children with syncope, a detailed medical history and routine electrocardiogram examination are necessary for identification of these clinical features, and further examination of heart disease for children with the above characteristics may enhance the efficiency and the accuracy of the diagnosis and save the patient and the family from intrusive procedures, lost time and money, and better utilise medical resources. The results of our study also can help general paediatricians to decide whether to hospitalise their patients with syncope.

Acknowledgement

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