
بسم الله الرحمن الرحيم

Comprehensive Management of Syncope in Children & Adolescents

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A Six-Part Systematic Framework for Pediatric Syncope

1. Epidemiology & Definitions
2. Pathophysiology & Etiologic Framework
3. Red Flags & Risk Stratification
4. The 12-Lead ECG: Reading for Lethal Causes
5. Advanced Workup: Monitoring, Exercise & Tilt
6. Management & Clinical types

Syncope Is Common in ; Cardiac Causes Are Rare but Critical

15–25%

Lifetime prevalence in youth

At least one episode before adulthood

Age 15

Peak age of onset

Peak incidence with female predominance

<5%

Cardiac etiologies

But carrying the highest mortality risk

- Syncope causes 1–3% of pediatric ED visits

Definition (1)

ESC
European Society of Cardiology

- **Syncope** is a TLOC, *due to transient global cerebral hypoperfusion,* characterized by rapid onset, short duration *and* spontaneous complete recovery.

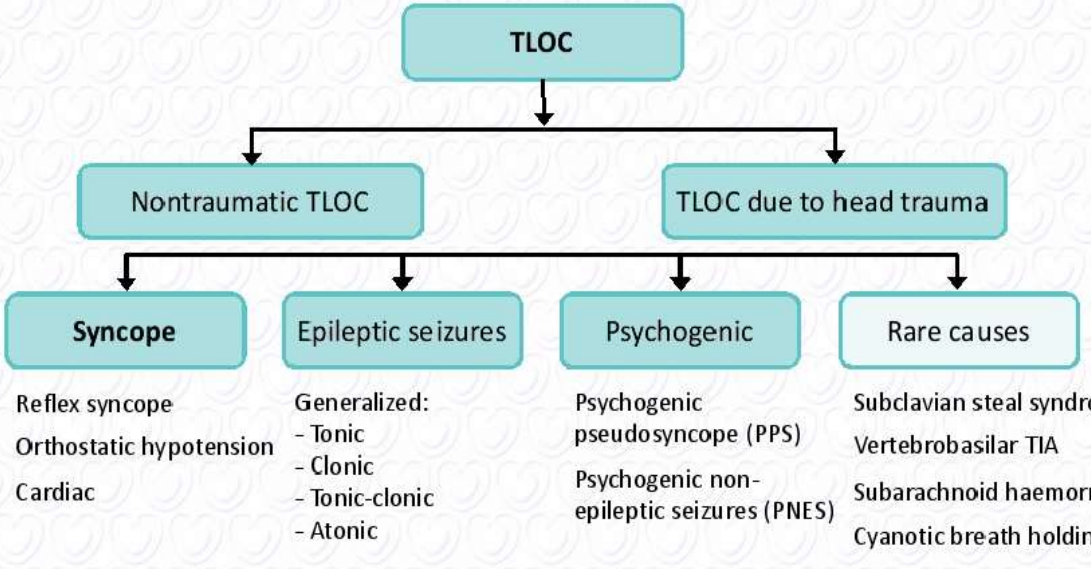
Definition (2)

ESC European Society of Cardiology

- **Transient loss of consciousness** (TLOC) is a state of real or apparent loss of consciousness with loss of awareness, characterized by amnesia for the period of unconsciousness, abnormal motor control, loss of responsiveness, and a short duration.
- **TLOC** is **syncope** when there is:
  a) presence of features specific for reflex, orthostatic hypotension, or cardiac syncope, *and;*
  b) absence of features specific for other forms of TLOC.

2018 ESC Guidelines on Syncope – Michele Brignole & Angel Moya
European Heart Journal 2018;39:1883–1948 - Doi:10.1093/eurheartj/ehy037

Classification



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Classification
Reflex (neurally-mediated) syncope

ESC European Society of Cardiology

- **Vasovagal:**
  - orthostatic VVS: standing, less common sitting,
  - emotional: fear, pain (somatic or visceral), instrumentation, blood phobia.
- **Situational:**
  - micturition,
  - gastrointestinal stimulation (swallow, defaecation),
  - cough, sneeze,
  - post-exercise,
  - others (e.g. laughing, brass instrument playing).
- **Carotid sinus syndrome.**
- **Non-classical forms** (without prodromes and/or without apparent triggers and/or atypical presentation.

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Classification
Syncope due to orthostatic hypotension

ESC European Society of Cardiology

- **Drug-induced OH (most common cause of OH):**
  - e.g. vasodilators, diuretics, phenothiazine, antidepressants.
- **Volume depletion:**
  - haemorrhage, diarrhoea, vomiting, etc.
- **Primary autonomic failure (neurogenic OH):**
  - pure autonomic failure, multiple system atrophy, Parkinson's disease, dementia with Lewy bodies.
- **Secondary autonomic failure (neurogenic OH):**
  - diabetes, amyloidosis, spinal cord injuries, auto-immune autonomic neuropathy, paraneoplastic autonomic neuropathy, kidney failure.

Note. Hypotension may be exacerbated by venous pooling during exercise (exercise-induced), after meals (postprandial hypotension), and after prolonged bed rest (deconditioning).

Classification
Cardiac syncope

ESC European Society of Cardiology

Arrhythmia as primary cause

- **Bradycardia:**
  - sinus node dysfunction (including bradycardia/tachycardia syndrome),
  - atrioventricular conduction system disease.
- **Tachycardia:**
  - supraventricular,
  - ventricular.
- **Structural cardiac:** aortic stenosis, acute myocardial infarction/ischaemia, hypertrophic cardiomyopathy, cardiac masses (atrial myxoma, tumours, etc.), pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valves dysfunction.
- **Cardiopulmonary and great vessels:** pulmonary embolus, acute aortic dissection, pulmonary hypertension.

2018 ESC Guidelines on Syncope – Michele Brignole & Angel Moya
European Heart Journal 2018;39:1883–1948 - Doi:10.1093/eurheartj/ehy037

Classification

Conditions (of real or apparent LOC) which may be incorrectly diagnosed as syncope

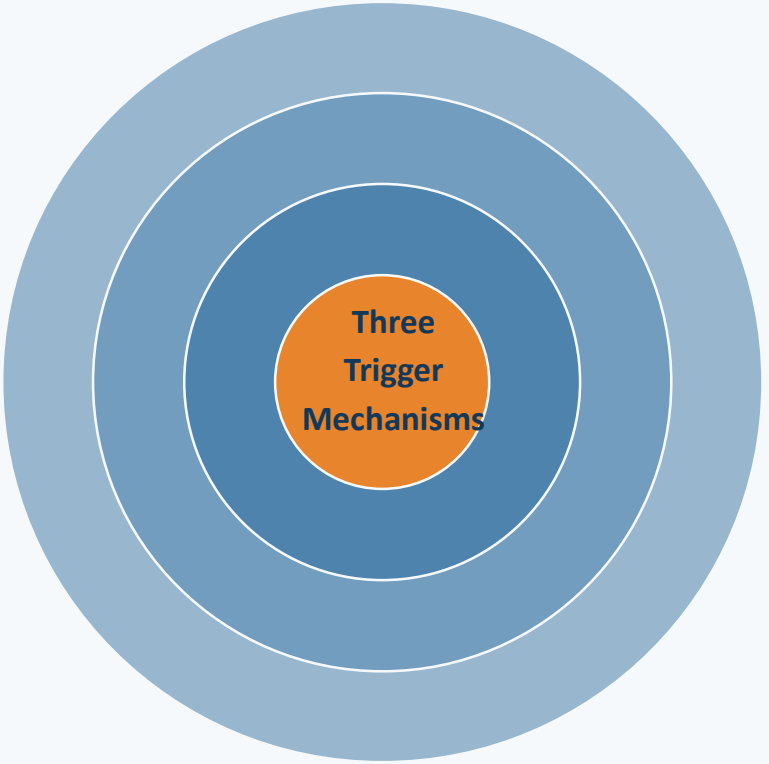
ESC European Society of Cardiology

- Generalized seizures, complex partial seizures, absence epilepsy.
- Psychogenic pseudosyncope.
- Falls without TLOC.
- Intracerebral or subarachnoid haemorrhage.
- Vertebrobasilar TIA.
- Carotid TIA.
- Subclavian steal syndrome.
- Cataplexy.
- Metabolic disorders including hypoglycaemia, hypoxia, hyper-ventilation with hypocapnia.
- Intoxication.
- Coma.
- Cardiac arrest.

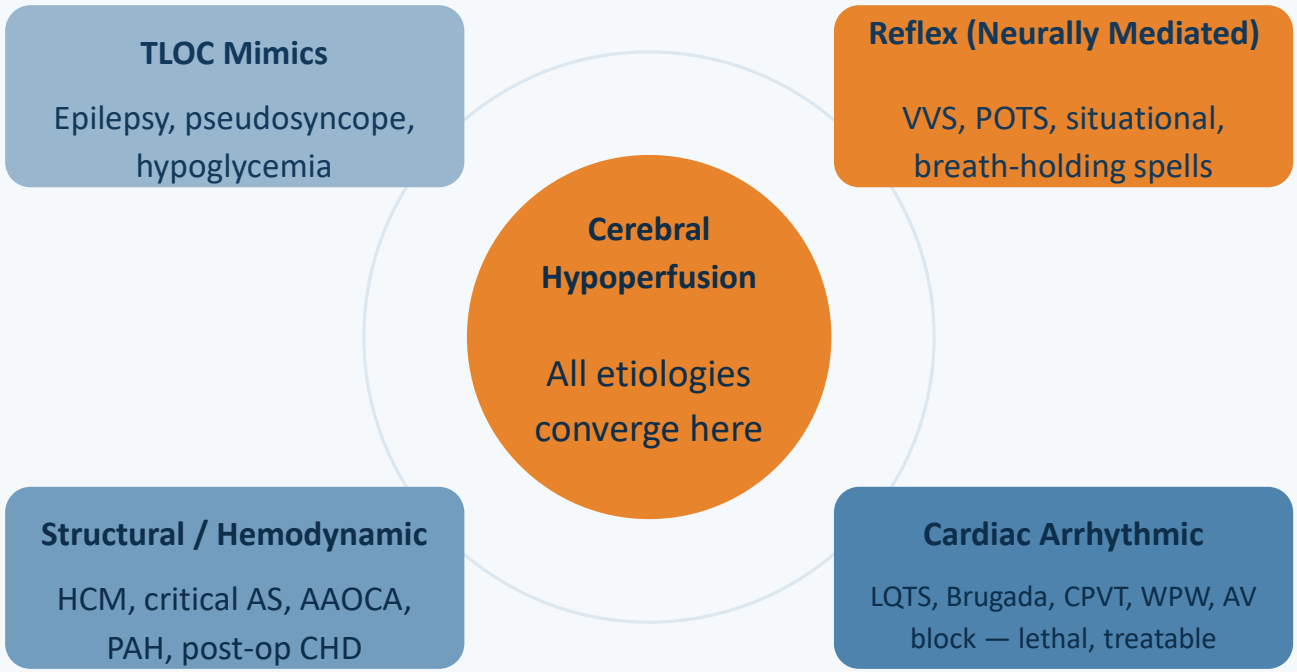
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Acute Cerebral Hypoperfusion Is the Final Common Pathway

- **Cerebral Hypoperfusion**
  The endpoint of every syncopal event
- **Brainstem & Cortex Fail**
  Loss of brainstem and cortical function with rapid recovery
- **Blood Pressure Falls**
  Blood pressure drops below the autoregulatory threshold
- **Three Trigger Mechanisms**
  Vasodilation, bradycardia, or low effective volume



A Four-Category Etiologic Framework for Pediatric Syncope



The Reflex (Neurally Mediated) Syncope Spectrum in Children

Vasovagal (VVS)

VVS, POTS, situational reflexes, breath-holding spells

POTS

Heart rate rises upright without BP drop; chronic daily dizziness

Situational Reflex

Cough, micturition, swallow, needle- or pain-provoked events

Breath-Holding

Cyanotic and pallid spells in infants and toddlers

Orthostatic Hypotension

Commonest benign cause; positive family history frequent

Pathophysiologic Cascade of Vasovagal Syncope

1

1. Upright Trigger

Venous pooling lowers central volume; sympathetic withdrawal follows

2

2. C-Fibers Fire

Vigorous contraction of the underfilled ventricle fires mechanoreceptors

3

3. Reflex Arc Fires

Central reflex shifts to vagal outflow with sympathetic withdrawal

4

4. Vasodepression

Hypotension plus bradycardia drop perfusion below threshold

5

5. Collapse & Recovery

Brief LOC with rapid recovery once supine restores flow

Pathophysiology of Neurally Mediated Syncope

1

1 · Venous Pooling

Standing shifts 500-800 mL of blood to splanchnic and leg vessels

2

2 · Reduced Return

Reduced preload; vigorous contraction of an underfilled ventricle

3

3 · Bezold-Jarisch

C-fiber afferents trigger vagal surge with sympathetic withdrawal

4

4 · Sympathovagal Shift

Bradycardia, vasodilation — nausea, pallor, warmth prodrome

5

5 · Hypoperfusion

BP below autoregulatory threshold -> transient LOC

Arrhythmic Syncope: The Silent Threat of Sudden Death

Long QT Syndrome

Repolarization abnormality; QTc >480 ms; event with exertion, swimming, emotion or auditory triggers

Brugada Syndrome

Type 1 ST elevation V1–V3; fever unmasking; South-East Asian male predominance

CPVT

Normal resting ECG; adrenergic bidirectional/polymorphic VT; exertional syncope

WPW Pre-excitation

Short PR with delta wave; AF with rapid conduction risks ventricular fibrillation

Conduction Disease

Congenital AV block, post-operative heart block, sinus node dysfunction

Structural and Hemodynamic Cardiac Causes of Syncope in CHD

1

Obstructive Lesions

Extreme LV outflow obstruction with exertion; systolic ejection murmur; also myocarditis and dilated cardiomyopathy

2

Coronary Anomalies (AAOCA)

Anomalous coronary origin with interarterial course compresses during exertion; a leading cause of SCD in young athletes

3

Pulmonary Hypertension

Elevated pulmonary vascular resistance limits exercise cardiac output; syncope is a poor prognostic sign

4

Post-Surgical CHD

Repaired tetralogy of Fallot with pulmonary regurgitation or ventricular tachycardia; Fontan circulation with limited reserve; conduit obstruction

Critical Differential: Convulsive Syncope vs. True Epilepsy

Convulsive Syncope

- Prodrome: lightheadedness, visual graying, warmth — vagal signs
- Trigger: upright posture, emotional stress, or painful stimulus
- Duration: brief (<30 s) LOC with a few myoclonic jerks
- Postictal: rapid, clear recovery without confusion
- Incontinence and tongue biting rare; EEG normal

True Epilepsy

- Prodrome: aura, déjà vu, or none — sudden onset
- Trigger: usually none; may occur supine or asleep
- Duration: typically >1 minute with tonic-clonic activity
- Postictal: prolonged confusion, headache, sleepiness
- Incontinence and tongue biting common; EEG may show epileptiform discharges

Upright trigger with rapid recovery favors convulsive syncope — do not stop at an epilepsy label.

Risk-Stratification Matrix: Red Flags Mandating Cardiac Workup

Background risk

High-Risk Events

Syncope during exertion; syncope while supine or asleep — presume cardiac until proven otherwise

Unheralded Onset

No prodrome or sudden LOC; palpitations immediately preceding collapse — arrhythmic signature

Family History

SCD <40y in family; inherited channelopathy or cardiomyopathy; congenital deafness with LQTS

Exam & ECG Findings

Abnormal ECG, murmur, chest pain with exercise, known CHD — proceed to echo and specialist referral

Event characteristics

12-Lead ECG: The Cornerstone Class I Test in Pediatric Syncope

The Cornerstone Test

Cheap, universal, and the highest-yield test — a Class I indication in every patient with syncope. It screens channelopathies, pre-excitation, and conduction disease.

01

QTc >460–480 ms

Bazett or Hodges formula; repeat when borderline QTc = QT + 1.75 × (HR - 60),

02

Delta Wave / Short PR

Short PR with wide QRS in any lead

03

Brugada Type 1 Pattern

Coved ST elevation V1–V3 in right precordial leads

04

T-Wave Inversion

T-wave inversion V1–V3 suggests arrhythmogenic cardiomyopathy

Lethal Channelopathies on ECG: LQTS, Brugada, CPVT

LQTS & CPVT

- QTc >460 ms (girls) / >450 ms (boys); notched T waves
- LQT1: exertion/swimming; LQT2: auditory/startle; LQT3: sleep
- Genotype-specific beta-blockers; nadolol often preferred in children
- CPVT (Catecholaminergic polymorphic V. tach) : normal resting ECG, exercise-induced
- bidirectional VT

CPVT: nadolol/propranolol ± flecainide; ICD reserved for refractory cases

Brugada & Diagnostic Pitfalls

- Coved type 1 ST elevation in V1–V3; unmasked by fever
- Type 2 pattern requires fever/drug challenge for confirmation
- Beware sinus bradycardia of athletes masking QT measurement errors
- Family screening cascade once a channelopathy is identified
- Shared decision-making for ICD in survivors of cardiac arrest

WPW, ACM, and Conduction Disease: Key ECG Markers

WPW Pre-excitation

Short PR (<120 ms) with delta wave and wide QRS; risk of AF with rapid conduction and ventricular fibrillation in a minority

Arrhythmogenic Cardiomyopathy (ACM)

Epsilon waves, prolonged terminal activation, T-wave inversion V1–V3; PVCs of left bundle morphology

Conduction Disease

Congenital complete AV block with wide QRS escape, prolonged QT, or ventricular dysfunction → pacemaker; post-operative heart block after CHD repair

First-Line Evaluation: The Detailed History

Circumstances

First episode timing, posture, activity, and environment at onset

Prodromes

Nausea, warmth, pallor, visual graying before the event

Trigger Analysis

Pain, fear, emotional stress, prolonged standing, crowded heat

Recovery Phase

Rapid spontaneous recovery without postictal confusion

Episode Count

Number of episodes, situational patterns, prior evaluations

Diagnostic Yield >50%

History alone establishes the cause in over 50% of cases

Family History: Sudden Cardiac Death Screening

01

SCD < 50 Years

Unexplained SCD in relatives under 50: sleep, exercise, or no cause

02

Inherited Disease

LQTS, Brugada, CPVT, HCM, or ACM in the extended pedigree

03

Concealed Events

Unexplained drownings, crashes, or sudden infant deaths at home

04

Devices & Pseudo-Epilepsy

Pacemaker or ICD in a parent before 50; resistant 'epilepsy' in family

05

Genetic Workup

Genetic testing when family SCD, long QT, or CPVT is suspected

Targeted Physical & Neurological Examination

General & Cardiac Exam

- Growth, nutrition, and syndromic appearance
- Dysmorphology and Marfan stigmata: arm span, joints, pectus
- Skin color, perfusion, central cyanosis
- Precordial auscultation: murmurs, clicks, gallops

Orthostatic & Neuro Exam

- Orthostatic BP: supine, then 3 minutes standing
- Abnormal: >=30 mmHg systolic or >=10 mmHg diastolic drop
- Focused neurological exam: tone, reflexes, gait
- Vital signs: baseline heart rate and rhythm

Differential Diagnosis: Syncope vs Mimics

Discriminator	Syncope	Epilepsy	Pseudosyncope	Breath-Holding
Trigger	Upright, pain, heat	Fever, photic	Audience, conflict	Crying, anger
Duration	Seconds	1-3 minutes	Prolonged	Under 1 minute
Motor activity	Brief myoclonic jerks	Tonic-clonic	Asynchronous thrashing	None; pallid
Incontinence	Uncommon	Common	Rare	Rare
Post-event state	Rapid recovery	Slow, confused	Rapid recovery	Rapid recovery
First test	12-lead ECG	EEG, brain MRI	Psychological evaluation	Clinical; EEG if atypical

Diagnostic Tier 1

Mandatory 12-Lead ECG Analysis: Core Parameters

Core parameters and pathological cutoffs for pediatric screening

01 Heart Rate: Persistent resting bradycardia (<50 bpm) or unexplained tachycardia warrants evaluation.

02 PR Interval: Normal 0.10–0.20 s; PR >0.20 s = first-degree AV block; PR <0.10 s suggests pre-excitation.

03 QRS Duration: Normal ≤0.10 s; QRS >0.12 s indicates bundle branch block or ventricular conduction delay.

04 QTc (Bazett): Abnormal if >440 ms (males), >460 ms (females), or <340 ms (short QT).

Electrical Disorders

ECG Channelopathies & Inherited Arrhythmias

Primary channelopathies, accessory pathways, and cardiomyopathy markers

Long QT: LQT1 — broad T on exertion; LQT2 — bifid T on auditory trigger; LQT3 — late T at rest.

Brugada: Coved ST elevation ≥2 mm in V1–V3 (Type 1); Types 2/3 show saddle-back morphology.

WPW: Short PR (<120 ms), delta wave, widened QRS; risk of pre-excited SVT/AF.

ARVC: Epsilon wave at terminal QRS, localized delay in V1–V3, T-wave inversion.

Risk Stratification

High-Risk Red Flags & ER Admission Criteria

Clinical features mandating emergent hospital admission and cardiac workup

High-Risk Clinical Red Flags

- Syncope during moderate-to-severe physical exertion or swimming
- Sudden collapse with zero prodromal warning or aura
- Syncope occurring in supine position or during sleep
- Family history of early sudden unexplained death (<50 years)
- Known structural congenital heart disease or cardiomyopathy

ER Admission & Workup Mandate

- Immediate emergency department transfer and telemetry admission
- Continuous 12-lead ECG monitoring and urgent pediatric cardiology consult
- Immediate transthoracic echocardiography for structural evaluation
- Exercise stress testing and advanced genetic/electrophysiology review
- Avoid discharge until lethal cardiac etiology is definitively ruled out

Diagnostic Tier 2

Echocardiography: Targeted Use, Not Routine Screening

1

Indications for Echo

Suspect structural disease: exertional or supine syncope, no prodrome, murmur, known CHD, family history of cardiomyopathy/SCD, or abnormal ECG.

2

Routine Echo Not Recommended

Typical vasovagal presentation with normal exam and ECG gains no diagnostic value; routine echo raises cost, anxiety, and downstream over-testing.

Advanced Diagnostic Workup: Monitoring, Exercise & Tilt Testing

1

1. History + ECG

History, red-flag screen, and 12-lead ECG complete the first pass

2

2. Echocardiography

Class I when structural disease, exertional syncope, or abnormal murmur is suspected

3

3. Exercise Test

Class I for exertional syncope — reproduces catecholamine-driven arrhythmias (CPVT, HCM, AAOCA)

4

4. Rhythm Monitoring

Holter for daily events; patch or external loop recorder for weekly-to-monthly events

5

5. ILR if Rare Events

Implantable loop recorder for rare, unexplained, suspected arrhythmic events

Long-Term Rhythm Monitoring: Matching the Device to Event Frequency

Device	Event Frequency	Diagnostic Yield	Best Suited For
24–48 h Holter	Daily events	Low	Frequent palpitations
Patch monitor (14–30 d)	Weekly events	Moderate	Weekly-monthly events
External loop recorder	Monthly events	Good	Infrequent presyncope
Implantable loop recorder	Rare events (1–2/y)	Highest	Unexplained syncope

Match the monitoring window to the interval between events — a 24-hour Holter will almost never capture a monthly event.

Recommendations for Pediatric Syncope

COR	LOE	Recommendations
I	C-LD	**VVS evaluation, including a detailed medical history, physical examination, family history, and a 12-lead ECG, should be performed in all pediatric patients presenting with syncope.**[315,618,620,624–630]
See Online Data Supplement 40.		Although VVS is the most common cause of pediatric syncope, cardiac syncope does represent 1.5% to 6% of pediatric cases (usually defined as up to 18 years of age).[617,619,620,629,631,632] Characteristics of presenting signs and symptoms differentiating VVS from cardiac causes of syncope are generally similar to those in adults. A family history of VVS and early SCD should be sought. VVS occurs in 33% to 80% of children with syncope.[624,628] Risk factors that raise suspicion of a cardiac etiology include the absence of prodromal symptoms, presence of preceding palpitations within seconds of loss of consciousness, lack of a prolonged upright posture, syncope during exercise or in response to auditory or emotional triggers, family history of SCD, abnormal physical examination, and abnormal ECG,[626,627] although the specificity is modest.[618,627,630,633] It should be remembered that children may not be able to clearly communicate specific symptoms. Exertional syncope has been associated with LQTS and CPVT.[315,318,337,630,634] Regardless of symptoms, exertional syncope, especially mid-exertional syncope, should result in a high index of suspicion for a cardiac etiology.[633]
I	C-LD	**Noninvasive diagnostic testing should be performed in pediatric patients presenting with syncope and suspected CHD, cardiomyopathy, or primary rhythm disorder.**[315,318,618,625,627,630,633]
See Online Data Supplement 40.		Channelopathies are major causes of cardiac-related syncope in young people. They may be associated with a family history of SCD, and they increase the risk of SCD in these patients.[315,337,630,632,634,635] Exercise stress testing may be helpful in the diagnosis of channelopathies, such as LQTS and CPVT, which have adrenergically mediated arrhythmias. Extended monitoring is reasonable when an arrhythmia diagnosis is suspected. The types of monitoring devices, their clinical utility, and their limitations are available in Table 8. Prolonged heart rhythm monitoring can often provide a correlation between symptoms and an arrhythmia. In 5 retrospective studies of prolonged monitoring in 87 children with either syncope or presyncope, the mean diagnostic yield was 43%.[636–640] Bradyarrhythmias and high-grade AV block or asystole, as well as tachyarrhythmias, SVT, and polymorphic VT, were documented.[636–640] The diagnostic yield of an ICM is higher if the clinical indication was exertional syncope or the patient had underlying CHD.[637,639,640]
I	C-EO	**Education on symptom awareness of prodromes and reassurance are indicated in pediatric patients with VVS.**
See Online Data Supplement 40.		Management of children with VVS should include reassurance about the generally benign nature of this condition.[641,642] Treatment should emphasize symptom awareness and avoidance of precipitating factors that might worsen the condition, such as dehydration, standing for prolonged periods of time, hot crowded environments, and diuretic intake.

Rhythm Monitoring Depth, Exercise Testing & Tilt: Yields and Limits

1

Holter: Low Yield

Yield <10% in unselected pediatric syncope — reserve for daily suspected arrhythmia

2

Patch / Loop Recorder

Better correlation; patches capture 90%+ of weekly events

3

ILR: Highest Yield

Highest yield in unexplained syncope with suspected arrhythmia; 2–3 years of coverage

4

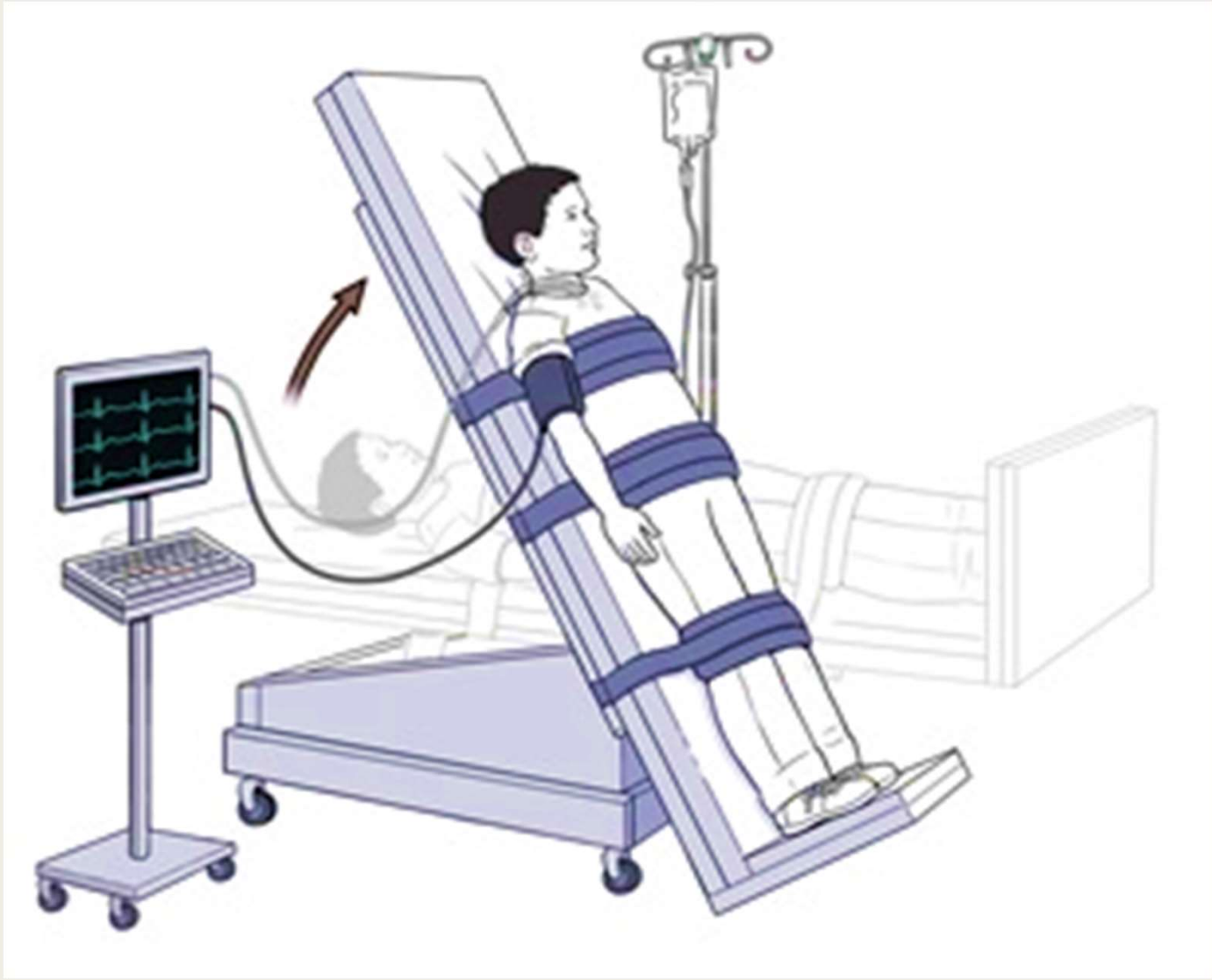
Exercise Test

Essential for exertional syncope; reproduces CPVT, HCM, and coronary compression

5

Tilt Test: Limited

Selective role in pediatrics; not needed to confirm classic vasovagal presentations



ED Triage: Admission Red Flags vs. Safe Discharge Pathway

Admit / Cardiac Workup

Exertional/supine syncope, no prodrome, palpitations, abnormal ECG or murmur, family history of SCD <40y

Safe Discharge

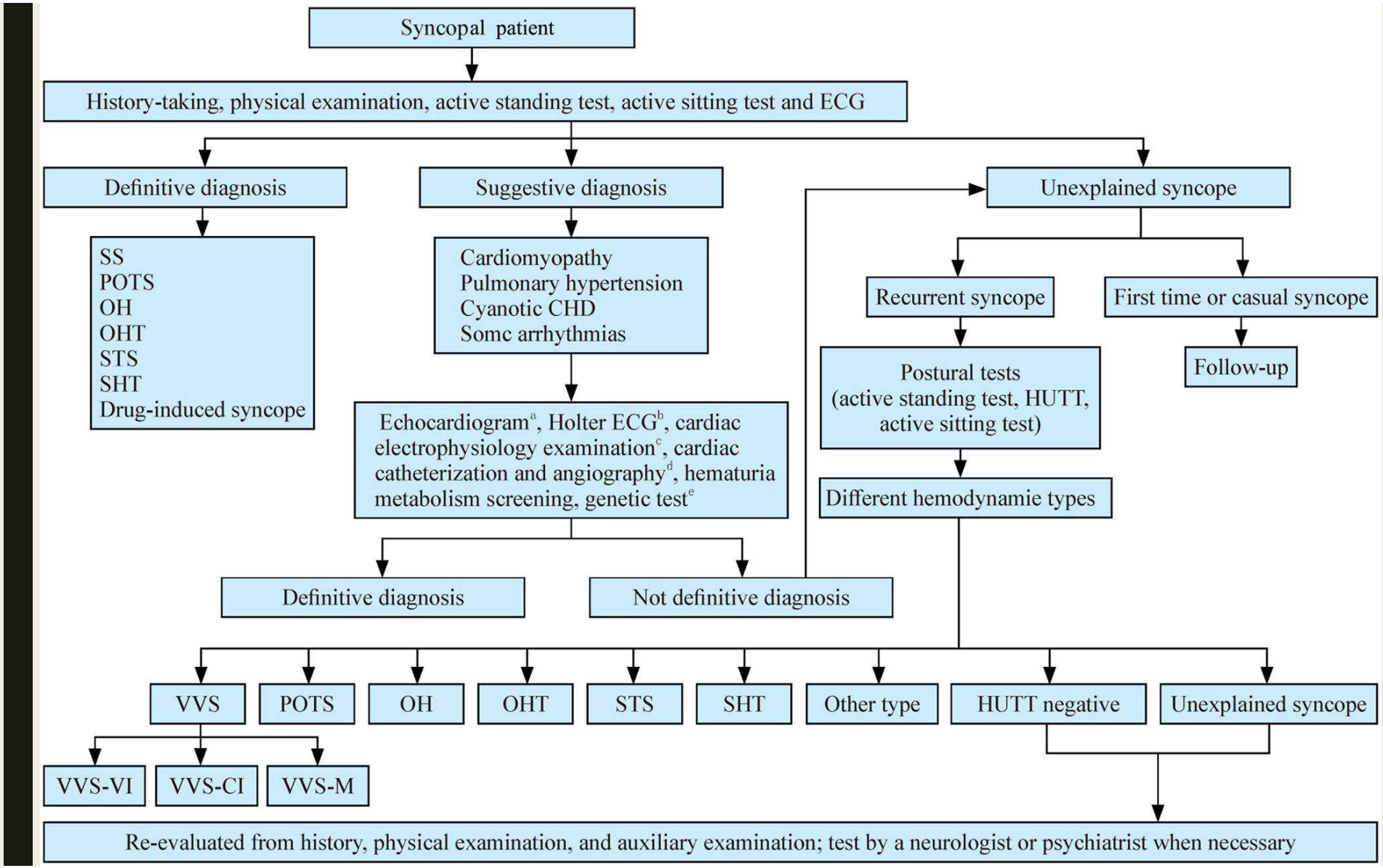
Typical vasovagal history, normal ECG and exam, no red flags

Emergent Measures

Ongoing chest pain, abnormal vital signs, or unstable arrhythmia

Discharge Counseling

Hydration, salt, counter-pressure maneuvers, review triggering medications


Syncopal patient
History-taking, physical examination, active standing test, active sitting test and ECG
Definitive diagnosis
Suggestive diagnosis
Unexplained syncope
SS
POTS
OH
OHT
STS
SHT
Drug-induced syncope
Cardiomyopathy
Pulmonary hypertension
Cyanotic CHD
Somc arrhythmias
Recurrent syncope
First time or casual syncope
Follow-up
Postural tests
(active standing test, HUTT,
active sitting test)
Echocardiogram[a], Holter ECG[b], cardiac electrophysiology examination[c], cardiac catheterization and angiography[d], hematuria metabolism screening, genetic test[e]
Different hemodynamie types
Definitive diagnosis
Not definitive diagnosis
VVS
POTS
OH
OHT
STS
SHT
Other type
HUTT negative
Unexplained syncope
VVS-VI
VVS-CI
VVS-M
Re-evaluated from history, physical examination, and auxiliary examination; test by a neurologist or psychiatrist when necessary

Evidence-Based Management: Lifestyle vs. Targeted Therapy

Reflex Syncope: Lifestyle First

- Education and reassurance — the majority need nothing more
- Aggressive hydration and liberalized salt intake
- Counter-pressure maneuvers at the prodrome
- Supine positioning at symptom onset prevents injury
- Beta-blockers NOT routine (Class III)

Cardiac Syncope: Targeted Therapy

- LQTS/CPVT: beta-blockers ± flecainide; ICD if high risk
- Brugada: fever avoidance; quinidine or ICD if high risk
- WPW with syncope: EP study and catheter ablation
- AV block: pacemaker implantation
- Structural disease: treat the lesion directly

Reassurance and volume expansion for the benign majority; targeted rhythm therapy for the lethal minority.

Recommendations for Pediatric Syncope AHA 2017

IIa	C-LD	**Tilt-table testing can be useful for pediatric patients with suspected VVS when the diagnosis is unclear.**[624,629,643-650]
See Online Data Supplement 40.		Tilt-table testing has a diminishing role in the diagnosis of children with unexplained syncope. The sensitivity of tilt-table testing ranges from 20% to 90%,[624,629,643,644,647,648,651,652] and the specificity ranges from 83% to 100%.[624,643,652] Pediatric patients with episodes of VVS may exhibit convulsive movements during loss of consciousness that mimic epileptic seizures. In children with syncope and convulsions on tilt-table testing, 64% exhibited cardiac asystole with pauses >3 seconds.[645] Upright tilt-table testing combined with a graded isoproterenol infusion identified 42% to 67% of patients previously thought to have a primary seizure disorder.[223,649] A combined cardiology and neurology evaluation may be warranted in this group of patients with syncope and seizure-like activity.
IIa	B-R	**In pediatric patients with VVS not responding to lifestyle measures, it is reasonable to prescribe midodrine.**[381,620,653]
See Online Data Supplement 40.		In a single-center prospective case series, pseudoephedrine reduced clinical symptoms in 94% of children with recurrent neurally mediated syncope.[653] In an RCT comparing patients receiving conventional therapy (health education, tilt-table training, and salt) and midodrine with patients receiving conventional therapy alone, the recurrence rate of syncope decreased from 80% to 22%.[381] In 2 prospective studies, side effects from midodrine were rare.[381,653]
IIb	B-R	**Encouraging increased salt and fluid intake may be reasonable in selected pediatric patients with VVS.**[642]
See Online Data Supplement 40.		In an RCT, conventional therapy and oral rehydration salts resulted in no further recurrence of syncope in 56% of patients, versus 39% in the placebo arm ($P<0.05$).[642]
IIb	C-LD	**The effectiveness of fludrocortisone is uncertain in pediatric patients with OH associated with syncope.**[380,654,655]
See Online Data Supplement 40.		In 2 single-center prospective case series of 0.1 mg of fludrocortisone, 83% of subjects demonstrated improvement or resolution of symptoms.[654,655] In the only pediatric RCT, children with recurrent syncope did better on placebo than on fludrocortisone.[380]
IIb	B-NR	**Cardiac pacing may be considered in pediatric patients with severe neurally mediated syncope secondary to pallid breath-holding spells.**[656,657]
See Online Data Supplement 40.		In 2 separate studies of 22 predominantly infants and toddlers with reflex anoxic seizures, pallid breath-holding spells, and documented prolonged asystole (pauses >4 seconds), 86% had either complete resolution or a significant reduction in the number of syncopal events with pacing.[656,657] Although the studies were not powered to address the specifics of pacing programming, either single- or dual-chamber pacing significantly reduced the number of syncopal episodes compared with a sensing-only strategy.[656,657] Single-chamber pacing with hysteresis appears as effective as dual-chamber pacing with rate drop response for the prevention of syncope and seizures. The beneficial response to pacing in these studies cannot exclude a placebo effect from pacemaker implantation itself; however, the young age of the patients with pallid breath-holding spells makes placebo effect less likely. The long-term outcome with pacing in this population has not been reported. Finally, it is important to remember that pallid breath-holding syncope does end, although some patients do present again at a later age with classic VVS. This should be balanced against the known complications of permanent cardiac pacing.
III: No Benefit	B-R	**Beta blockers are not beneficial in pediatric patients with VVS.**[655,658]
See Online Data Supplement 40.		In an RCT comparing metoprolol and conventional therapy, the treatment group actually had a higher recurrence rate. Side effects of beta blockers occur frequently in children.[655,659]



Leg-cross / muscle tensing



Squat



Arm-tensing / Hand-grip



Tilt training

Clinical Types: Red Flags in Action

1

Type 1: Exertional Syncope

Boy, 15 — faints during basketball, no prodrome. ECG: QTc 510 ms. Sibling died suddenly at 19. → Presume LQTS; beta-blocker, screen relatives.

2

Type 2: Prolonged Standing

Girl, 14 — faints standing in a hot classroom, prodrome of graying vision. ECG normal. → Classic vasovagal; hydration, salt, maneuvers, reassurance.

3

Type 3: Post-Operative CHD

Child, 12 — repaired tetralogy of Fallot, syncope while running with palpitations. Holter: sustained VT. → Admit; EP referral for ablation.

Every Child With Syncope Deserves a 12-Lead ECG

History, red flags, ECG — every child.

دستورالعمل بالینی تشخیص، مدیریت و درمان کودکان و نوجوانان مبتلا به سنکوپ

تدوین و گردآوری:

دکتر علیرضا احمدی:

فوق تخصص قلب کودکان، استاد، عضو هیات علمی دانشگاه علوم پزشکی اصفهان

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Original Article

The Impact of Self-Care Recommendations with and without Tilt-Training on Quality of Life in Children and Adolescents with Vasovagal Syncope: A Randomized Clinical Trial

Abstract

Background: Syncope is among the most common paroxysmal disorders in children and adolescents. Vasovagal syncope is the most common syncope in children and adolescents. The aim of this study was to evaluate the impact of self-care recommendations with and without tilt training on the Quality of Life (QoL) of children and adolescents with syncope. **Materials and Methods:** This randomized controlled clinical trial was conducted in Isfahan, Iran, from April 2017 to June 2021 and included 120 patients with syncope. Eligible children and adolescents (aged 6–18 years) who met inclusion criteria were recruited by the simple sampling method and then assigned randomly into two groups. The intervention group (n = 60) received routine self-care recommendations such as dietary advice, behaviors to prevent syncope, and counter-pressure maneuvers along with tilt training, while the control group (n = 60) received self-care recommendations without tilt training. The education training included two face-to-face sessions, each of which lasted for 45–60 min. Then, both groups were followed up by telephone (once a month) for six months. A researcher-made self-care questionnaire and Pediatric Quality of Life Inventory (PedsQL™ 4.0) were completed for both groups before and after the intervention. Data were analyzed using descriptive and inferential statistical methods. **Results:** The Wilcoxon test results showed a significant difference in the mean scores of physical functioning, emotional functioning, social functioning, school functioning, psychosocial functioning, understanding of health, and total QoL in the intervention and control groups before and after the intervention ($p < 0.05$). Also, the paired t-test results showed a significant difference in the mean scores of self-care domains and total self-care in the intervention and control groups before and after the intervention ($p < 0.05$). **Conclusions:** Self-care recommendations with and without tilt training can improve QoL in children and adolescents with syncope.

Keywords: *Adolescent, behavior, child, quality of life, self-care, syncope*

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Conclusion

- The challenges in managing pediatric syncope primarily involve confirming a definitive diagnosis.
- Once a diagnosis is established, the next hurdle is obtaining consent from both the patient and their parents for follow-up care that focuses on lifestyle recommendations rather than a prescription for medication.


Thank
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