

LA SINCOPE

IL PUNTO DI VISTA DEL CARDIOLOGO

INTRODUZIONE

Management of Patients With Syncope

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society

Developed in

European Heart Journal (2018) 39, 1883-1948

2018 ESC Guidelines for the diagnosis and management of syncope

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Available on www.escardio.org/Guidelines



Table 3. Relevant Terms and Definitions*

Term	Definition/Comments and References
Syncope	A symptom that presents with an abrupt, transient, complete loss of consciousness, associated with inability to maintain postural tone, with rapid and spontaneous recovery. The presumed mechanism is cerebral hypoperfusion (24,30). There should not be clinical features of other nonsyncope causes of loss of consciousness, such as seizure, antecedent head trauma, or apparent loss of consciousness (i.e., pseudosyncope) (24,30).
Loss of consciousness	A cognitive state in which one lacks awareness of oneself and one's situation, with an inability to respond to stimuli.
Transient loss of consciousness	Self-limited loss of consciousness (30) can be divided into syncope and nonsyncope conditions. Nonsyncope conditions include but are not limited to seizures, hypoglycemia, metabolic conditions, drug or alcohol intoxication, and concussion due to head trauma. The underlying mechanism of syncope is presumed to be cerebral hypoperfusion, whereas nonsyncope conditions are attributed to different mechanisms.
Presyncope (near-syncope)	The symptoms before syncope. These symptoms could include extreme lightheadedness; visual sensations, such as "tunnel vision" or "graying out"; and variable degrees of altered consciousness without complete loss of consciousness. Presyncope could progress to syncope, or it could abort without syncope.
Unexplained syncope (syncope of undetermined etiology)	Syncope for which a cause is undetermined after an initial evaluation that is deemed appropriate by the experienced healthcare provider. The initial evaluation includes but is not limited to a thorough history, physical examination, and ECG.
Orthostatic intolerance	A syndrome consisting of a constellation of symptoms that include frequent, recurrent, or persistent lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, exercise intolerance, and fatigue upon standing. These symptoms can occur with or without orthostatic tachycardia, OH, or syncope (24). Individuals with orthostatic intolerance have ≥1 of these symptoms associated with reduced ability to maintain upright posture.

Terminologia e Classificazione

Orthostatic	A sustained increase in heart rate of ≥30 bpm within 10 min of moving from a recumbent				
tachycardia	to a quiet (nonexertional) standing position (or ≥40 bpm in individuals 12–19 y of age)				
	(24,30,31).				
Orthostatic	A drop in systolic BP of ≥20 mm Hg or diastolic BP of ≥10 mm Hg with assumption of an				
hypotension (OH)	upright posture (31).				
 Initial (immediate) 	A transient BP decrease within 15 s after standing, with presyncope or syncope (31,32).				
ОН					
Classic OH	A sustained reduction of systolic BP of ≥20 mm Hg or diastolic BP of ≥10 mm Hg within				
	3 min of assuming upright posture (31).				
Delayed OH	A sustained reduction of systolic BP of ≥20 mm Hg (or 30 mm Hg in patients with supine				
	hypertension) or diastolic BP of ≥10 mm Hg that takes >3 min of upright posture to				
	develop. The fall in BP is usually gradual until reaching the threshold (31).				
Neurogenic OH	A subtype of OH that is due to dysfunction of the autonomic nervous system and not				
	solely due to environmental triggers (e.g., dehydration or drugs) (33,34). Neurogenic OH				
	is due to lesions involving the central or peripheral autonomic nerves.				
Cardiac	Syncope caused by bradycardia, tachycardia, or hypotension due to low cardiac index,				
(cardiovascular)	blood flow obstruction, vasodilatation, or acute vascular dissection (35,36).				
syncope					
Noncardiac syncope	Syncope due to noncardiac causes which include reflex syncope, OH, volume depletion,				
	dehydration, and blood loss (35).				

Terminologia e Classificazione

Reflex (neurally	Syncope due to a reflex that causes vasodilation, bradycardia, or both (24,30,31).					
mediated) syncope						
 Vasovagal syncope 	The most common form of reflex syncope mediated by the vasovagal reflex. VVS 1) may					
(VVS)	occur with upright posture (standing or seated or with exposure to emotional stress, pain,					
	or medical settings; 2) typically is characterized by diaphoresis, warmth, nausea, and					
	pallor; 3) is associated with vasodepressor hypotension and/or inappropriate bradycardia;					
	and 4) is often followed by fatigue. Typical features may be absent in older patients (24).					
	VS is often preceded by identifiable triggers and/or by a characteristic prodrome. The					
	diagnosis is made primarily on the basis of a thorough history, physical examination, and					
	eyewitness observation, if available.					
Carotid sinus	Reflex syncope associated with carotid sinus hypersensitivity (30). Carotid sinus					
syndrome	hypersensitivity is present when a pause ≥3 s and/or a decrease of systolic pressure ≥50					
	mm Hg occurs upon stimulation of the carotid sinus. It occurs more frequently in older					
	patients. Carotid sinus hypersensitivity can be associated with varying degrees of					
	symptoms. Carotid sinus syndrome is defined when syncope occurs in the presence of					
	carotid sinus hypersensitivity.					
 Situational syncope 	Reflex syncope associated with a specific action, such as coughing, laughing, swallowing,					
	micturition, or defecation. These syncope events are closely associated with specific					
	physical functions.					

Terminologia e Classificazione

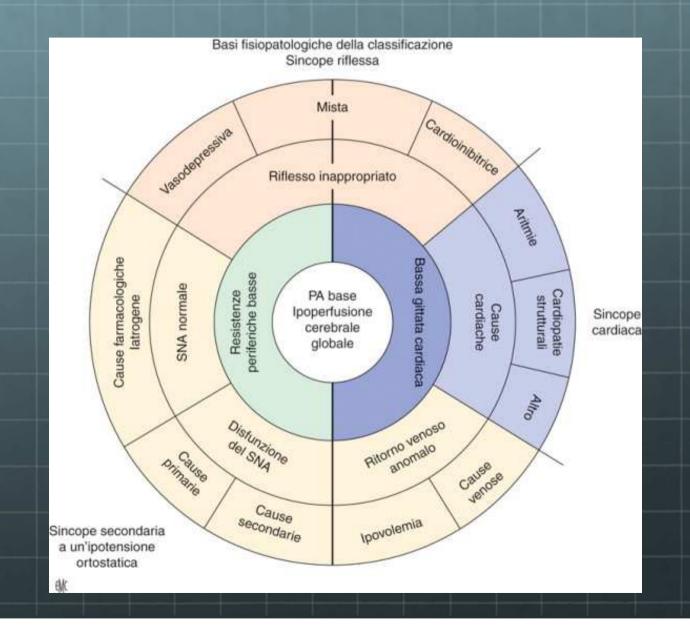
Postural (orthostatic) tachycardia syndrome (POTS)

A clinical syndrome usually characterized by all of the following: 1) frequent symptoms that occur with standing (e.g., lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, exercise intolerance, and fatigue); and 2) an increase in heart rate of ≥30 bpm during a positional change from supine to standing (or ≥40 bpm in those 12–19 y of age); and 3) the absence of OH (>20 mm Hg reduction in systolic BP). Symptoms associated with POTS include those that occur with standing (e.g., lightheadedness, palpitations); those not associated with particular postures (e.g., bloating, nausea, diarrhea, abdominal pain); and those that are systemic (e.g., fatigue, sleep disturbance, migraine headaches) (37). The standing heart rate is often >120 bpm (31,38-42).

Psychogenic pseudosyncope

A syndrome of apparent but not true loss of consciousness that may occur in the absence of identifiable cardiac, reflex, neurological, or metabolic causes (30).

Classificazione



Key messages

Diagnosis: initial evaluation



1. At the initial evaluation answer the following 4 key questions:

Question #1

Does the event concern TLOC?

If yes

Question #2

Is TLOC of syncopal origin?

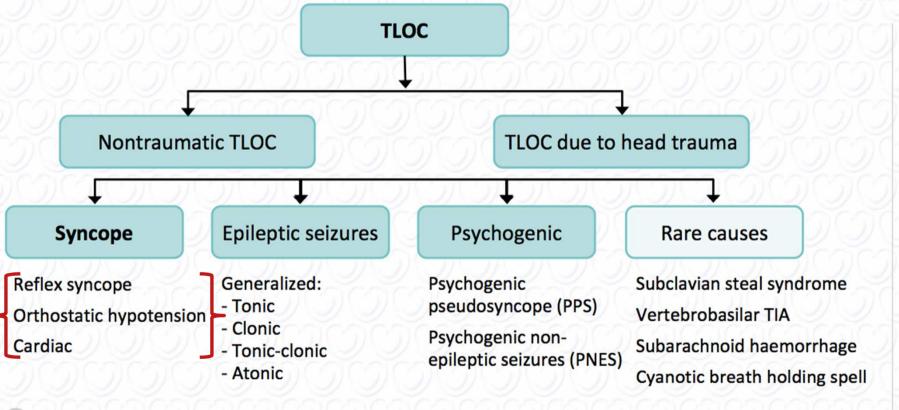
If yes

Question #3
Which is the risk?

Question #4
Is there a diagnosis?

Classification





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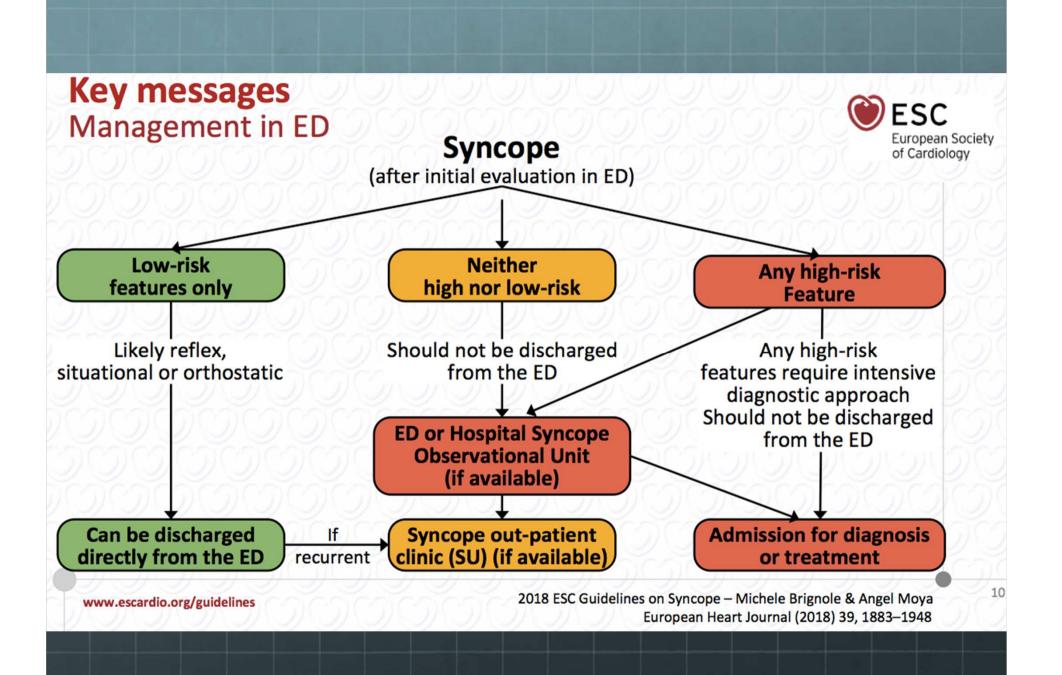
Classification

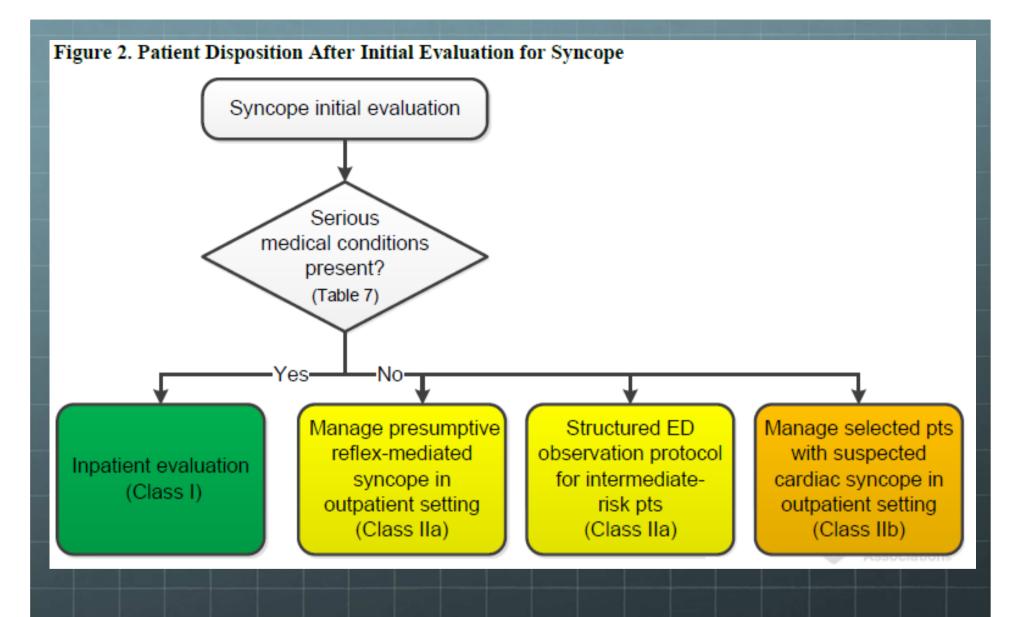
Conditions (of real or apparent LOC) which may be incorrectly diagnosed as syncope



- Generalized seizures, complex partial seizures, absence epilepsy.
- Psychogenic pseudosyncope.
- Falls without TLOC.
- Intracerebral or subarachnoid haemorrhage.
- Vertebrobasilar TIA.
- **SO** Carotid TIA.

- Subclavian steal syndrome.
- **50** Cataplexy.
- Metabolic disorders including hypoglycaemia, hypoxia, hyperventilation with hypocapnia.
- Intoxication.
- 🔊 Coma.
- Cardiac arrest.





"Intermediate" risk factors included the following: ≥50 years of age; prior history of cardiac disease, cardiac device without evidence of dysfunction, concerning ECG findings, or family history of early SCD; and symptoms not consistent with reflex-mediated syncope.

Management of syncope in the ED

Should the patient be admitted to hospital?



Favour initial management in ED observation unit and/or fast-track to syncope unit	Favour admission to hospital
unit and/or fast-track to syncope unit High-risk features AND: Stable, known structural heart disease. Severe chronic disease. Syncope during exertion. Syncope while supine or sitting. Syncope without prodrome. Palpitations at the time of syncope. Inadequate sinus bradycardia or sinoatrial block. Suspected device malfunction or inappropriate intervention. Pre-excited QRS complex. SVT or paroxysmal atrial fibrillation. ECG suggesting an inheritable arrhythmogenic	 High-risk features AND: Any potentially severe coexisting disease that requires admission. Injury caused by syncope. Need of further urgent evaluation and treatment if it cannot be achieved in another way (i.e. observation unit), e.g. ECG monitoring, echocardiography, stress test, electrophysiological study, angiography, device malfunction, etc. Need for treatment of syncope.
disorders. • ECG suggesting ARVC.	

Table 7. Examples of Serious Medical Conditions That Might Warrant Consideration of Further Evaluation and Therapy in a Hospital Setting

Cardiac Arrhythmic Conditions	Cardiac or Vascular Nonarrhythmic Conditions	Noncardiac Conditions
Sustained or symptomatic VT Symptomatic conduction system disease or Mobitz II or third-degree heart block Symptomatic bradycardia or sinus pauses not related to neurally mediated syncope Symptomatic SVT Pacemaker/ICD malfunction Inheritable cardiovascular conditions predisposing to arrhythmias	Cardiac ischemia Severe aortic stenosis Cardiac tamponade HCM Severe prosthetic valve dysfunction Pulmonary embolism Aortic dissection Acute HF Moderate-to-severe LV dysfunction	Severe anemia/gastrointestinal bleeding Major traumatic injury du to syncope Persistent vital sign abnormalities

HCM indicates hypertrophic cardiomyopathy; HF, heart failure; ICD, implantable cardioverter-defibrillator; LV, left ventricular; SVT, supraventricular tachycardia; and VT, ventricular tachycardia.

Table 5. Short- and Long-Term Risk Factors*

8	
Short-Term Risk Factors (≤30 d)	Long-Term Risk Factors (>30 d)
History: Outpatient Clinic or ED Evaluation	
Male sex (74,85,101,102)	Male sex (68,90)
Older age (>60 y) (88)	Older age (90)
No prodrome (68)	Absence of nausea/vomiting preceding syncopal event (93)
Palpitations preceding loss of consciousness (83)	VA (68,90)
Exertional syncope (83)	Cancer (68)
Structural heart disease (70,83,88,101,103)	Structural heart disease (68,103)
HF (74,83,85,88)	HF (90)
Cerebrovascular disease (70)	Cerebrovascular disease (68)
Family history of SCD (70)	Diabetes mellitus (104)
Trauma (68,101)	High CHADS-2 score (95)
Physical Examination or Laboratory Investigat	ion
	Abnormal ECG (84,90,93)
Evidence of bleeding (83)	Lower GFR
Persistent abnormal vital signs (70)	
Abnormal ECG (68,72,74,75,105)	
Positive troponin (75)	

^{*}Definitions for clinical endpoints or serious outcomes vary by study. The specific endpoints for the individual studies in this table are defined in Data Supplements 3 and 4 and summarized in Table 6 for selected studies. This table includes individual risk predictors from history, physical examination, and laboratory studies associated with adverse outcomes from selected studies.

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Study/Reference	Year	Sample N	Events N	Outcome Definition	ED Events*	Predictors	NPV (%)†
Martin (90)	1997	252	104 (41%)	1-y death/ arrhythmia	Yes	Abnormal ECG#; >45 y of age; VA; HF	93
Sarasin (74)	2003	175	30 (17%)	Inpatient arrhythmia	Yes	Abnormal ECG#; >65 y of age; HF	98
OESIL (67)	2003	270	31 (11%)	1-y death	N/A	Abnormal ECG#; >65 y of age; no prodrome; cardiac history	100
SFSR (72)	2004	684	79 (12%)	7-d serious events§	Yes	Abnormal ECG#; dyspnea; hematocrit; systolic BP <90 mm Hg; HF	99
Boston Syncope Rule (70)	2007	293	68 (23%)	30-d serious events‡	Yes	Symptoms of acute coronary syndrome; worrisome cardiac history; family history of SCD; VHD; signs of conduction disease; volume depletion; persistent abnormal vital signs; primary central nervous event	100
Del Rosso (69)	2008	260	44 (17%)	Cardiac etiology	N/A	Abnormal ECG#/cardiac history; palpitations; exertional; supine; precipitant (low-risk factor); autonomic prodrome (low-risk factors)	99
STePS (68)	2008	676	41	10-d serious	Yes	Abnormal ECG#; trauma; no	888

STePS (68)	2008	676	41 (6%)	10-d serious events	Yes	Abnormal ECG#; trauma; no prodrome; male sex	
Syncope Risk Score (75)	2009	2,584	173 (7%)	30-d serious events¶	No	Abnormal ECG#; >90 y of age; male sex; positive troponin; history of arrhythmia; systolic BP >160 mm Hg; near-syncope (a low-risk factor)	97
ROSE (73)	2010	550	40 (7%)	30-d serious events¶	Yes	Abnormal ECG#; B-natriuretic peptide; hemoglobin; O ₂ Sat; fecal occult blood	98

TAB. 8

OESIL risk score.

•	Età > 65 anni	Punti 1

• Storia di cardiopatia Punti 1

• ECG anormale Punti 1

• Assenza di prodromi Punti 1

OESIL risk score ≥ 2: alto rischio.

Per concludere, sistemi di stratificazione del rischio che sembrano essere i più adatti a questo scopo sono l'OESIL *risk score* (Tabella 8) e l'EGSYS *risk score* (Tabella 9).

TAB. 9

EGSYS risk score.

• Prodromi (nausea e vomito)

Malattia strutturale cardiaca	Punti	4
• ECG anormale	Punti	4
• Palpitazioni	Punti	3
• Sincope da supino	Punti	2

• Fattori predisponenti* e/o precipitanti** Punti -1

Punti -1

EGSYS *risk score* ≥ 3: alto rischio di sincope cardiaca.

EGSYS risk score < 3: basso rischio di sincope cardiaca.

^{*} Fattori predisponenti sono: luoghi caldi e affollati, prolungato ortostatismo, periodo post-prandiale.

^{**} Fattori precipitanti sono: paura, dolore intenso, movimento del collo.

Figure 3. Additional Evaluation and Diagnosis for Syncope Syncope additional evaluation and diagnosis Initial evaluation: history, physical exam, ECG (Class I) Initial evaluation Initial evaluation Stress testing clear unclear (Class IIa)† TTE Initial Initial Initial (Class IIa)† No additional Targeted blood evaluation evaluation evaluation evaluation testing -Options suggests suggests reflex suggests CV (Class IIa)† needed* **EPS** neurogenic OH, abnormalities syncope (Class IIa)† Referral for MRI or CT Cardiac monitor Tilt-table (Class IIb)† autonomic selected based testing evaluation on frequency (Class IIa)† (Class IIa)† and nature American (Class I) Heart Association_® Options Ambulatory Implantable external cardiac cardiac monitor monitor (Class IIa)† Class IIa)†

Key messages

Diagnosis: initial syncope evaluation



material To all:

- history,
- physical examination (including standing BP),
- standard ECG.

n selected cases (when appropriate):

- echocardiogram (if SHD is suspected),
- in-hospital monitoring (if arrhythmia is suspected),
- tilt testing (if OH or NMS are suspected),
- carotid sinus massage (in pts >40 yrs),
- blood tests (pO₂ & gas analysis, haematocrit & blood cells count, troponin, d-dimer).

Blood testing

Although broad-panel testing is common in clinical practice at the point of triage, there are no data on the utility of this approach.

Complete blood count and electrolyte panel are frequently obtained during syncope evaluation. The diagnostic yield is low when these are used routinely;

Usefulness of brain natriuretic peptide and high-sensitivity troponin measurement is uncertain in patients for whom a cardiac cause of syncope is suspected (IIb)

Routine and comprehensive laboratory testing is not useful in the evaluation of patients with syncope (III)

Cardiac imaging

Transthoracic echocardiography can be useful in selected patients presenting with syncope if structural heart disease is suspected (lia)

Computed tomography (CT) or magnetic resonance imaging (MRI) may be useful in selected patients presenting with syncope of suspected cardiac etiology (IIb)

Routine cardiac imaging is not useful in the evaluation of patients with syncope unless cardiac etiology is suspected on the basis of an initial evaluation, including history, physical examination, or ECG (III)

MSC

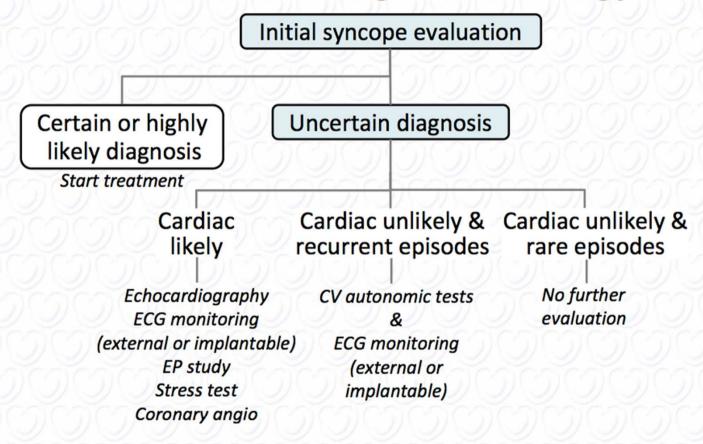
Carotid sinus massage

Recommendations	Class	Level
Indication		
1. CSM is indicated in patients >40 years of age with syncope of unknown origin compatible with a reflex mechanism.	1	В
Diagnostic criteria		
 CSS is confirmed if CSM causes bradycardia (asystole) and/or hypotension that reproduce spontaneous symptoms and patients have clinical features compatible with a reflex mechanism of syncope. 	ı	В

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The initial evaluation: diagnostic strategy





Exercise testing



Recommendations	Class	Level
Indications		
Exercise testing is indicated in patients who experience syncope during or shortly after exertion.	1	C
Diagnostic criteria		
 Syncope due to second- or third-degree AV block is confirmed when the AV block develops during exercise, even without syncope. 	J	C
 Reflex syncope is confirmed when syncope is reproduced immediately after exercise in the presence of severe hypotension. 	1	C

Recomme	Recommendation for Stress Testing		
COR	LOE	Recommendation	
IIa	C-LD	Exercise stress testing can be useful to establish the cause of syncope in selected patients who experience syncope or presyncope during exertion (132,143).	
	ine Data	Exertion can result in syncope in a variety of conditions, including structural lesions, such as hypertrophic obstructive cardiomyopathy and aortic stenosis; interarterial anomalous coronary artery and pulmonary arterial hypertension; and channelopathies, such as LQTS (type 1) and catecholaminergic polymorphic ventricular tachycardia (CPVT). Subjecting a patient to a treadmill exercise test to reproduce the symptoms or evaluate the hemodynamic response to exertion (e.g., hypotension) must be done with extreme caution and in an environment with proper advanced life support.	
Supplement 10.		In a prospective evaluation of 433 patients in which tachyarrhythmia was studied as the etiology for exertional syncope (132), an ECG stress evaluation was felt to be the sole test useful in identifying a presumptive cause of syncope in only 2 patients. However, bradyarrhythmia may ultimately be responsible for exertional syncope as well, and may only be elicited during stress testing. In rare instances, exercise-induced ischemia (143-146) or coronary vasospasm (147) may lead to high grade/infranodal AV block in patients with underlying coronary disease.	

Key messages

Diagnosis: subsequent investigations



- 9. Perform prolonged ECG monitoring (external or implantable) in patients with recurrent severe unexplained syncope who have all of the following three features:
 - Clinical or ECG features suggesting arrhythmic syncope.
 - A high probability of recurrence of syncope in a reasonable time.
 - Who may benefit from a specific therapy if a cause for syncope is found.

ECG monitoring: indications **ESC T-LOC** suspected syncope **European Society** of Cardiology Certain Uncertain diagnosis/mechanism diagnosis/mechanism Treat appropriately T-LOC Syncope non-syncopal High risk, Low risk, reflex Low risk & Low risk. Unconfirmed arrhythmia likely & need rare episodes arrhythmia likely epilepsy likely for specific & recurrent therapy Unexplained episodes falls Not **ILR** ILR ILR In-hospital indicated (Class IIa) (Class IIb) monitoring (Class I) (Class I) **ELR** If negative (Class IIa)

ILR (Class I) Holter

(Class IIa)

ECG monitoring: Indications (1)

Recommendations		Level
In-hospital monitoring		
1. Immediate in-hospital monitoring (in bed or by telemetry) is indicated in high-risk patients.	1	С
Holter monitoring		
 Holter monitoring should be considered in patients who have frequent syncope or presyncope (≥1 episode per week). 		В
External loop recorder		
3. Reflex syncope, OH, POTS, or PPS should be considered likely if tilt testing reproduces symptoms along with the characteristic circulatory pattern of these conditions.	lla	В

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ECG monitoring: Indications (2)

Recommendations	Class	Level
Implantable loop recorder		
4. ILR is indicated in an early phase of evaluation in patients with recurrent syncope of uncertain origin, absence of high-risk criteria (listed in <i>Table 6</i>), and a high likelihood of recurrence within the battery life of the device.	_	A
ILR should be considered in patients with suspected or certain reflex syncope presenting with frequent or severe syncopal episodes.	lla	В
6. ILR may be considered in patients in whom epilepsy was suspected but the treatment has proven ineffective.	lla	В
7. ILR may be considered in patients with unexplained falls.	IIb	В

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ECG monitoring: Diagnostic criteria

Recommendations	Class	Level
Arrhythmic syncope is confirmed when a correlation between syncope and an arrhythmia (bradyarrhythmia or tachyarrhythmia) is detected.	ı	В
2. In the absence of syncope, arrhythmic syncope should be considered likely when periods of Mobitz II second- or third-degree AV block or a ventricular pause >3 seconds (with possible exception of young trained persons, during sleep or rate-controlled atrial fibrillation), or rapid prolonged paroxysmal SVT or VT are detected.	lla	С

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Recomme	Recommendations for Cardiac Monitoring		
COR	LOE	Recommendation	
I	С-ЕО	The choice of a specific cardiac monitor should be determined on the basis of the frequency and nature of syncope events.	
		The technology of cardiac rhythm monitoring is dynamic and advancing at rapid	
		speed. Several types of ambulatory cardiac rhythm monitoring are summarized in	
N/	Α	Table 8. Their selection and usefulness are highly dependent on patient	
		characteristics with regard to the frequency of syncope and the likelihood of an	
		arrhythmic cause of syncope (148).	
		To evaluate selected ambulatory patients with syncope of suspected	
		arrhythmic etiology, the following external cardiac monitoring approaches	
	B-NR	can be useful:	
IIa		1. Holter monitor (149-153)	
		2. Transtelephonic monitor (150,154,155)	
		3. External loop recorder (150,154-156)	
		4. Patch recorder (157-159)	
		5. Mobile cardiac outpatient telemetry (160,161).	

Ша	II. D.D.	To evaluate selected ambulatory patients with syncope of suspected
IIa	B-R	arrhythmic etiology, an ICM can be useful (149,150,153,161-175).

Electrophysiological study: Indications



Recommendations	Class	Level
 In patients with syncope and previous myocardial infarction or other related conditions, EPS is indicated when syncope remains unexplained after non-invasive evaluation. 		
In patients with syncope and bifascicular BBB, EPS should be considered when syncope remains unexplained after non- invasive evaluation.	lla	В
3. In patients with syncope and asymptomatic sinus bradycardia, EPS may be considered in a few instances when non-invasive tests (e.g. ECG monitoring) have failed to show a correlation between syncope and bradycardia.	llb	В
4. In patients with syncope preceded by sudden and brief palpitations, EPS may be considered when syncope remains unexplained after non-invasive evaluation.	llb	С

17

EPS-guided therapy



European Society of Cardiology

Recommendations		
 In patients with unexplained syncope and bifascicular BBB, a pacemaker is indicated in the presence of either a baseline H-V interval of ≥70 ms, or second- or third-degree His-Purkinje block during incremental atrial pacing, or with pharmacological challenge. 		В
 In patients with unexplained syncope and previous myocardial infarction or other scar-related conditions, it is recommended to manage induction of sustained monomorphic VT according to the current ESC guidelines for VA. 	1)	В
3. In patients without structural heart disease with syncope preceded by sudden and brief palpitations, it is recommended to manage the induction of rapid SVT or VT, which reproduces hypotensive or spontaneous symptoms, with appropriate therapy according to the current ESC Guidelines.	1)	С
 In patients with syncope and asymptomatic sinus bradycardia, a pacemaker should be considered if a prolonged corrected SNRT is present. 	lla	В

Recommer	Recommendations for EPS		
COR	LOE	Recommendations	
IIa	B-NR	EPS can be useful for evaluation of selected patients with syncope of suspected arrhythmic etiology (91,151,199-205).	
		Diagnostic results detected during EPS occur predominantly in patients who have cardiac disease (e.g., conduction system delay, coronary artery disease,	
See Online Data Supplement 14.		cardiomyopathy, and valvular heart disease). Most of the literature evaluating EPS as a means to diagnose syncope is relatively old, and the data were obtained in referral centers where there was a high pretest probability of an arrhythmia.	
		Eight of these small retrospective studies (91,199-205) (total n=625) found that, of the 406 patients with cardiac disease or an abnormal ECG, 41% had a positive	
		result (of these, 21% had VT and 34% had a bradycardia) (151). Of 219 patients without evidence of heart disease, only 5% had a positive result (1% with VT	
		and 10% with evidence of substrate for symptomatic bradycardia). Overall, the diagnostic yield of EPS was approximately 50% and 10% in patients with and without structural heart disease, respectively.	
III: No Benefit	B-NR	EPS is not recommended for syncope evaluation in patients with a normal ECG and normal cardiac structure and function, unless an arrhythmic etiology is suspected (205-207).	
See Online Data Supplement 14.		One prospective evaluation of 247 patients with syncope of undetermined etiology who underwent EPS found that the diagnostic yield was significantly higher in patients with an abnormal ECG than in those with a normal ECG (22%	
		versus 3.7%) and that the diagnostic yield was low in patients with a normal	

Tilt table test

Ha

B-R

If the diagnosis is unclear after initial evaluation, tilt-table testing can be useful for patients with suspected VVS (208-213).

See Online Data Supplement 15. Tilt-table testing has been used to evaluate patients with syncope for nearly 3 decades (208). It is an orthostatic stress test to assess the susceptibility of a vasovagal response to a postural change from a supine to an upright position. A positive response is defined as inducible presyncope or syncope associated with hypotension, with or without bradycardia (less commonly asystole). The hemodynamic response to the tilt maneuver determines whether there is a cardioinhibitory, vasodepressor, or mixed response (214). There is general consensus that a tilt-table angle of 70 degrees for 30 to 40 minutes would provide optimal yield (211,213,215). Adjunctive agents, such as a low dose of isoproterenol infusion or sublingual nitrates, may improve sensitivity but decrease specificity (210,212,216,217). A positive tilt-table test suggests a tendency or predisposition to VVS induced in the laboratory. This observation during tilt-table testing cannot necessarily define a causal etiology or be entirely conclusive of a reflex mechanism for syncope in the clinical setting. Correlation of tilt-table-induced findings to patients' clinical presentation is critically important to prevent consequences of false-positive results from tilt-table testing.

The utility of tilt-table testing is highest in patients with a suspected VVS when syncope is recurrent. Several factors have reduced the role of tilt-table testing in the evaluation of syncope: the overall moderate sensitivity, specificity, and reproducibility of tilt-table testing; the presence of false-positive response in controls; the increasing recognition of VVS from a structured history taking; and the availability of long-term cardiac monitoring (24,211,213).

Tilt table test

IIa	B-NR	Tilt-table testing can be useful for patients with syncope and suspected delayed OH when initial evaluation is not diagnostic (218,219).
	ine Data ment 15.	OH with standing, or a similar fall in blood pressure within 3 minutes of upright tilt-table testing to 60 degrees (220), is distinct from delayed OH, characterized by a sustained decrease in blood pressure occurring beyond 3 minutes of standing or upright tilt-table testing (220,221). Delayed OH may be responsible for syncopal episodes or symptoms of orthostatic intolerance only after prolonged standing. In 1 retrospective study of 230 patients with OH, only 46% had OH within 3 minutes of head-up tilt; 15% had OH between 3 and 10
		minutes; and 39% had OH only after 10 minutes of tilt-table testing (218). In 10-year follow-up data from 165 of these patients, 54% of individuals with delayed OH progressed to classic OH (219). The 10-year death rate in individuals with delayed OH was 29%, compared with 64% and 9% in individuals with baseline OH and controls, respectively.

Tilt table test

Tilt-table testing is reasonable to distinguish convulsive syncope from Ha B-NR epilepsy in selected patients (222-225). Convulsive syncope is a term that can be used to describe any form of syncope manifesting with convulsive movements (e.g., myoclonus). Prolonged convulsions and marked postictal confusion are uncommon in patients with syncope associated with convulsive movements (226), and fatigue is frequent after reflex syncope and may be confused with a postictal state (226). Tilt-table testing has been shown to be of value in this clinical setting when a detailed history cannot clearly determine whether the convulsive movements were secondary to syncope, given the need for objective evidence to help distinguish this entity from true epileptic seizures. In a prospective study of 15 patients with See Online Data recurrent unexplained seizure-like episodes who were unresponsive to Supplement 15. antiepileptic therapy (223), 67% had convulsive movements associated with rican hypotension and bradycardia during tilt-table testing. In another study of 74 patients with a questionable diagnosis of epilepsy (because of drug-refractory seizures or clinically suspected not to be true epilepsy), a cardiac diagnosis was established in 42% of patients, with >25% developing profound hypotension or bradycardia during the head-up tilt-table test, confirming the diagnosis of VVS (225). Taken together, it can be estimated from these studies that approximately 50% of patients with either questionable or drug-refractory epilepsy have positive tilt-table tests suggestive of a vasovagal etiology (226).

Tilt table test

		Tilt-table testing is reasonable to establish a diagnosis of pseudosyncope		
IIa	B-NR	(227-229).		
		Psychogenic pseudosyncope should be suspected when patients present with		
		frequent (even daily) symptoms that mimic VVS (and, in some cases, with a		
		history of true VVS). It is often challenging to differentiate psychogenic syncope		
		from true syncope. However, tilt-table testing may help to elucidate the		
		diagnosis. During tilt-table testing, the apparent unconsciousness with loss of		
		motor control, combined with normal blood pressure and heart rate (and a normal		
		electroencephalogram [EEG] if such a recording is obtained), rules out true		
See Onli	nline Data syncope and most forms of epilepsy (227-229). In 1 study of 800 patients who			
Supplen	nent 15.	underwent tilt-table testing, approximately 5% were diagnosed with		
		pseudosyncope. Compared with patients with VVS, eye closure during the event,		
		long periods of apparent transient loss of consciousness, and increased heart rate		
		and blood pressure are highly specific for pseudosyncope. One study of 21		
		patients with suspected pseudosyncope who were subjected to tilt-table testing		
		with continuous monitoring of the ECG, EEG, and blood pressure revealed 17		
		patients with non-epileptiform limb shaking without significant changes on an		
	EEG or hemodynamic changes (227).			
III: No	B-R	Tilt-table testing is not recommended to predict a response to medical		
Benefit	D IX	treatments for VVS (230,231).		

Tilt testing

Recommendations	Class	Level
Indications		
Tilt testing should be considered in patients with suspected reflex syncope, OH, POTS, or PPS.	lla	В
Tilt testing may be considered to educate patients to recognize symptoms and learn physical manoeuvres.	llb	В
Diagnostic criteria		
Reflex syncope, OH, POTS, or PPS should be considered likely if tilt testing reproduces symptoms along with the characteristic circulatory pattern of these conditions.	lla	В

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2018 ESC Guidelines on Syncope – Michele brignole & Angel Moya EHJ Doi:10.1093/eurheartj/ehy037

Key messagesTreatment



To all patients with reflex syncope and OH:

- Explain the diagnosis
- Reassure
- Explain the risk of recurrence
- ESC information sheet for patients
- Give advice on how to avoid triggers and situations
- Advice to drink at least 2 litters of fluid a day
- How to perform counterpressure manoeuvres

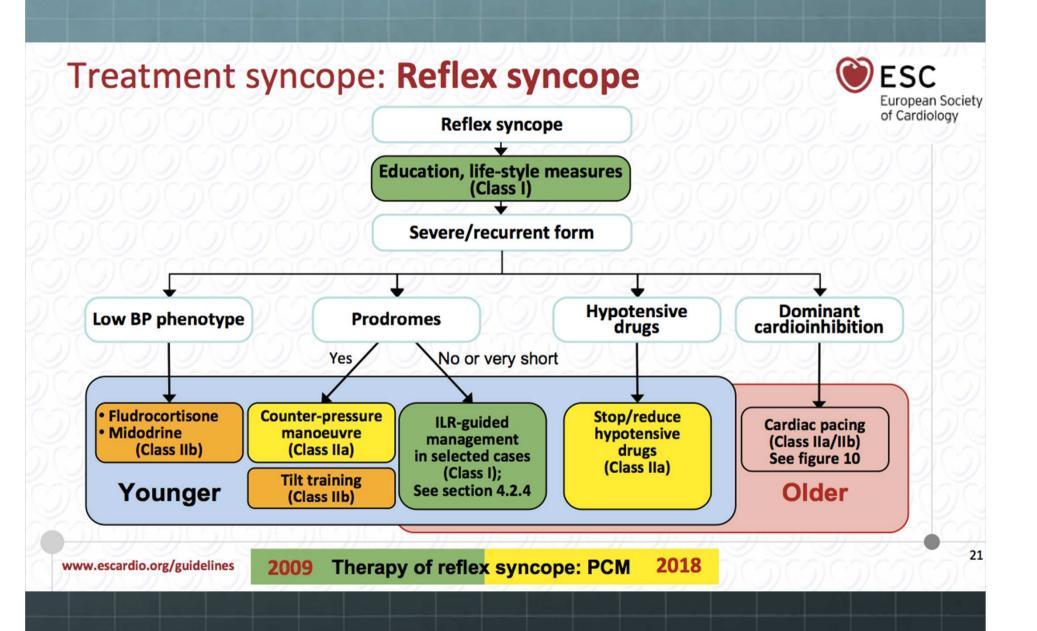
These measures are the cornerstone of treatment and have a high impact in reducing the recurrence of syncope.

Key messages Treatment



Patients with severe forms of reflex syncope (very frequent syncope that alters quality of life, exposes the patient to a risk of trauma, or when syncope occurs during a high-risk activity)

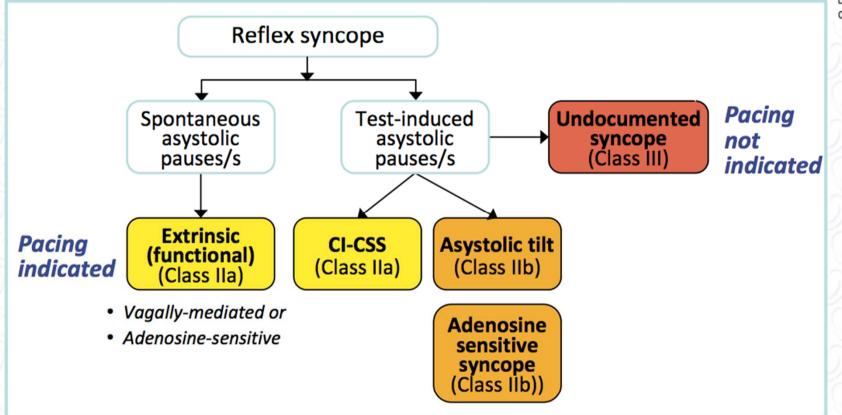
→ select one or more additional specific treatments according to the clinical features and age:



Pacing for reflex syncope



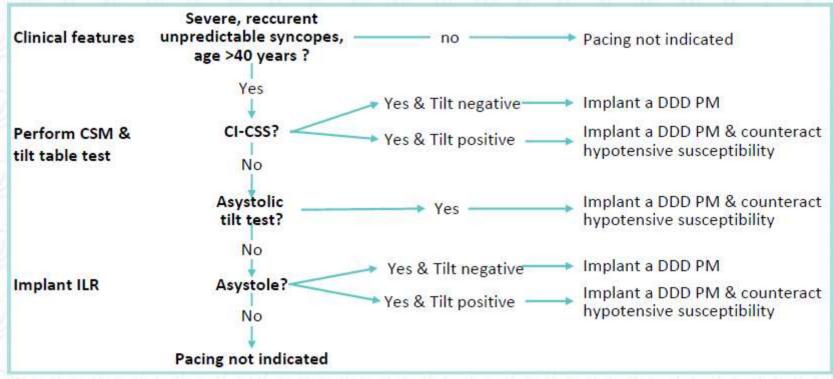
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Pacing for reflex syncope: decision pathway





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Mechanism of Syncope in Patients With Positive Adenosine Triphosphate Tests

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OBJECTIVES We prospectively evaluated the mechanism of syncope in patients with positive adenosine

triphosphate (ATP) tests (defined as the induction of atrioventricular [AV] block with a

ventricular pause ≥6 s after an intravenous bolus of 20 mg ATP).

BACKGROUND Patients with unexplained syncope tend to have more positive ATP tests results than those

without syncope.

An implantable loop recorder (ILR) was inserted in 36 ATP-positive patients (69 ± 10 years; METHODS

22 women; median of 6 syncopal episodes); 15 of them also had a positive response to tilt

testing.

RESULTS During the follow-up of 18 ± 9 months, 18 patients (50%) had syncopal recurrence and 16

(44%) had an electrocardiographically documented episode: AV block (n = 3: paroxysmal in 2 and permanent in 1), AV block followed by sinus arrest (n = 1), sinus arrest (n = 5), sinus bradycardia <40 beats/min (n = 2), normal sinus rhythm (n = 2), sinus tachycardia (n = 1), rapid atrial fibrillation (n = 1), and ectopic atrial tachycardia (n = 1). Bradycardia was documented in a total of 11 cases (69%), and a long ventricular pause (4 to 29 s) was present in eight cases (50%). All three patients with ILR-documented AV block had previously had a negative tilt test, whereas seven of eight with ILR-documented sinus bradycardia or sinus

arrest had previously had a positive tilt test.

CONCLUSIONS In patients with adenosine-sensitive syncope, the mechanism of syncope is heterogeneous,

although bradycardia is the most frequent finding. Adenosine triphosphate-induced AV block predicts AV block as the mechanism of spontaneous syncope in only a few tilt-negative patients. (J Am Coll Cardiol 2003;41:93-8) © 2003 by the American College of

Cardiology Foundation

Efficacy of theophylline in patients affected by low adenosine syncope @



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Introduction

Patients with unexplained syncope of sudden onset, normal heart, and normal electrocardiogram (ECG; so-called unexplained syncope, no prodrome, and normal heart) have been shown to be different from patients with vasovagal syncope.12 Rather, their clinical and biological features are close to those observed in patients affected by idiopathic paroxysmal atrioventricular (AV) block.3 Patients with syncope without prodrome and normal heart and patients with idiopathic paroxysmal AV block have an adenosine profile that is opposite to that observed in patients with vasovagal syncope and is characterized by low plasma adenosine values, low expression of A2A adenosine receptors, and a high induction rate of transient complete heart block during exogenous injections of adenosine.4 Unlike in patients with vasovagal syncope, tilt testing is usually negative.4 Adenosine is suspected to be involved in the mechanism of syncope in such patients. These forms of syncope have been labeled low adenosine syncope,1 and this terminology is used throughout this article.

Since patients with low plasma adenosine levels are highly susceptible to exogenous and endogenous adenosine, 2-6 we wanted to investigate whether treatment with theophylline, a nonselective adenosine receptor antagonist, should result in the prevention of syncopal recurrences. We found the opportunity to test this hypothesis in a highly selected subset of patients who will be described in this article.

major clinical features: (1) a long-standing history (median 8 years; range 3–30 years) of recurrent unexplained syncope without prodrome, normal heart, and normal ECG and (2) baseline values of plasma adenosine (0.11 ± 0.03 μmol/L) well below the 5th percentile of the value of normal subjects (0.40 μmol/L). Multiple episodes of paroxysmal AV block and sinus arrest were documented at the time of (pre) syncope in 4 and 1 patients, respectively; in another patient, documentation of the mechanism was lacking, as no ECG monitoring was active at the time of symptoms. Patient characteristics are listed in Table 1.

We had the opportunity to perform an intrapatient comparison between a period with and a period without theophylline therapy with the support of prolonged ECG monitoring in the majority of them (Table 2). The follow-up of all patients was updated to July 2015.

Specifically,

• patient 1 was seen in 1995 and followed up for 20 years. In 1995, Holter monitoring fortuitously recorded idiopathic paroxysmal AV block, which was reproduced on the adenosine triphosphate test. For the next 10 years, she underwent theophylline therapy, during which time she had no syncopal recurrences. In 2005, soon after discontinuing theophylline, she had a syncopal recurrence, for which she received an implantable loop recorder (ILR); over the next 3 years, 2 further episodes of syncope due to idiopathic paroxysmal AV block with long asystolic pauses up to 22 seconds were documented. Overall, she had 4

Table 1 Characteristics of the 6 patients with low adenosine syncope who were treated with theophylline

Patient no., sex, age	documentation of the index event (total duration/longest pause)	Adenosine plasma level (µmol/L) (normal range 0.40-0.78 µmol/L)	A2A adenosine receptor expression (AU) (normal range 0.40-0.80 AU)	Adenosine intravenous test, maximum pause	Tilt table testing	Carotid sinus massage	Electro physiology study
1, F, 50 y	Idiopathic AVB (34/22 s)	0.12	0.20	11 s	Negative	Negative	Negative
2, M, 72 y	Idiopathic AVB (11/7 s)	0.09	0.50	Negative	Negative	Negative	Negative
3, F, 20 y	None (ILR only with theophylline)	0.09	0.55	5.4 s	Negative	пр	Negative
4, F, 71 y	Idiopathic AVB (24/7 s)	0.10	Np	7 s	Positive mixed	Negative	пр
5, M, 41 y	Sinus arrest (18/ 6 s)	0.18	0.80	7.4 s	Positive cardioinhibitory	Negative	np
6, F, 52 y	Paroxysmal AVB (-/9 s)	0.10	0.45	7.6 s	Negative	Negative	Negative

Adenosine plasma level was evaluated using high-performance liquid chromatography, as described previously. Adenosine A₂ receptor expression was evaluated with the Western blot, as described previously. The normal ranges are between 5th and 95th percentiles of the values recorded in healthy control subjects.

AU = arbitrary units; AVB = atrioventricular block; ECG = electrocardiographic; ILR = implantable loop recorder; F = female; M = male; np = not performed.

*Documentation with prolonged monitoring using an ILR in patients 1, 2, 4, and 5 and Holter monitoring in patient 6.

Table 2 Comparative effect of outcomes in 5 patients who responded to the ophylline and the 1 patient who did not

	History of syncope before diagnosis		Observation without therapy			Observation during the ophylline therapy		
Patient no.	Duration (y)	No. of episodes	Months	Episodes of syncope	Episodes of asystole ≥ 3 s	Months	Episodes of syncope	Episodes of asystole ≥ 3 s
1	30	20	36	4	3	120	0	Np
2	12	2	11	0	19	14	0	4
3	18	40	24	8	np	24	0	0
4	1	2	24 13	0	27	6	0	0
5	3	5	2	1	1	20	0	0
Median (IQR)	12 (3-18)	5 (2-20)	13 (11-24)	0.11 (0-0.33)	1.11 (0.4-1.8)	20 (14-24)	0 (0-0)	0 (0-0.7)
6	2.5	Frequent presyncopes	6	Frequent presyncopes	14 per day (maximum pause 9.2 s)	1.5	Frequent presyncopes	7 per day (maximum pause 6.7 s)

Wilcoxon matched-pairs signed rank test: too few cases to use this test.

IQR = interquartile range; np = not performed.

Conclusion

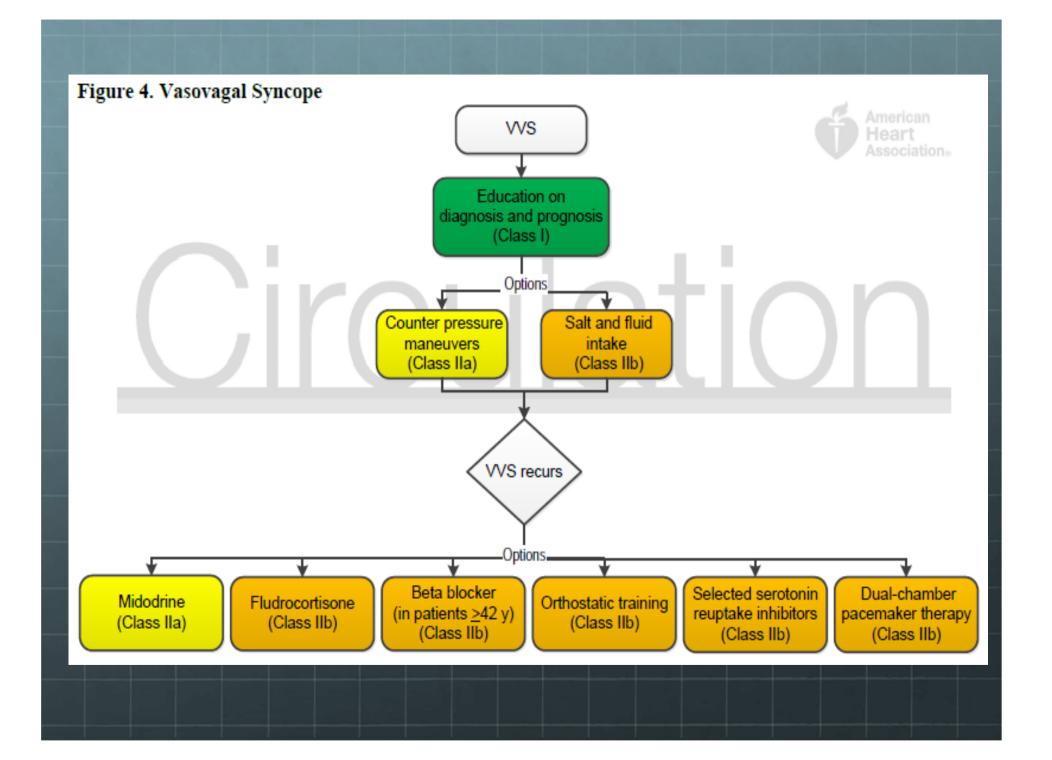
Theophylline may prevent syncopal recurrences and asystolic events in patients with low adenosine syncope and may be considered as an alternative to permanent pacing therapy in such patients. This study provides the rationale for

^{*}Observation with prolonged monitoring using an implantable loop recorder in patients 1-5 and Holter monitoring in patient 6.

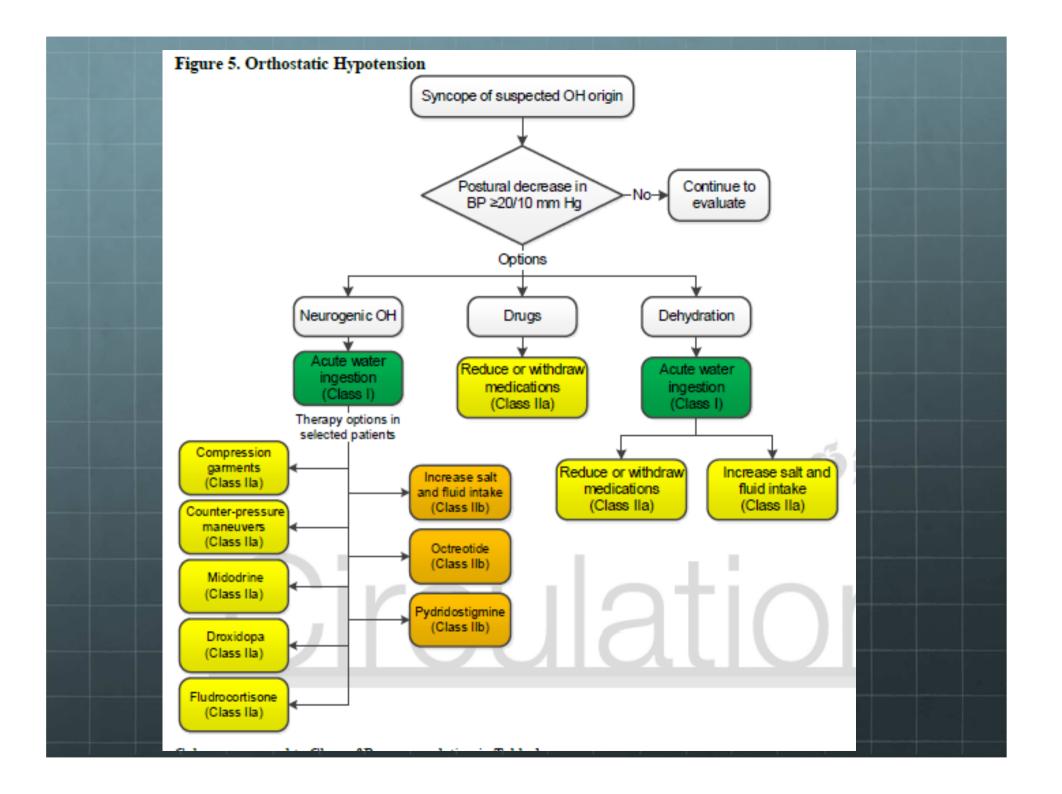
Vasovagal Syncope: Recommendations

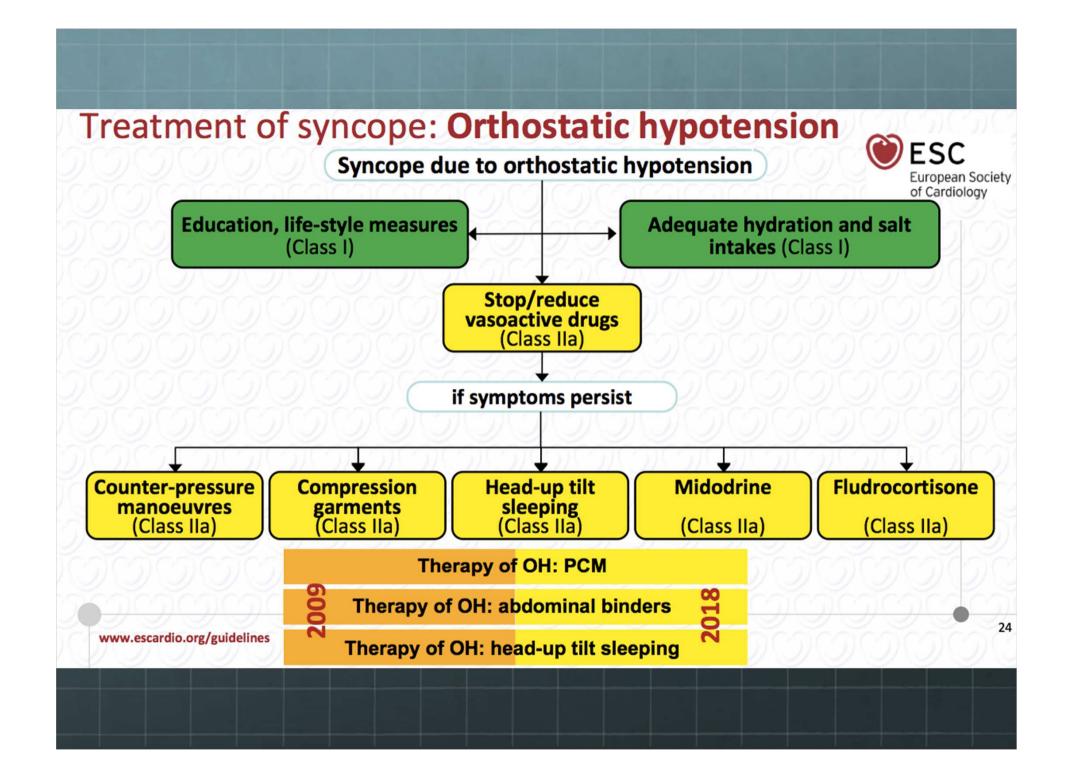
Recommen	Recommendations for VVS				
COR	LOE	Recommendations			
I	C-EO	Patient education on the diagnosis and prognosis of VVS is recommended.			
See Onli Supplemer 26	nts 25 and	In all patients with the common faint or VVS, an explanation of the diagnosis, education targeting awareness of and possible avoidance of triggers (e.g., prolonged standing, warm environments, coping with dental and medical settings), and reassurance about the benign nature of the condition should be provided.			
IIa	B-R	Physical counter-pressure maneuvers can be useful in patients with VVS who have a sufficiently long prodromal period (373-375).			

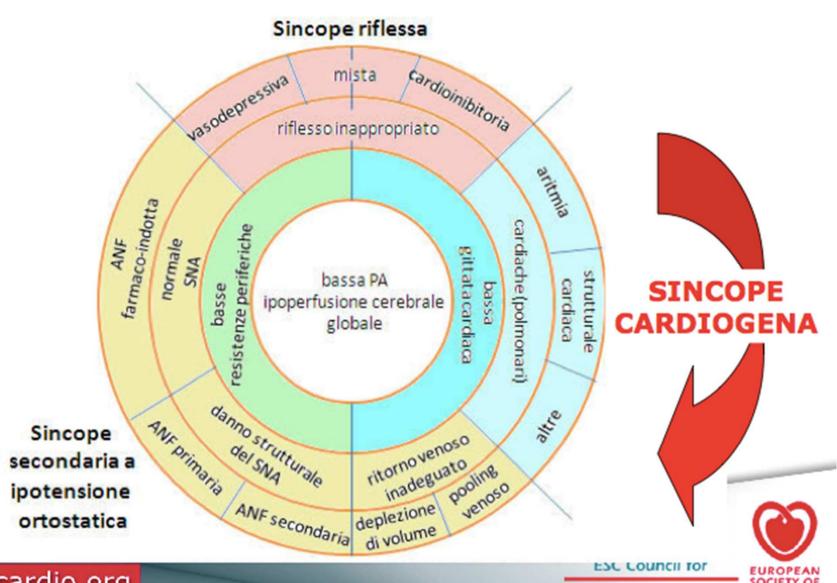
See Online Data Supplements 25 and 26. Patients with a syncope prodrome should be instructed to assume a supine position to prevent a faint and minimize possible injury. In patients with a sufficiently long prodrome, physical counter-maneuvers (e.g., leg crossing, limb and/or abdominal contraction, squatting) are a core management strategy. In a randomized, parallel, openlabel trial, leg crossing with conventional therapy (i.e., fluid, salt intake, counseling, and avoidance) was superior to conventional therapy in preventing syncope recurrence (375).



Recommen	ıdation fo	r Pacemakers in VVS
COR	LOE	Recommendation
IIb	B-R ^{SR}	Dual-chamber pacing might be reasonable in a select population of patients 40 years of age or older with recurrent VVS and prolonged spontaneous pauses (404-408,410).
See Onlin Suppleme and 2	ents 27	Among patients with a positive tilt-table test, a benefit of pacing for treatment of recurrent syncope was evident as compared with medical or no therapy in open-label trials (52,404,406,410-412), but this result must be interpreted with caution because of the possibility of outcome ascertainment bias. In 2 RCTs, there was no statistically significant benefit seen with active pacing (407,408). However, in a select population of patients >40 years of age with recurrent syncope and documented spontaneous pauses ≥3 seconds correlated with syncope or an asymptomatic pause ≥6 seconds, dual-chamber pacing reduced syncope recurrence. There was less benefit in patients with a positive tilt-table test that induced a vasodepressor response (405).
SR indicates s	ystematic r	eview.







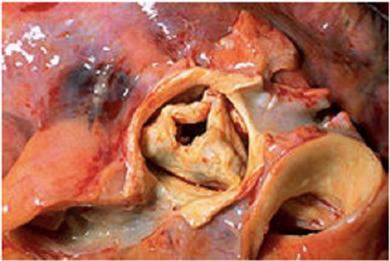
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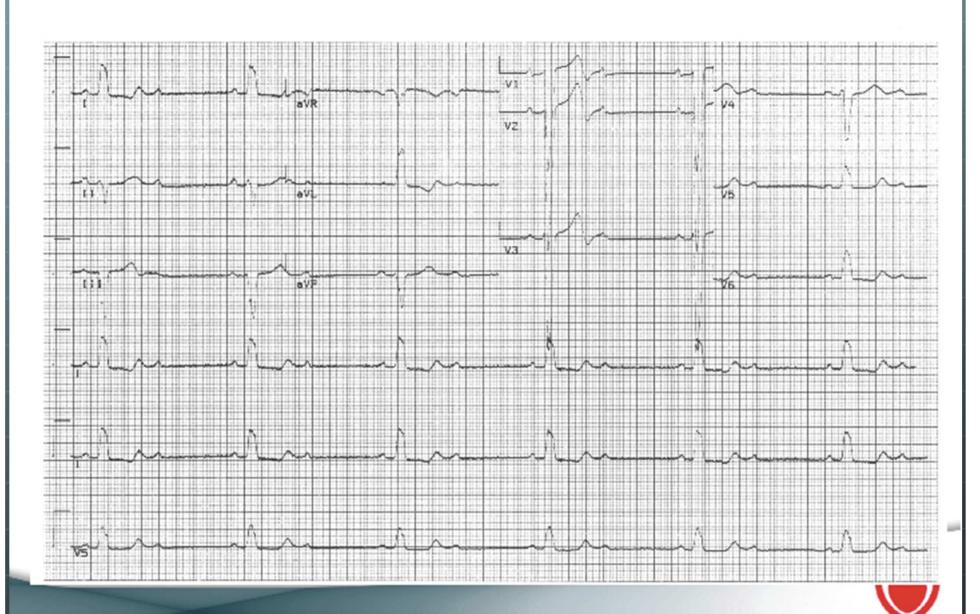


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CAUSA ARITMICA

- Bradicardia:
 - Malattia Nodo del Seno (Brady-Tachy)
 - Malattia del sistema di conduzione
 - Malfunzione di PM/AICD
- Tachicardia:
 - Sopraventricolare
 - Ventricolare (idiopatica, da cardiopatia strutturale, da canalopatia)
- Bradi o Tachicardia indotte da farmaci

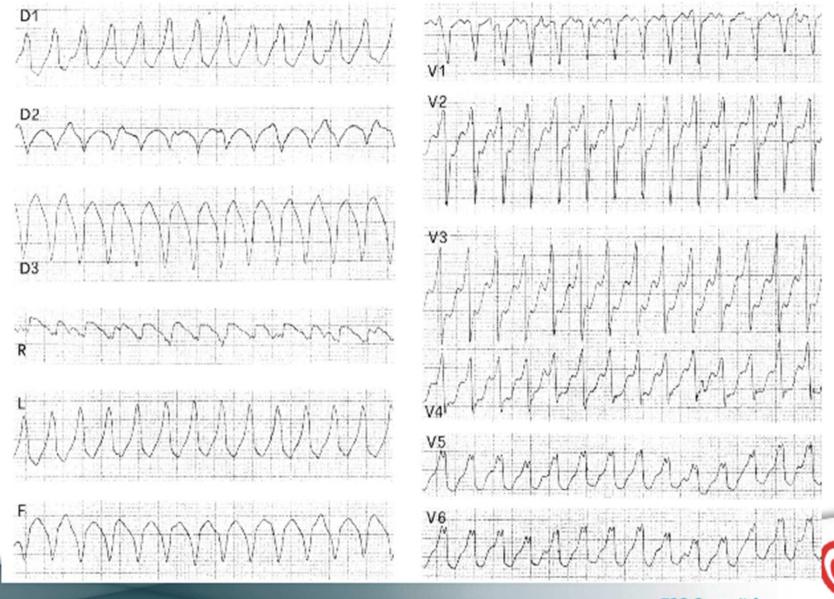




EUROPEAN

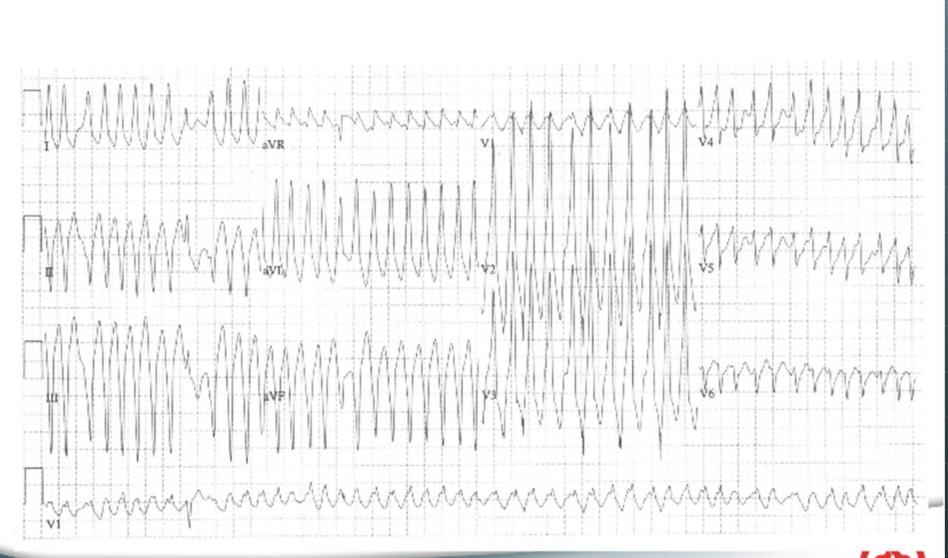
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MALATTIA STRUTTURALE

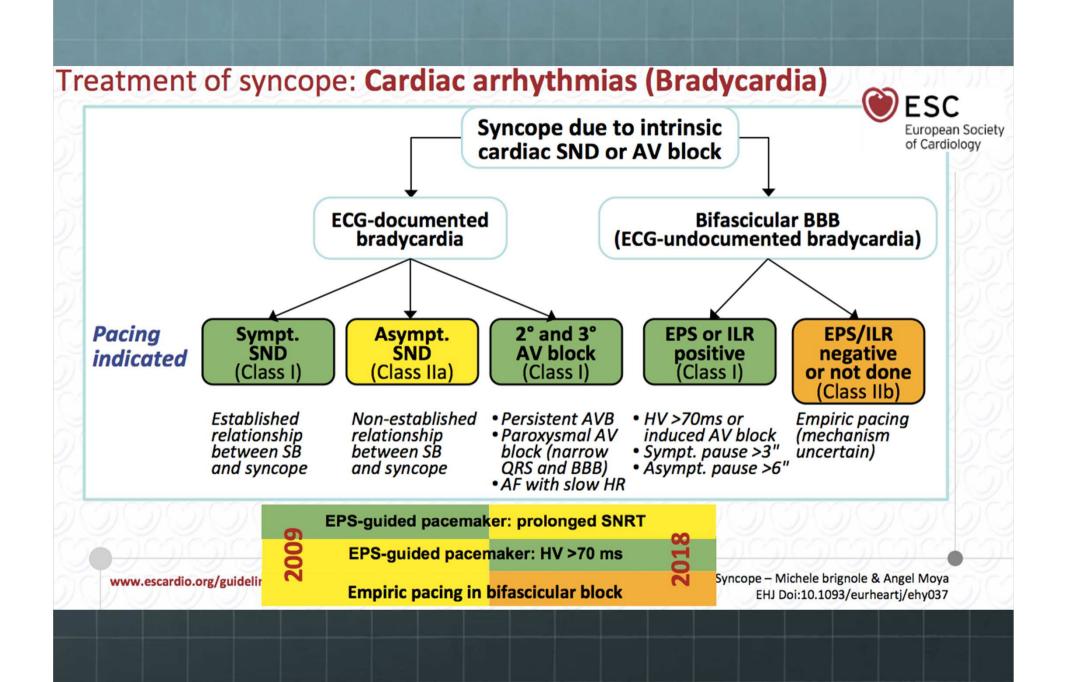
- Valvulopatie
- Cardiopatia ischemica
- Cardiomiopatia ipertrofica
- Masse intracardiache
- Tamponamento pericardico
- Cardiopatie congenite
- Anomalia congenita delle coronarie
- Disfunzione protesi valvolare



ALTRE

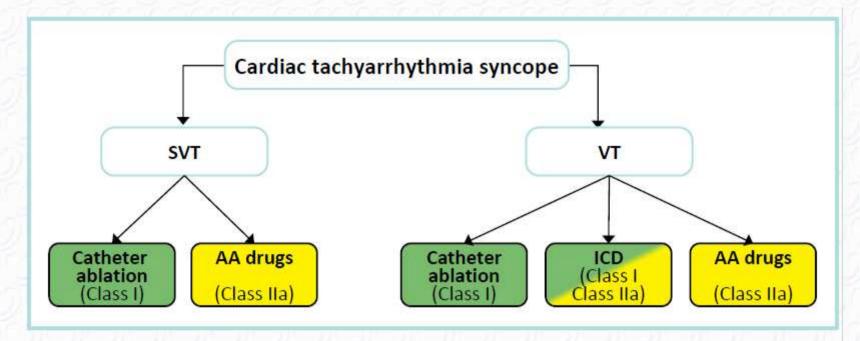
- Embolia polmonare
- Dissezione aortica
- Ipertensione polmonare





Treatment of syncope: Cardiac tachyarrhythmias





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2018 ESC Guidelines on Syncope – Michele brignole & Angel Moya EHJ Doi:10.1093/eurheartj/ehy037 Treatment of syncope: Cardiac arrhythmias

Recommendations	Class	Level
Tachycardia		
 Catheter ablation is indicated in patients with syncope due to SVT or VT in order to prevent syncope recurrence. 	ı	В
 An ICD is indicated in patients with syncope due to VT and ejection fraction ≤35%. 	ı	A
An ICD is indicated in patients with syncope and previous myocardial infarction who have VT induced during EPS.	ī	С
4. An ICD should be considered in patients with ejection fraction >35% with recurrent syncope due to VT when catheter ablation and pharmacological therapy have failed or could not be performed.	lla	С
Antiarrhythmic drug therapy, including rate-control drugs, should be considered in patients with syncope due to SVT or VT.	lla	С

Syncope & SVT/VT: AA drugs

Key messagesTreatment



In patiens with unexplained syncope (defined as syncope that does not meet any class I diagnostic criterion definded in the 2018 guidelines) at risk of SCD (e.g. those affected by left ventricle systolic dysfunction, HCM, ARVC, or inheritable arrhythmogenic disorders), but without class I indicaion of ICD

Balance the benefits and harm of ICD implantation



Recommendations	Class	Level
Left ventricular systolic dysfunction		
 ICD therapy is recommended to reduce SCD in patients with symptomatic heart failure (NYHA class II–III) and LVEF ≤35% after ≥3 months of optimal medical therapy who are expected to survive for at least 1 year with good functional status 	1	A
An ICD should be considered in patients with unexplained syncope with systolic impairment but without a current indication for ICD to reduce the risk of sudden death	lla	С
 Instead of an ICD, an ILR may be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD 	IIb	С

ICD: LVEF >35% and syncope



Recommendations	Class	Level
Hypertrophic cardiomyopathy		
 It is recommended that the decision for ICD implantation in patients with unexplained syncope is made according to the ESC HCM Risk-SCD score http://www.doc2do.com/hcm/webHCM.html 	1	В
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD. 	lla	С
Arrhythmogenic right ventricular cardiomyopathy		
ICD implantation may be considered in patients with ARVC and a history of unexplained syncope.	IIb	С
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD. 	lla	С



Recommendations	Class	Level
Hypertrophic cardiomyopathy		
 It is recommended that the decision for ICD implantation in patients with unexplained syncope is made according to the ESC HCM Risk-SCD score http://www.doc2do.com/hcm/webHCM.html 	1	В
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD. 	lla	С
Arrhythmogenic right ventricular cardiomyopathy		
ICD implantation may be considered in patients with ARVC and a history of unexplained syncope.	IIb	С
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD. 	lla	С

 In patients with primary cardiomyopathy or inheritable arrhythmogenic disorders who are at low risk of sudden cardiac death, ILR should be considered as alternative to ICD

Moya

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Recommendations	Class	Level
Long QT syndrome		
 ICD implantation in addition to beta-blockers should be considered in LQTS patients who experience unexplained syncope while receiving an adequate dose of beta-blockers. 	lla	В
 Left cardiac sympathetic denervation should be considered in patients with symptomatic LQTS when: (a) beta-blockers are not effective, not tolerated, or are contraindicated; (b) ICD therapy is contraindicated or refused; or (c) when patients on beta-blockers with an ICD experience multiple shocks. 	lla	С
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncopea who are at low risk of SCD based on a multiparametric analysis that takes into account the other known risk factors for SCD 	lla	С



Recommendations	Class	Level
Long QT syndrome		
 ICD implantation in addition to beta-blockers should be considered in LQTS patients who experience unexplained syncope while receiving an adequate dose of beta-blockers. 	lla	В
 Left cardiac sympathetic denervation should be considered in patients with symptomatic LQTS when: (a) beta-blockers are not effective, not tolerated, or are contraindicated; (b) ICD therapy is contraindicated or refused; or (c) when patients on beta-blockers with an ICD experience multiple shocks. 	lla	С
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncopea who are at low risk of SCD based on a multiparametric analysis that takes into account the other known risk factors for SCD 	lla	С

 In patients with primary cardiomyopathy or inheritable arrhythmogenic disorders who are at low risk of sudden cardiac death, ILR should be considered as alternative to ICD

- 3

Moya



Recommendations	Class	Level	
Brugada syndrome			
 ICD implantation should be considered in patients with a spontaneous diagnostic type I ECG pattern and a history of unexplained syncope. 	lla	В	
 Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD. 	lla	C	

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Recommendations	Class	Level	
Brugada syndrome			
 ICD implantation should be considered in patients with a spontaneous diagnostic type I ECG pattern and a history of unexplained syncope. 	lla	В	
2. Instead of an ICD, an ILR should be considered in patients with recurrent episodes of unexplained syncope with systolic impairment but without a current indication for ICD.	lla	С	

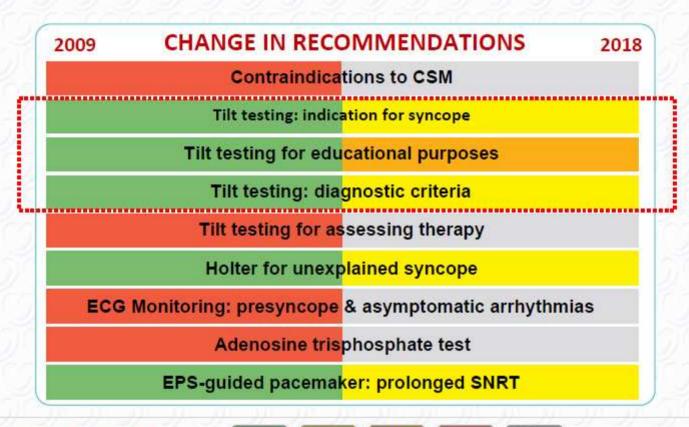
 In patients with primary cardiomyopathy or inheritable arrhythmogenic disorders who are at low risk of sudden cardiac death, ILR should be considered as alternative to ICD

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Moya

What is new in 2018 syncope guidelines? (1)





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lla



III

Taken out

What is new in 2018 syncope guidelines? (2)





EPS-guided pacemaker: HV >70 ms

Empiric pacing in bifascicular block

Therapy of reflex syncope: PCM

Therapy of OH: PCM

Therapy of OH: abdominal binders

Therapy of OH: head-up tilt sleeping

Syncope & SVT/VT: AA drugs
Expert opinion

CHANGE IN RECOMMENDATIONS 2009 2018

Syncope & AF: catheter ablation Expert opinion

ICD: LVEF >35% and syncope

Syncope & high risk HCM: ICD

Syncope & ARVC: ICD

Psychiatric consultation for PPS Expert opinion

1

lla

Ilb

III

Taken

Key messages Treatment



Re-evaluate the diagnostic process and consider alternative therapies if the above rules fail or are not applicable to an individual patient. Bear in mind that Guidelines are only advisory. Even though they are based on the best available scientific evidence, treatment should be tailored to an individual patient's need

RICORDATI: OGNI SINCOPE PUO' ESSERE



CARDIOLOGO

"CERCARE"
sincopi
in pazienti
a rischio

Aritmie osiaortica ardiomiopatia ipertrofica Canalopatie









Anamnesi

Paziente di 33 anni, non precdenti patologici significativi. Dall'età di 15 anni riferisce episodi sincopali con cadenza biennale, preceduti da prodromi, mai traumi. Il paziente riferisce, in realtà, la presenza dei prodromi anche in altre occasioni ma con regressione prima dell'evento sincopale.

Anamnesi

- Questi episodi sono occorsi in concomitanza con piccoli infortuni con conseguente impatto emotivo, durante la minzione, in corso di rilassamento fisico ma mai da sforzo.
- Il paziente stesso riferisce una importante componenete emotiva sulla quale ha lavorato nel corso degli anni ma che, naturalmente, non riesce ad eliminare completamente.

Anamnesi

- Il paziente svolge regolarmente attività fisica (calcetto) senza problemi.
- Nel 2008 già sottoposto a Tilt Test nel 2008, all'età di 23 anni, risultato non indicativo di sincope neuromediata.
- Doppler TSA naturalmente negativo per patologie a carico dei vasi esaminati.
- Molter-ecg del 2008 indicativo di ritmo sostanzialmente bradicardico in assenza di pause e/o aritmie significative

Tilt test

- Bradicardia di base (45-50 b.p.m.)
- MSC clino destro: NEGATIVO
- MSC clino sinistro: NEGATIVO
- Ipotensione ortostatica: NEGATIVA
- MSC orto destro: NEGATIVO
- MSC orto sinistro: Bradicardizzazione seguita da rapida perdita di coscienza con asistolia di 13". Ripresa spontanea dello stato di coscienza.
- Conclusioni: ipersensibilità del seno carotideo con sincope e pausa asistolica di 13".

ECG aVL aVL aVF



- **6** Che fare?
- Limitazioni lavorative ma paziente molto giovane!!!!
- **6** ILR
- In sala elettrofisiologia, dopo anestesia locale, pausa asistolica di 16" dopo anestesia locale

Impianto di PM bicamerale

