
La richiesta di competenza
neurologica nel prossimo futuro
Quinta edizione
Hotel Villa Pamphili, Roma
22-24 ottobre 2021
Sin
SOCIETÀ ITALIANA DI NEUROLOGIA
Sin
Sin
SEZIONE ITALIANA GIOVANI NEUROLOGI

Domenica 24 ottobre 2021

9.30 Sincope/Perdita di coscienza
P. Cortelli, *Bologna*

Pietro Cortelli

IRCCS Istituto delle Scienze Neurologiche di Bologna

DIBINEM, Alma Mater Studiorum - Università di Bologna

1. Disturbi transitori di coscienza

Coordinatore: Giuseppe Micieli
Gruppo di lavoro: Anna Cavallini, Pietro Cortelli, Umberto Aguglia, Serena Nannucci, Giovanna Calandra Bonaura

Introduzione

Il presente algoritmo decisionale è volto all'inquadramento, alla diagnosi e alla gestione terapeutica di pazienti che presentino una perdita di coscienza transitoria con recupero completo, cioè in assenza di qualsiasi residuo deficit neurologico, dovuta a qualsiasi causa, neurologica, cardiologica, traumatica o metabolica. L'inquadramento diagnostico prevede una tempestiva inchiesta anamnestica associata a un accurato esame obiettivo, corredati, anche in acuto, dal supporto di indagini strumentali, che consentano di escludere le situazioni a maggior rischio e di formulare un'iniziale diagnosi differenziale (ECG). Sono necessarie ulteriori indagini strumentali per indirizzare e confermare la diagnosi finale (EEG, Tilt table test, RM encefalo, colloquio psicodiagnostico). L'approccio terapeutico è orientato dalle molteplici diagnosi differenziali.

Definizione

Per **disturbi transitori di coscienza** s'intende un gruppo eterogeneo di condizioni che possono essere catalogate come caratterizzate da una perdita di coscienza, ad esordio improvviso, di breve durata e con completo recupero in assenza di qualsiasi residuo deficit neurologico.[1] Clinicamente ciò può comprendere la perdita

(N Engl J Med 2002;347:878-85.)

The New England Journal of Medicine

INCIDENCE AND PROGNOSIS OF SYNCOPE

ELPIDOFOROS S. SOTERIADES, M.D., JANE C. EVANS, D.Sc., MARTIN G. LARSON, Sc.D., MING HUI CHEN, M.D., LEWAY CHEN, M.D., EMELIA J. BENJAMIN, M.D., AND DANIEL LEVY, M.D.

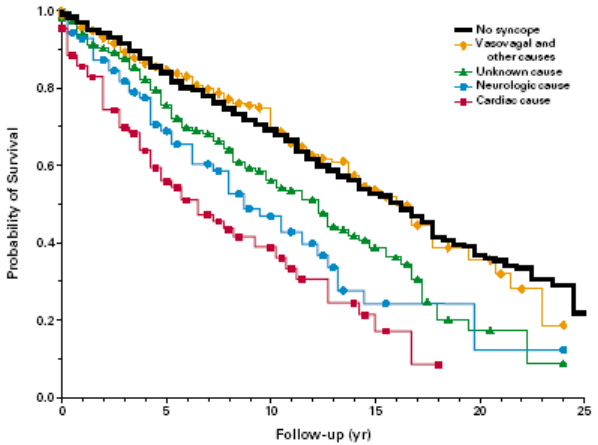


Figure 2. Overall Survival of Participants with Syncope, According to Cause, and Participants without Syncope.

P<0.001 for the comparison between participants with and those without syncope. The category "Vasovagal and other causes" includes vasovagal, orthostatic, medication-induced, and other, infrequent causes of syncope.

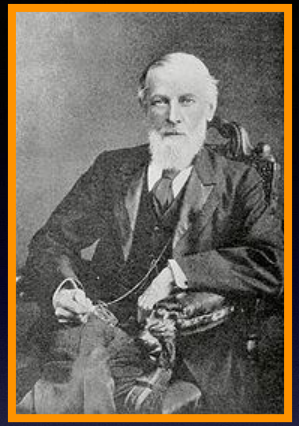
TABLE 1. CAUSES OF SYNCOPE ACCORDING TO SEX AND THE PRESENCE OR ABSENCE OF CARDIOVASCULAR DISEASE AT BASE LINE.

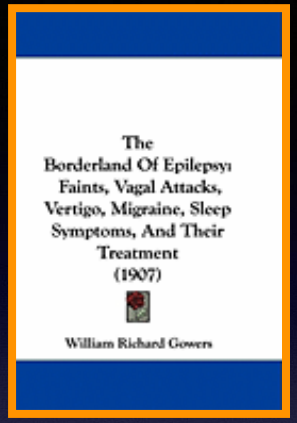
CAUSE	CARDIOVASCULAR DISEASE ABSENT (N=599)		CARDIOVASCULAR DISEASE PRESENT (N=223)		TOTAL SAMPLE (N=822)
	MEN (N=232)	WOMEN (N=367)	MEN (N=116)	WOMEN (N=107)	
	percent of subjects				
Cardiac	6.5	3.8	26.7	16.8	9.5
Unknown*	31.0	41.7	31.0	37.4	36.6
Stroke or transient ischemic attack	1.7	2.5	9.5	9.4	4.1
Seizure	7.3	3.3	6.9	2.8	4.9
Vasovagal	24.1	24.5	11.2	14.0	21.2
Orthostatic	9.5	10.9	6.9	6.5	9.4
Medication	7.3	6.5	4.3	9.4	6.8
Other†	13.0	6.8	3.5	3.7	7.5

*When a participant did not seek medical attention for syncope and the history, physical examination, and electrocardiographic findings were not consistent with any of the specific causes, the cause was considered to be unknown.

†Cough syncope, micturition syncope, and situational syncope were included in the category of other causes.

“E caddi come corpo morto cade”
Dante Alighieri. Inferno Canto V





Review

The borderland of epilepsy: clinical and molecular features of phenomena that mimic epileptic seizures

Douglas E Crompton, Samuel F Berkovic

Lancet Neurol 2009; 8: 370–81

PROBLEMI NELLA DIAGNOSI DIFFERENZIALE DELLA PERDITA DI COSCIENZA TRANSITORIA

- Non tutte le perdite di coscienza transitorie sono sincopi
- La perdita di coscienza può essere apparente e non reale
- Le condizioni cliniche che possono determinare sincope sono molteplici
- La sincope è un tipo di PdCT
- La sincope può essere un sintomo prognosticamente infausto anche a breve
- Non esiste un gold standard diagnostico

Difficoltà per il medico

- Fare diagnosi
  - È difficile vedere il paziente durante la crisi spontanea, il flusso cerebrale non è misurabile direttamente, il quadro clinico è simile a quello di altre PdC
- Definirne l'eziologia
  - Le cause sono molteplici, non esiste un gold standard diagnostico
- Stratificarne il rischio
  - La prognosi può essere benigna o maligna a breve o a lungo termine a seconda dell'eziologia

2018 ESC Guidelines for the diagnosis and management of syncope

The Task Force for the diagnosis and management of syncope of the European Society of Cardiology (ESC)

Developed with the special contribution of the European Heart Rhythm Association (EHRA)

Endorsed by: European Academy of Neurology (EAN), European Federation of Autonomic Societies (EFAS), European Federation of Internal Medicine (EFIM), European Union Geriatric Medicine Society (EUGMS), European Society of Emergency Medicine (EuSEM)

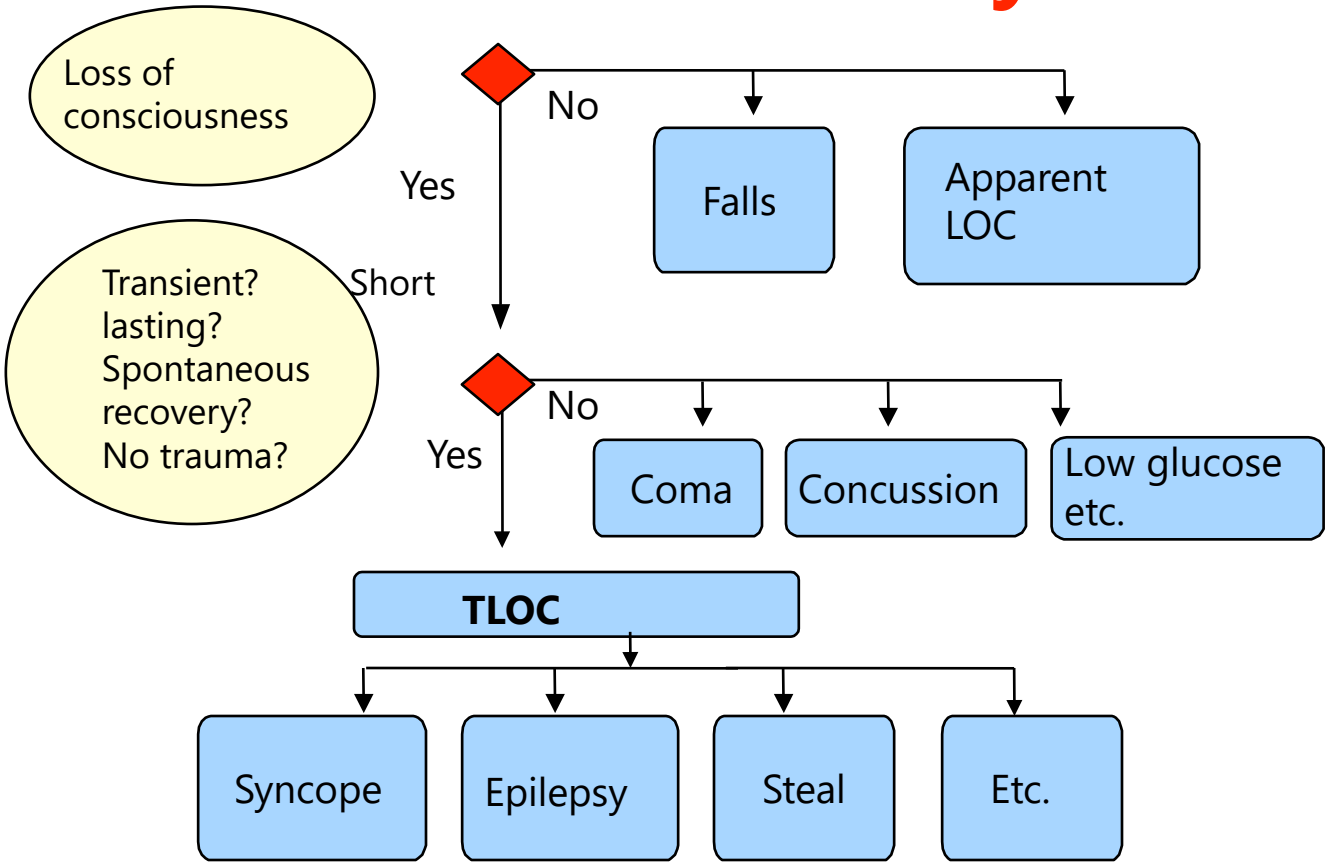
Authors/Task Force Members: Michele Brignole* (Chairperson) (Italy), Angel Moya* (Co-chairperson) (Spain), Frederik J. de Lange (The Netherlands), Jean-Claude Deharo (France), Perry M. Elliott (UK), Alessandra Fanciulli (Austria), Artur Fedorowski (Sweden), Raffaello Furlan (Italy), Rose Anne Kenny (Ireland), Alfonso Martín (Spain), Vincent Probst (France), Matthew J. Reed (UK), Ciara P. Rice (Ireland), Richard Sutton (Monaco), Andrea Ungar (Italy), and J. Gert van Dijk (The Netherlands)

Definitions

- Syncope is defined as TLOC due to cerebral hypoperfusion, characterized by a rapid onset, short duration, and spontaneous complete recovery

- TLOC is defined as a state of real or apparent LOC with loss of awareness, characterized by amnesia for the period of unconsciousness, abnormal motor control, loss of responsiveness, and a short duration.

A Hierarchy


Loss of consciousness
No
Falls
Apparent LOC
Yes
Transient? lasting? Spontaneous recovery? No trauma?
Short
No
Yes
Coma
Concussion
Low glucose etc.
TLOC
Syncope
Epilepsy
Steal
Etc.

TLoC: diagnostic work-up

- We need to be accurate about what we know and do not know
- Step-wise approach:
  - 1. Is it TLOC?
  - 2. Is it syncope?
  - 3. What type of syncope?

Anamnesi

- Generale
- Contesto della PdCT
- Prodromi
- Fase critica
- Fase Post-Critica

European Heart Journal Advance Access published December 16, 2014

European Heart Journal
doi:10.1093/eurheartj/ehu478

CURRENT OPINION

History taking as a diagnostic test in patients with syncope: developing expertise in syncope

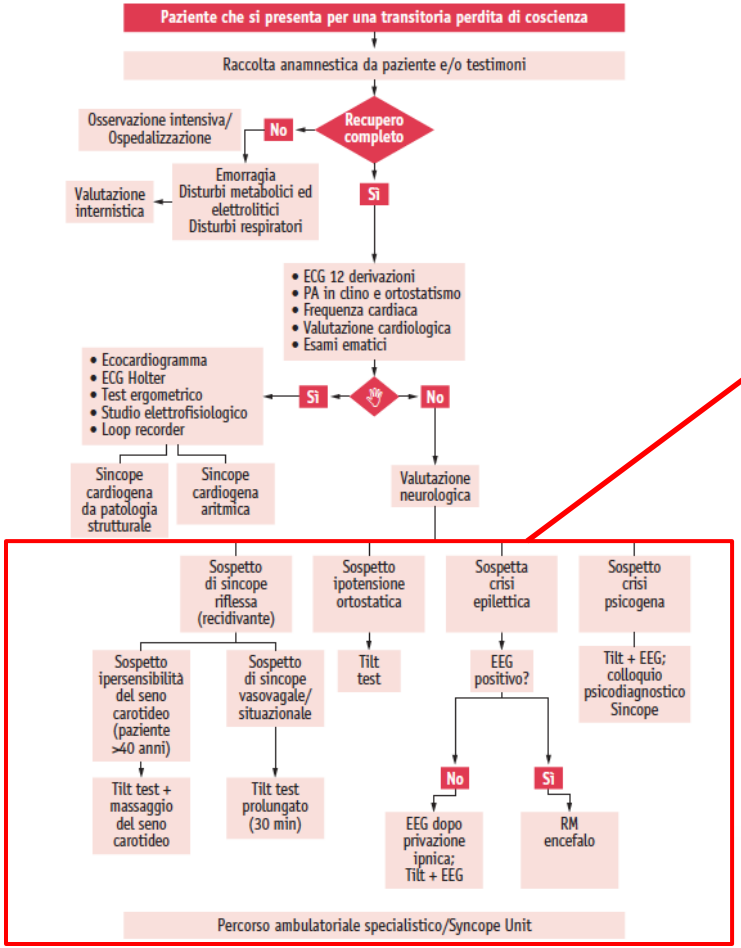
Wouter Wieling[1]*, Nynke van Dijk[2], Frederik J. de Lange[3], Louise R.A. Olde Nordkamp[3], Roland D. Thijs[4], J. Gert van Dijk[5], Mark Linzer[6,7], and Richard Sutton[8]

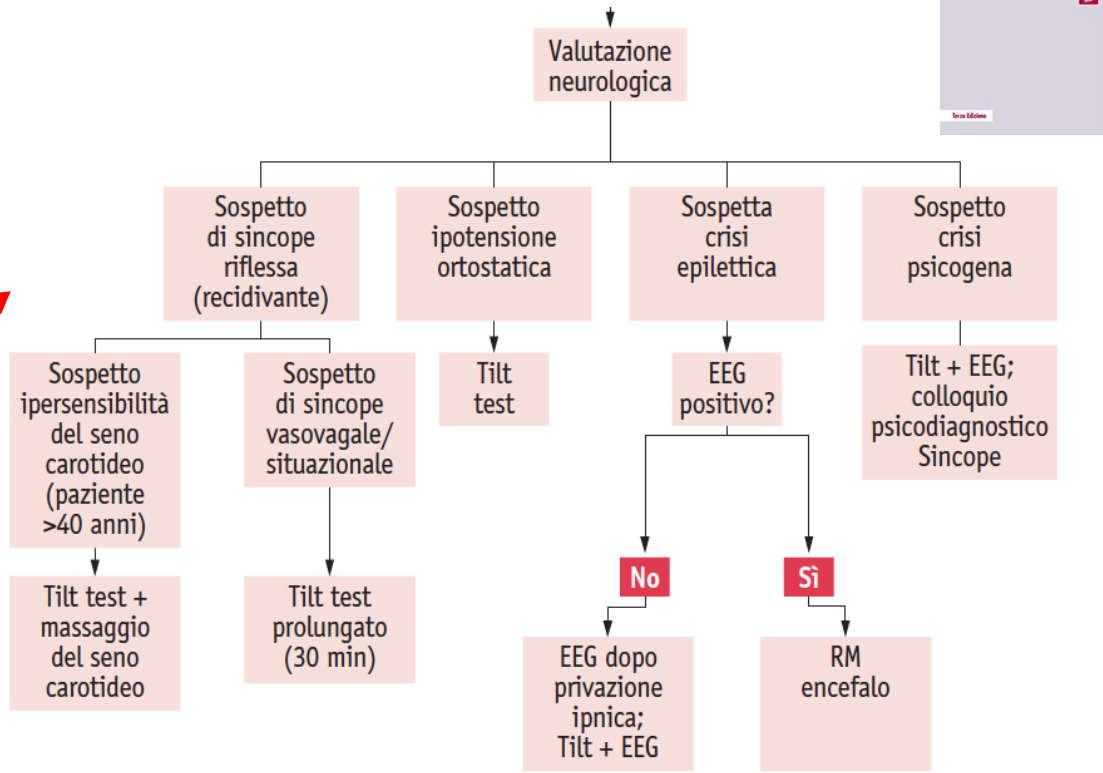
[1]Department of Internal Medicine, Medical Centre, University of Amsterdam, Amsterdam, The Netherlands; [2]Department of Family Medicine, Medical Centre, University of Amsterdam, Amsterdam, The Netherlands; [3]Department of Cardiology, Academic, Medical Centre, University of Amsterdam, Amsterdam, The Netherlands; [4]SEIN – Stichting Epilepsie Instellingen Nederland, Heemstede, The Netherlands; [5]Department of Neurology, Leiden University Medical Centre, Leiden, The Netherlands; [6]Department of Medicine, University of Minnesota, Minneapolis, MN 55455, USA; [7]Division of General Internal Medicine, Hennepin County Medical Center, Minneapolis, MN, USA; and [8]National Heart & Lung Institute, Imperial College, London, UK

Received 11 July 2014; revised 20 August 2014; accepted 3 November 2014

	sincope	epilessia	pseudo-sincopi
Fattori precip	++	-	+/-
Esordio	graduale	improvviso	improvviso
Durata	< 60 sec	2-3 min	1-45 min
Frequenza	<1/die	variabile	alta
Tono musc	flaccido>rigido	rigido	flaccido
Attività motoria	Myoclonic jerks breve durata dopo inizio episodio	Movimenti sincroni, simmetrici, prolungati	Eterogenea, disordinata
Occhi	aperti, transit	aperti, sosten	chiusi
Colore del viso	pallido	cianotico	normale
Incontinenza U	possibile	comune	assente
Incontinenza F	-	possibile	-
Morsus	raro (punta lingua)	frequente (lato)	assente
Confusione postcrit	<30 sec	prolungata	variabile
CPK	normale	elevato	normale

Figura 1.2 **Algoritmo diagnostico/gestionale dei pazienti con PTdC in PS**





Tilt Test Neurologico
- 10 min
- EEG
- senza farmaci

Clinical Neurophysiology 127 (2016) 1022–1030

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Review

Tilt table testing in neurology and clinical neurophysiology

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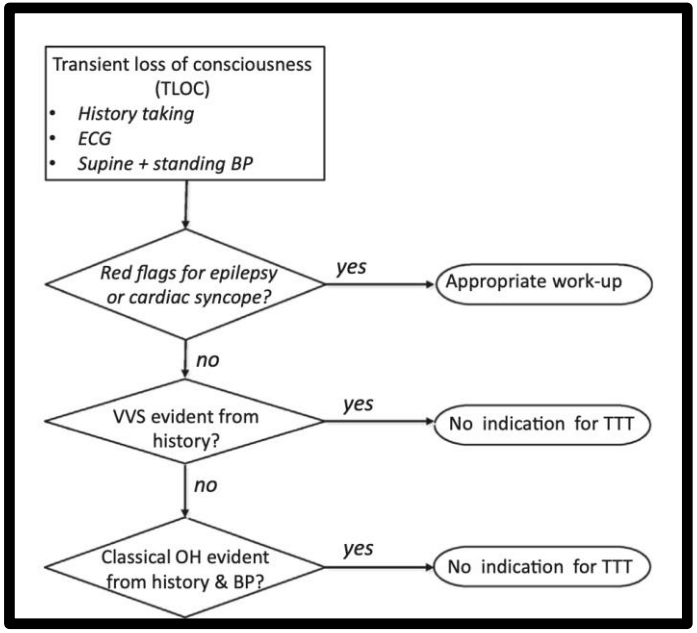
[a] Dept. of Neurology & Clinical Neurophysiology, Leiden University Medical Centre, Leiden, The Netherlands
[b] Epilepsy Institute in the Netherlands (SEIN), Heemstede, The Netherlands

HIGHLIGHTS

- Tilt table testing helps to differentiate the forms of transient loss of consciousness (TLOC) and of syncope.
- Tilt table testing adds to history taking, and cannot be used as its substitution.
- Neurologists and clinical neurophysiologists can play an important role diagnosing syncope or other forms of TLOC, given their experience with extensive history taking.



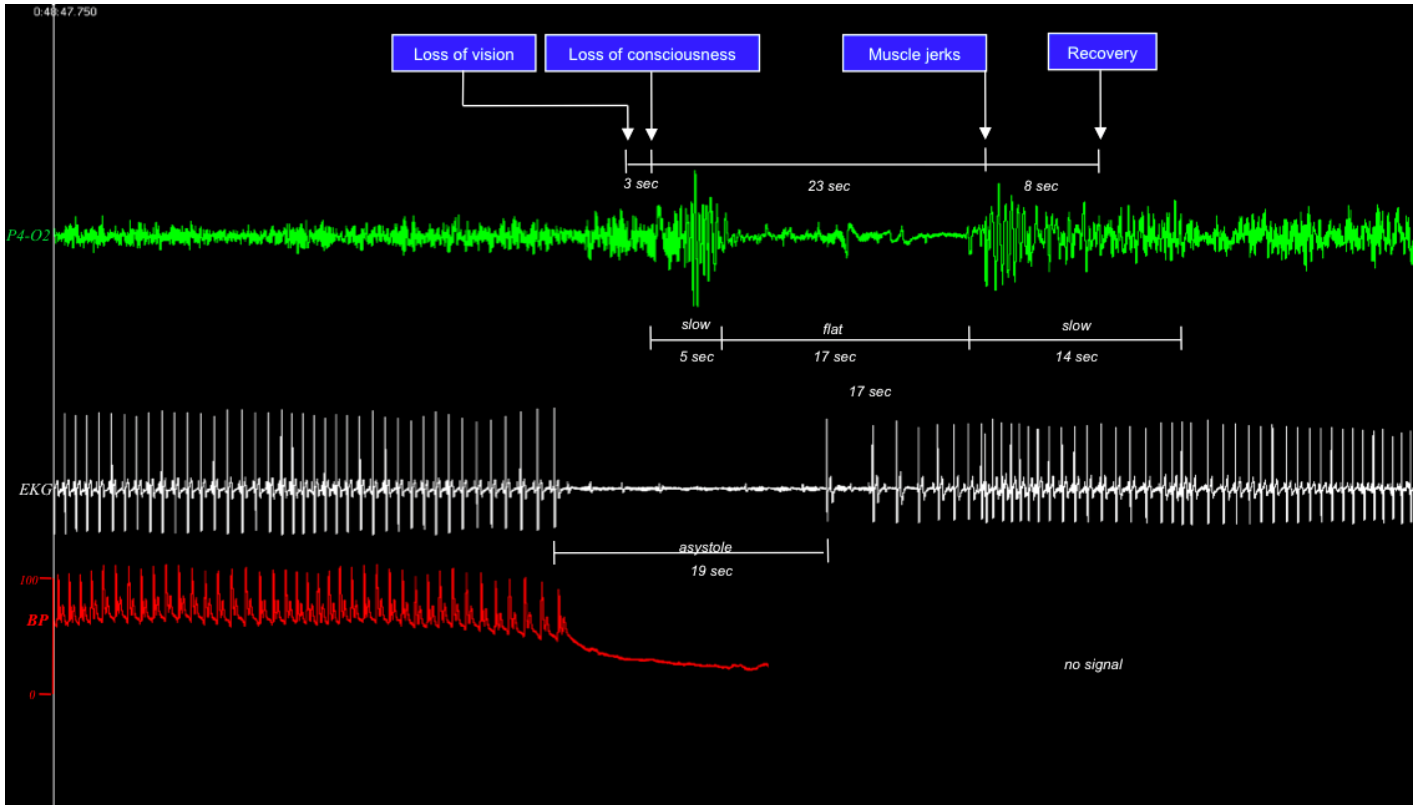
Definite diagnosis considering

- Hemodynamic pattern
- Recognition TLOC (patient/eyewitness)

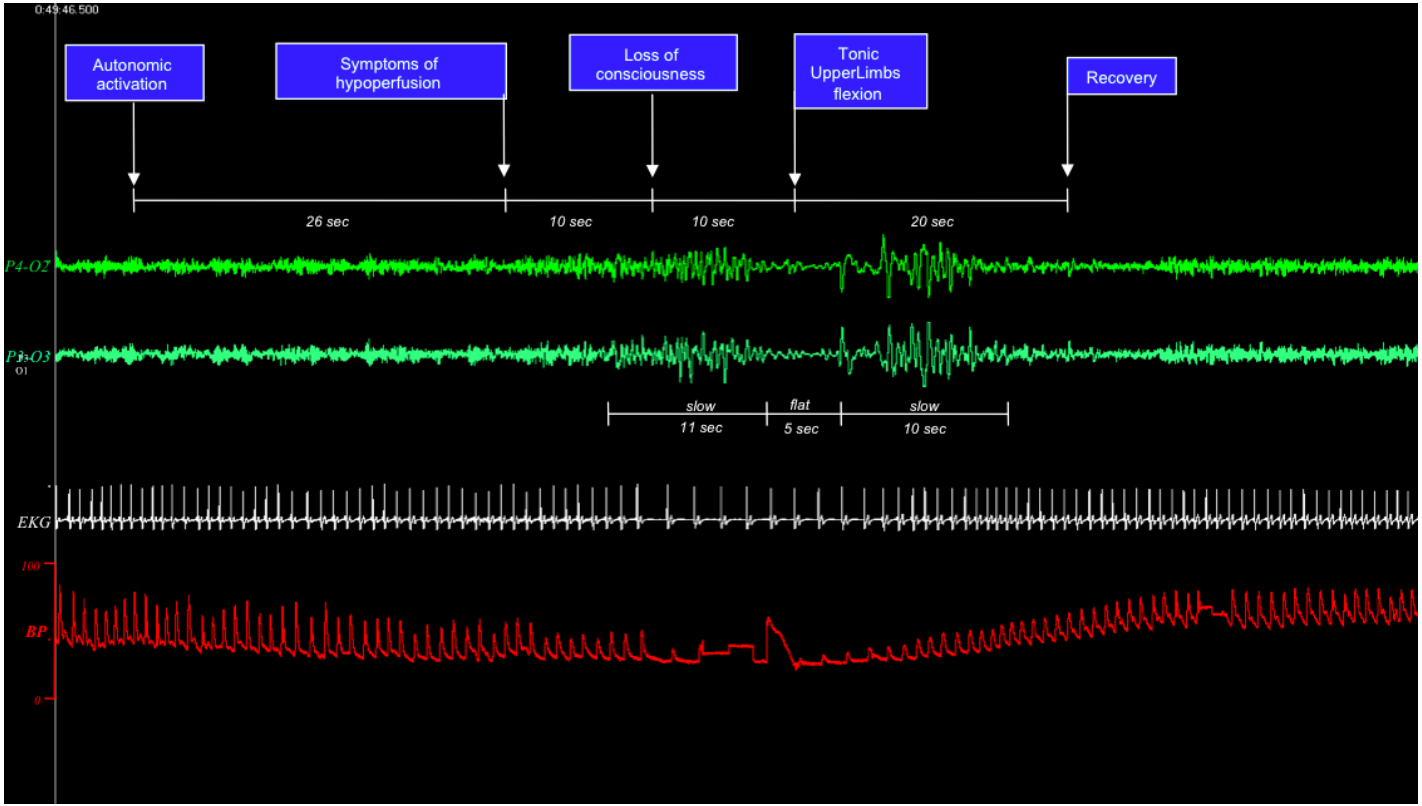
Laboratorio per lo studio del Sistema Nervoso Vegetativo

- Stanza a temperatura e umidità controllate
- Silenziosa
- Illuminazione indiretta
- Test eseguiti al mattino (inizio ore 8:00)
- Personale tecnico specializzato
- Terapie farmacologiche sospese dalla sera precedente
- Colazione leggera senza tè nè caffè
- Parametri registrati in continuo: PA (Finometer), FC, respiro oronasale e addominale, pletismogramma, ECG, posizione del corpo, video, (EEG)

Sincope vasovagale (cardioinibitoria)



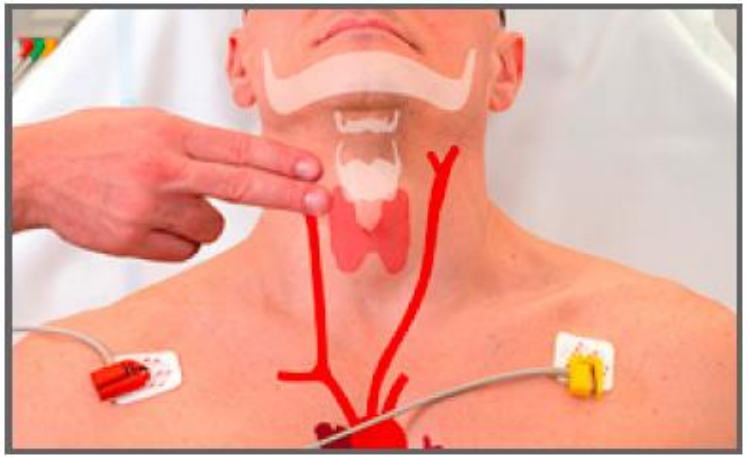
Sincope vasovagale (vasodepressoria)



Massaggio del seno carotideo

- Effettuato prima in clinostatismo, poi durante TILT test. Inizialmente il SC destro viene massaggiato per 10 secondi; dopo 1-2 minuti si ripete la procedura a sinistra
- Deve essere eseguito durante monitoraggio continuo ECG e della pressione arteriosa battito-battito

- Viene stimolata zona di massima pulsatilità dell'arteria carotide, fra l'angolo della mandibola e la cartilagine cricoidea e il margine anteriore del muscolo sternocleidomastoideo

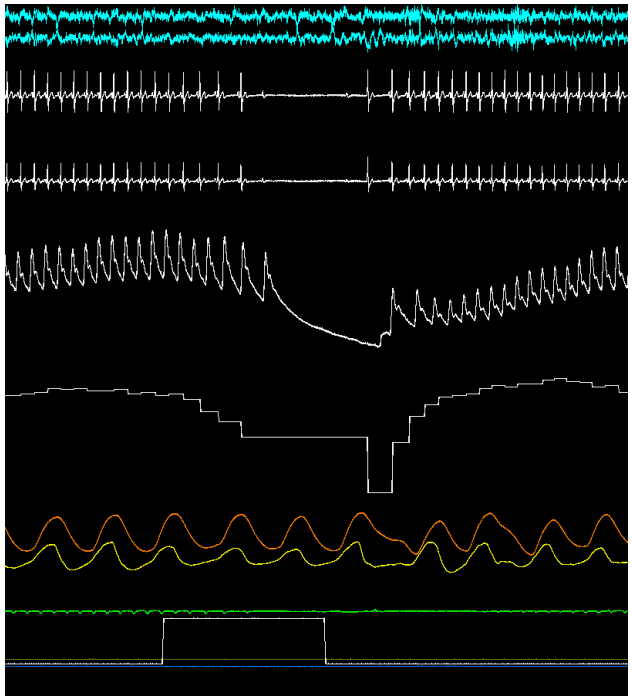


Pasquier et al. NEJM 2017

Ipersensibilità del seno carotideo

- M, 60 anni
- Motivo della richiesta: episodi di disturbi visivi in ortostatismo collegati a movimenti del capo
- Terapia: ramipril, clopidogrel, simvastatina

Ipersensibilità del seno carotideo



Risposta al massaggio del seno carotideo

Ipersensibilità del seno carotideo:

- Cardioinibitoria: asistolia > di 3 sec
- Vasodepressoria: caduta della PA > 50 mmHg o valore assoluto PA sistolica < 80 mmHg
- Mista: sia vasodepressoria che cardioinibitoria

Sincope da ipersensibilità del seno carotideo

- **Anamnesi**: soggetti di età > 40 anni
- **Contesto**: episodi stimolati da movimenti forzati di rotazione o di estensione del capo
- **Prodromi**: molto brevi o assenti

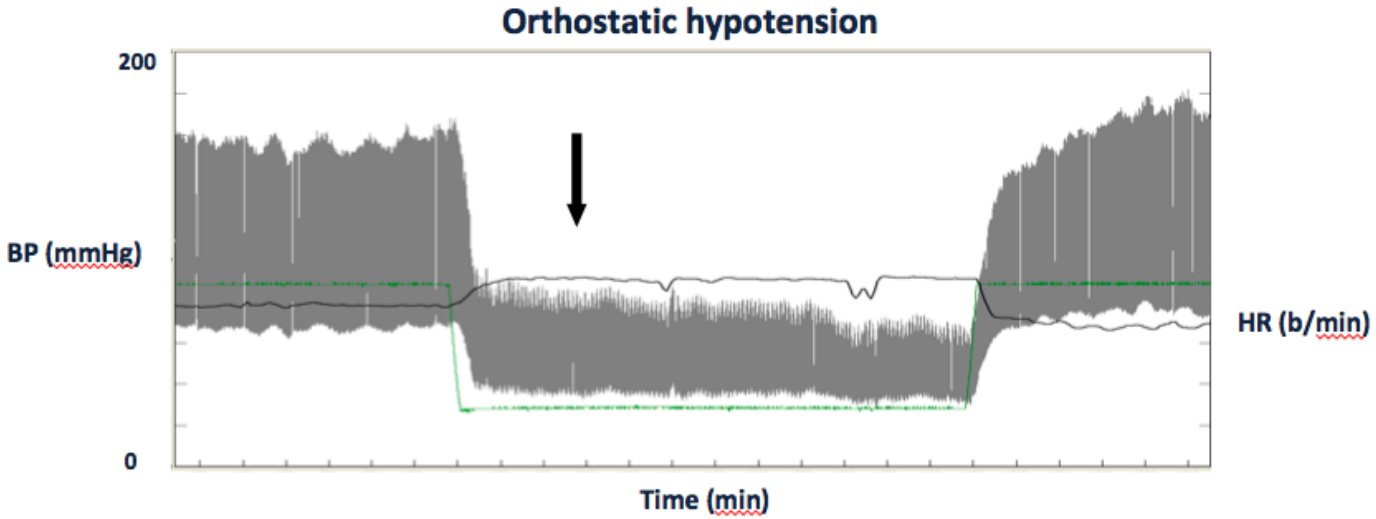
Ipotensione ortostatica

its prevalence ranges between 6% and 35% or more, depending on age and associated comorbidities, showing a strong association with elevated BP.

Sincope da ipotensione ortostatica

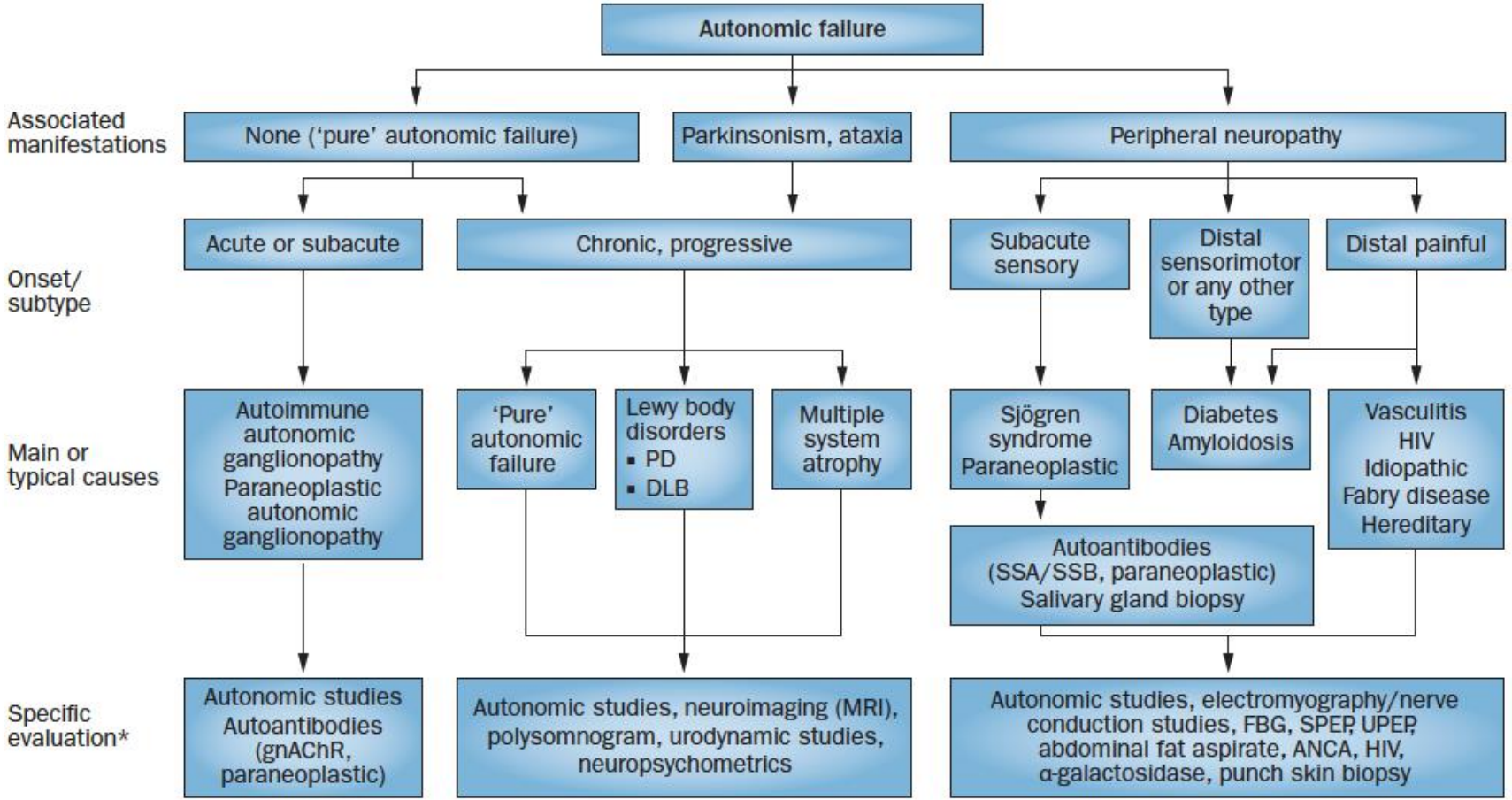
IPOTENSIONE ORTOSTATICA: riduzione sostenuta della pressione arteriosa sistolica di almeno 20mmHg e/o della pressione arteriosa diastolica di almeno 10mmHg entro 3 minuti di ortostatismo

Freeman et al 2011



Sincope da ipotensione ortostatica

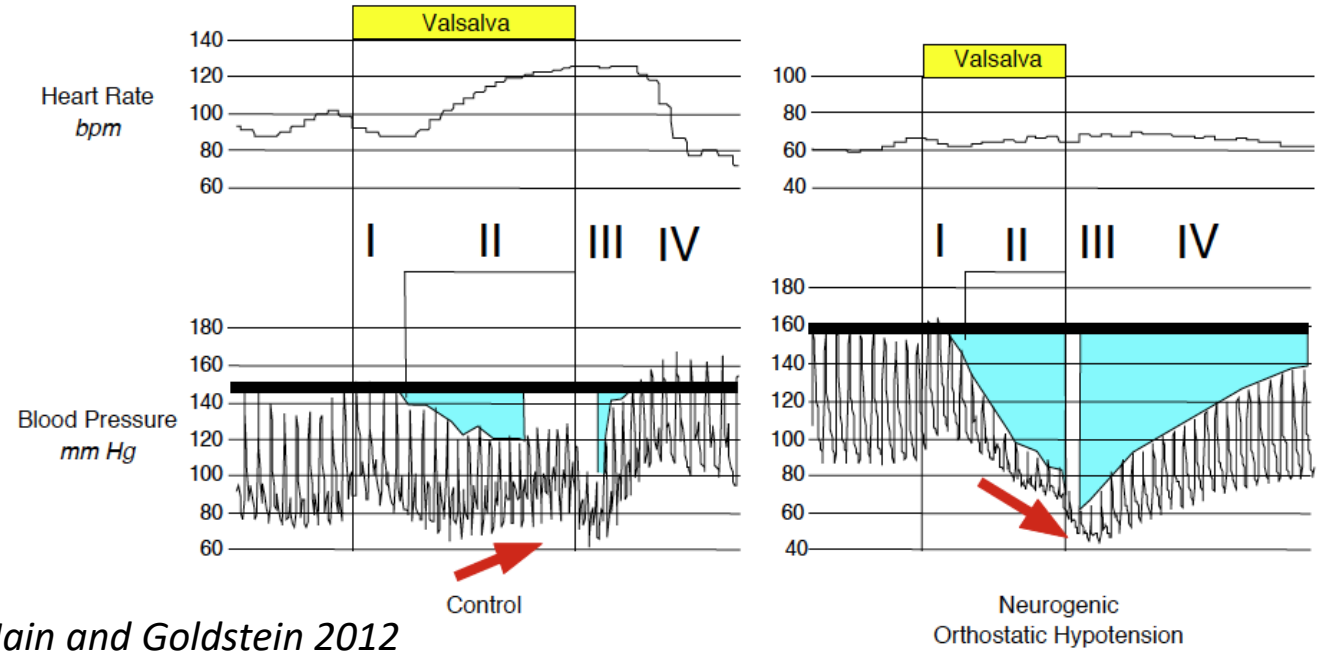
- **Anamnesi/EON**: insufficienza vegetativa (genito-urinario, gastrointestinale), disidratazione, terapie associate (anti-ipertensivi, antipsicotici, diuretici, alfalitici, antianginosi) parkinsonismo, segni cerebellari
- altri segni di **Contesto**: periodo post-prandiale, post-esercizio fisico, ambiente caldo, passaggio posturale in ortostatismo
- **Prodromi**: sensazione di testa vuota/confusa, visione offuscata, dolore ai muscoli delle spalle, pallore, nausea, astenia
- **Fase post-critica**: rapido recupero con assunzione clinostatismo



Benarroch 2014

Come differenziare ipotensione ortostatica neurogena da secondaria?

1) Manovra di Valsalva (laboratorio SNV)

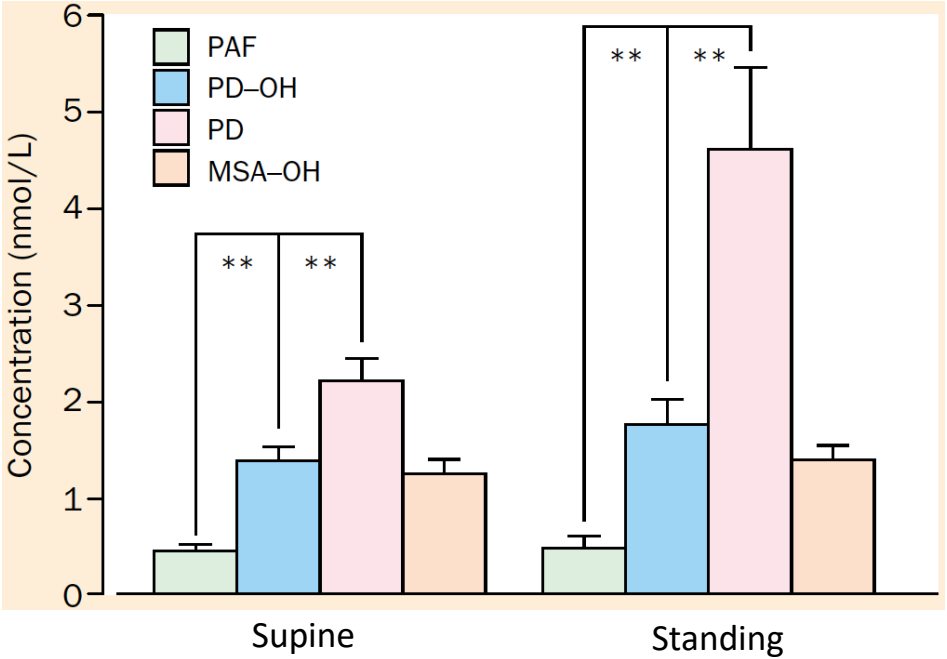


Jain and Goldstein 2012

Il soggetto soffia in un boccaglio connesso ad un manometro e mantiene una pressione espiratoria forzata di 40mmHg per 15 s

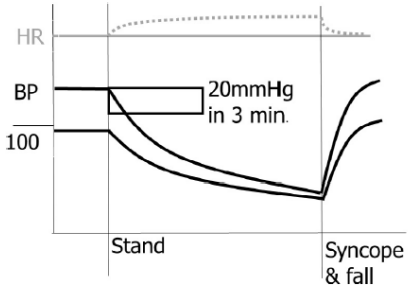
Come differenziare ipotensione ortostatica neurogena da secondaria?

2) Dosaggio catecolamine plasmatiche in posizione supina e durante tilt test

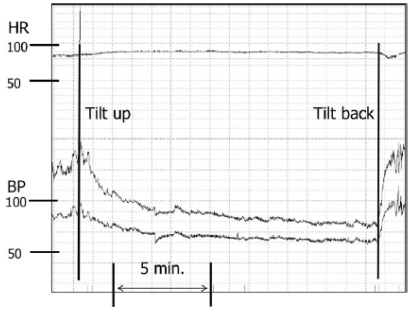


Goldstein 2003

Sincope da ipotensione ortostatica



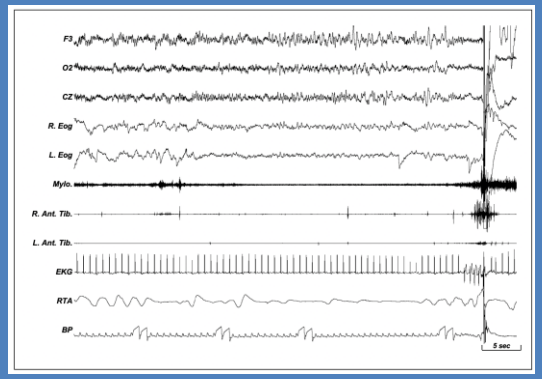
. **6a.** Circulatory patterns in syncope due to orthostatic hypotension, similar to . 3. Note that blood pressure drops immediately after standing up and that the e of drop decreases (van Dijk et al., 2009).

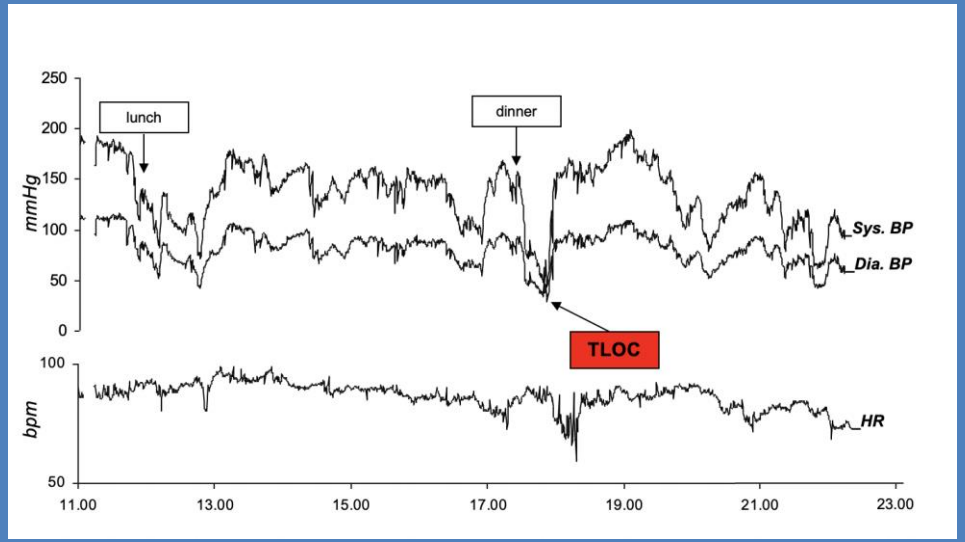


. **6b.** Circulatory patterns due to orthostatic hypotension in real life: similar to . 3b.

“Demenza ortostatica” ?

Ipotensione ortostatica post-prandiale


F3
O2
CZ
R. Eog
L. Eog
Myo.
R. Ant. Tib.
L. Ant. Tib.
EKG
RTA
BP
5 sec


250
200
150
100
50
0
mmHg
lunch
dinner
Sys. BP
Dia. BP
TLOC
100
50
bpm
HR
11.00
13.00
15.00
17.00
19.00
21.00
23.00

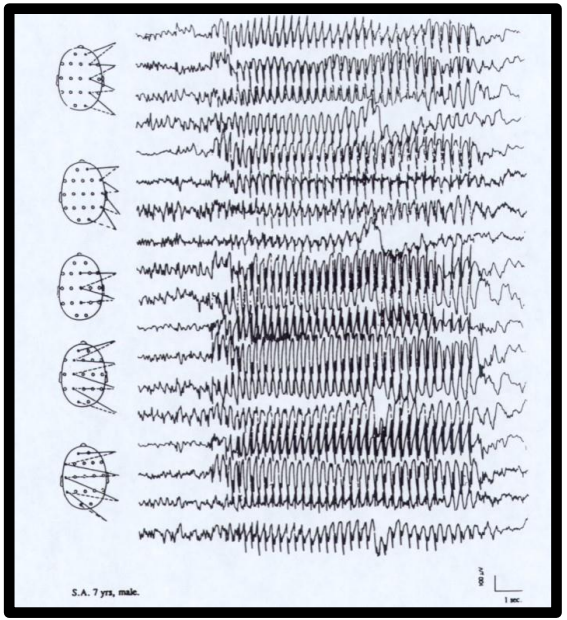
Tilt + EEG

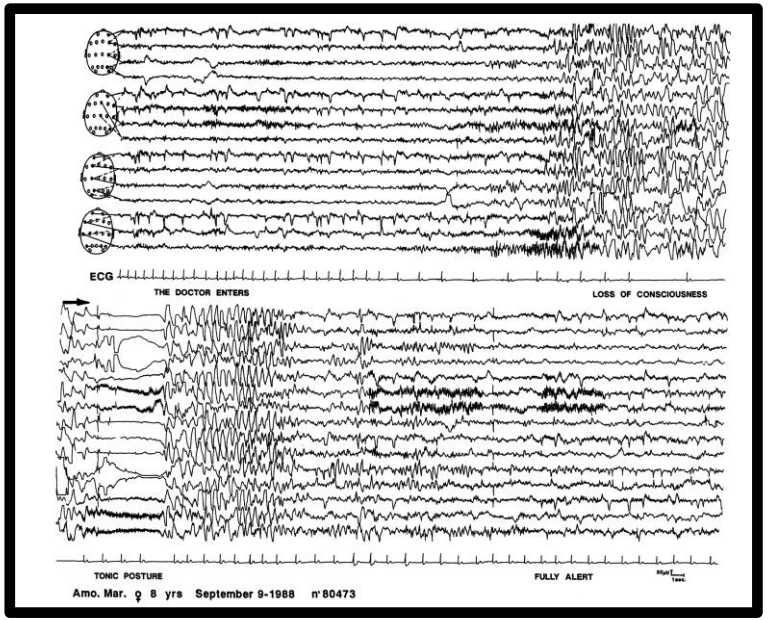
- Sospetta crisi epilettica
- Sospetta pseudosincope

“Cadute” nel paziente anziano

- Le **cadute** non altrimenti giustificate, specie se in assenza di testimoni, potrebbero essere associate ad una non consapevolezza della perdita di coscienza e andrebbero indagate come PdCT
- Nei pazienti con deterioramento cognitivo l’**anamnesi** potrebbe **non** essere **attendibile**

Sincope e crisi epilettica: fisiopatologia diversa!





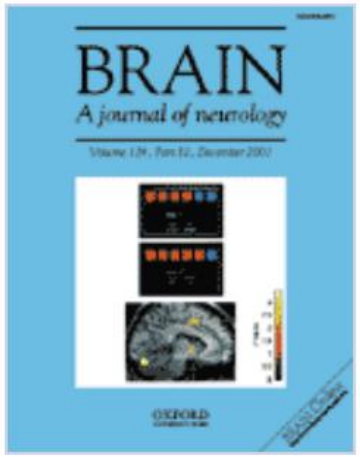
PA (m. 29 a)

A. Patologica Prossima: dai 27aa

Episodi caratterizzati da rossore al volto, occhi sbarrati con sguardo fisso associati a midriasi, calore diffuso, respiro stertoroso, sensazione di paura, impressione di estraneità nei confronti dell'ambiente, malessere gastrico, arresto della parola, automatismi gestuali e deambulatori. A volte perdita di urine. Al termine dell'episodio il paziente racconta dettagliatamente ciò che è accaduto.

Durata : 50"

Frequenza: alternanza di periodi a frequenza pluriquotidiana a periodi liberi anche di 4-5 mesi



Volume 124, Issue 12
December 2001

Brain (2001), **124,** 2361–2371

Ictal bradycardia in partial epileptic seizures

Autonomic investigation in three cases and literature review

P. Tinuper,[1] F. Bisulli,[1] A. Cerullo,[1] R. Carcangiu,[1] C. Marini,[1] G. Pierangeli[1] and P. Cortelli[2]

[1]*Neurological Institute, University of Bologna and* [2]*Neurological Section, University of Modena and Reggio Emilia, Italy*

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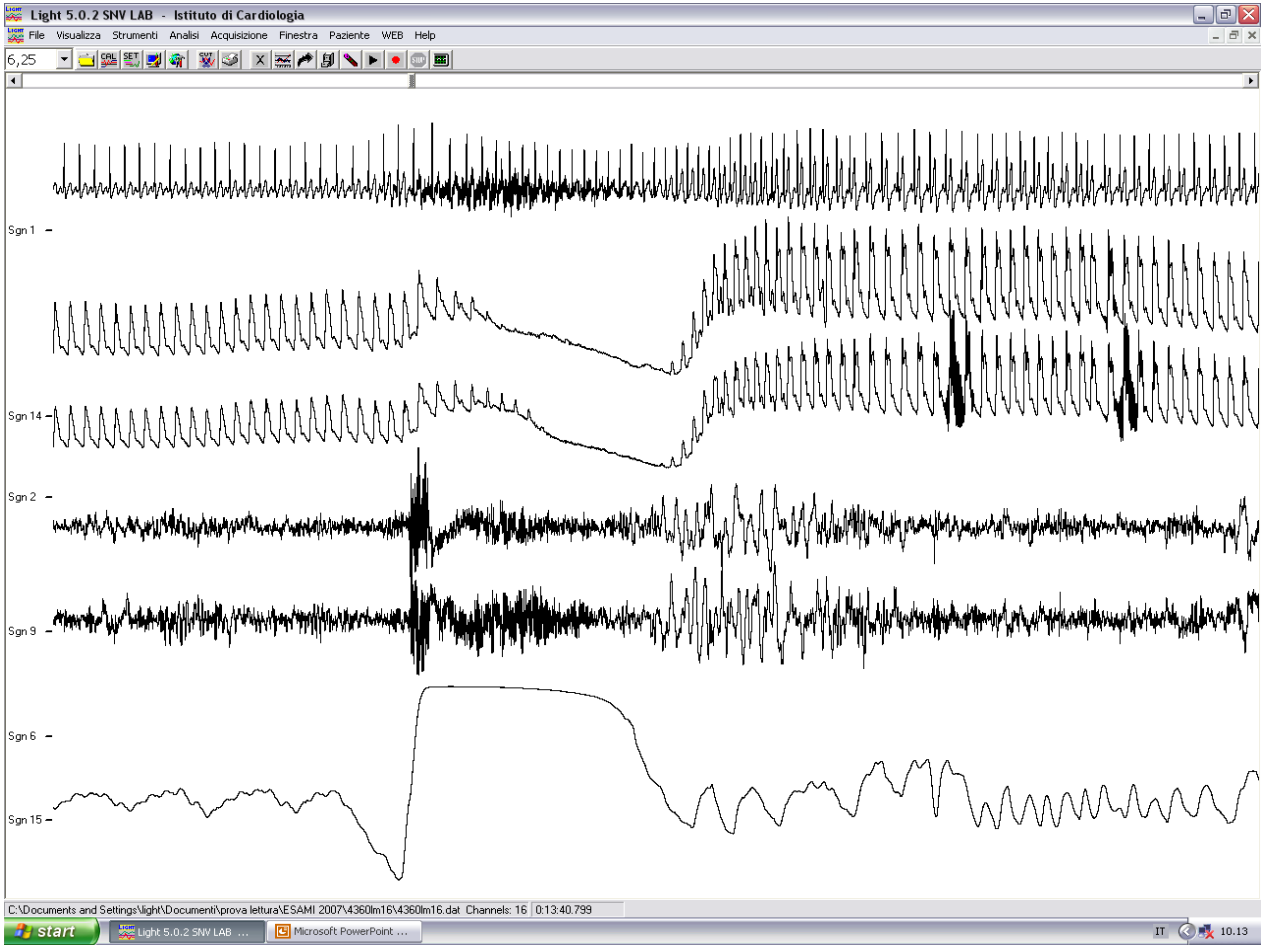
E-mail: tinuper@neuro.unibo.it

Sincope o Epilessia?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6,25
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Microsoft PowerPoint ...
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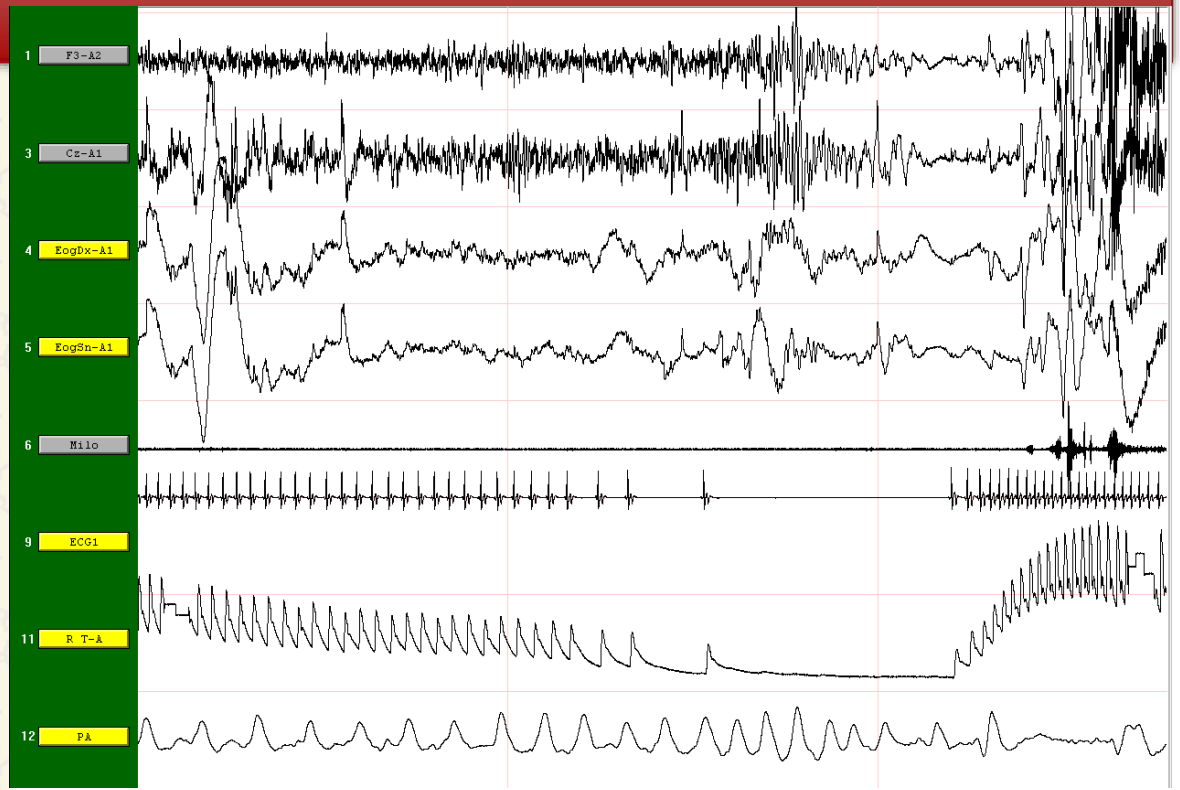
Video episodio critico

Video–EEG: registrazione episodio

Poligrafia notturna:

"si sveglia dicendo che forse ha avuto qualcosa"



TAC del collo

JACC: CASE REPORTS

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VIEWPOINT

BEGINNER

EDUCATIONAL CORNER

An Elderly Woman With Recurrent Transient Loss of Consciousness Preceded by Hallucinatory Attacks

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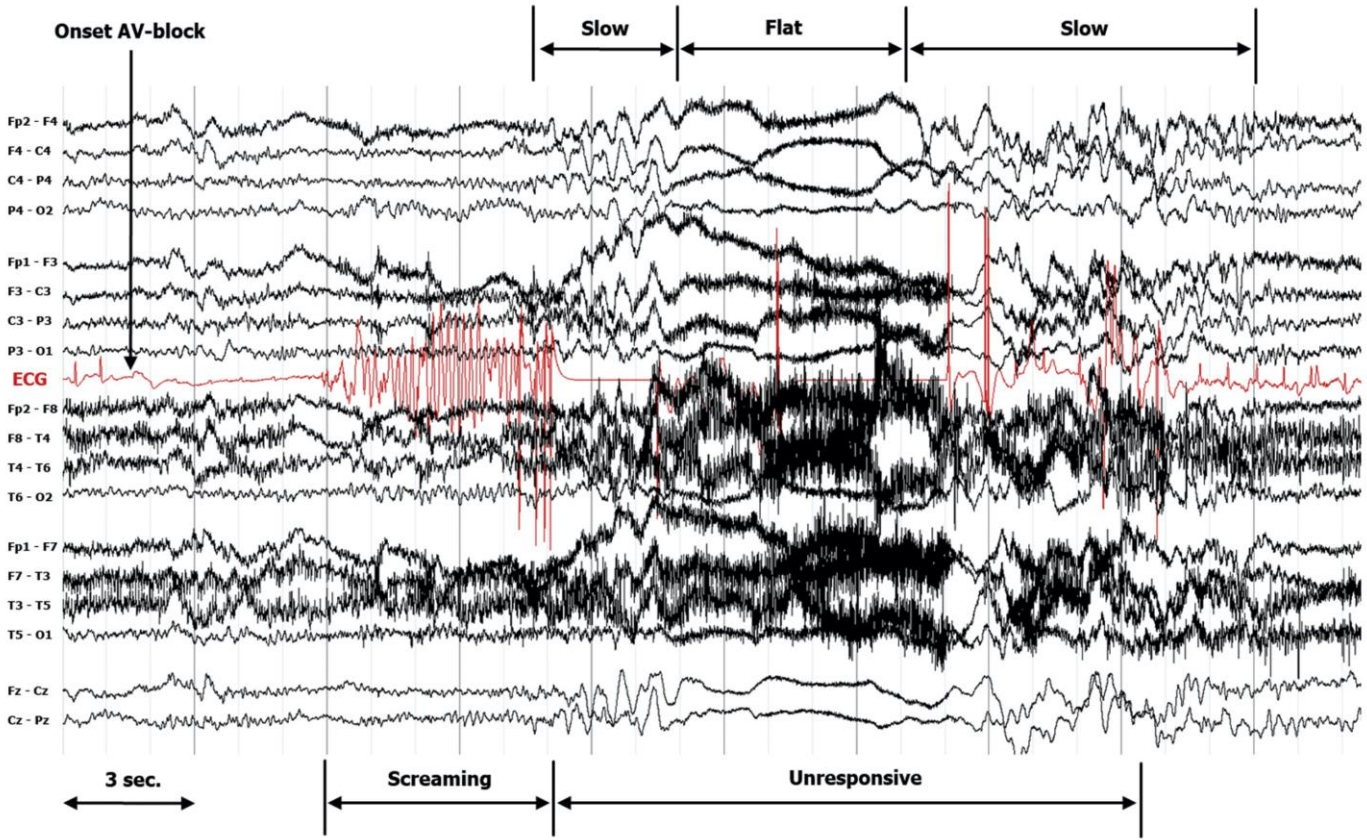
ABSTRACT

We report a patient with transient loss of consciousness preceded by hallucinations caused by cardiac syncope. The diagnosis was confirmed by simultaneous video-electrocardiogram-electroencephalogram recording of an event. The attacks no longer occurred after pacemaker implantation. Clinical features helpful in guiding decision making in transient loss of consciousness are discussed. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:1824–7) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

FIGURE 1 21-Lead EEG According to International 10/20 System (Bipolar Montage; Sensitivity 70 μV) and Simultaneous ECG Monitoring



In chronological order, the figure depicts onset of atrioventricular block, screaming and shaking with motion artifact on electrocardiogram (ECG), onset of slow high-amplitude electroencephalogram (EEG) activity, which is soon followed by loss of consciousness, and flattening of the EEG signal. Soon after, QRS complexes reappear on the ECG and slow EEG activity returns, the patient regains consciousness. This slow-flat-slow EEG pattern is a typical feature of syncope.

LEARNING OBJECTIVES

- To make a differential diagnosis in cases of transient loss of consciousness based on history-taking and interictal examinations.
- To recognize atypical cases of transient loss of consciousness and be aware of the need to register an event to establish a definitive diagnosis in such cases.
- To be aware that hallucinations can occur in syncope, either before loss of consciousness or when regaining consciousness.

Autonomic Unit – Clinica Neurologica UNIBO
Founded 1985

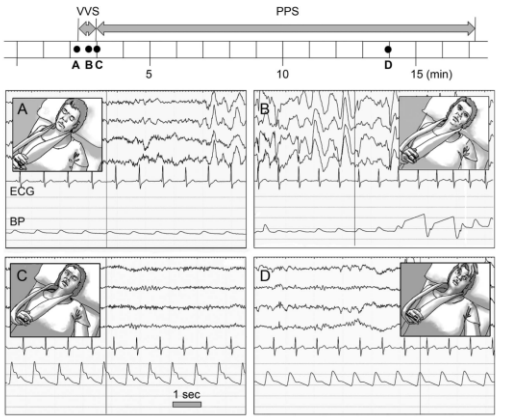
Perdita di coscienza transitoria: sintomo comune di diagnosi difficile

Apparent LOC

Altre cause di PdCT

- Cataplexy (part of narcolepsy)
  - No LOC! No amnesia!
  - Paralysis evoked by emotions
- Remember:
  - Laughter is a trigger

Figure Example of consecutive VVS and PPS



ARTICLES

Tilt-induced vasovagal syncope and psychogenic pseudosyncope

Overlapping clinical entities

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ABSTRACT

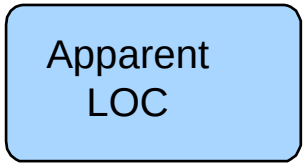
Objective: To describe the combination of tilt-induced vasovagal syncope (VVS) and psychogenic pseudosyncope (PPS) and aid its clinical recognition.

Methods: We identified people with tilt-induced VVS/PPS from 2 tertiary syncope referral centers. For each case, 3 controls with tilt-induced VVS were selected at random from the same center. Clinical characteristics were compared between both groups adjusting for multiple comparisons.

Results: Of 1,164 tilt-table tests, 23 (2%) resulted in VVS/PPS; these 23 cases were compared with 69 VVS controls. VVS and PPS coincided more often than chance would predict: 2% vs 0.6%, $p < 0.001$. Typical VVS prodromes and triggers were reported in all people with VVS/PPS and in controls with VVS. Attack frequency was significantly higher in the VVS/PPS (2 per month, range 0.1–60) than in the VVS group (0.25 per month, range 0.02–4; $p < 0.001$). Delayed recovery of consciousness was more frequently reported in the VVS/PPS group (likelihood ratio [+LR] 8.14, 95% confidence interval [CI] 3.94–16.84), as well as episodes without prodromes (+LR 5.57, 95% CI 2.53–12.26), atypical triggers (+LR 5.00, 95% CI 2.04–12.24), eye closure (+LR 3.75, 95% CI 1.68–8.35), and apparent loss of consciousness >1 minute (+LR 2.86, 95% CI 1.98–4.13).

Conclusions: VVS/PPS presents with a complex phenotype. High attack frequency, delayed recovery of consciousness, apparent loss of consciousness >1 minute, ictal eye closure, atypical triggers, and the absence of prodromes may serve as indicators that PPS coincides with VVS.

Neurology® 2015;85:2006–2010


Apparent
LOC