Syncope

이 태 경
순천향의대

Coverage

- **Classification of syncope**
  Pathomechanism of syncope
- **Clinical approach to syncope**
  Identify syncope by history
  Initial evaluation
  Essential tests for syncope

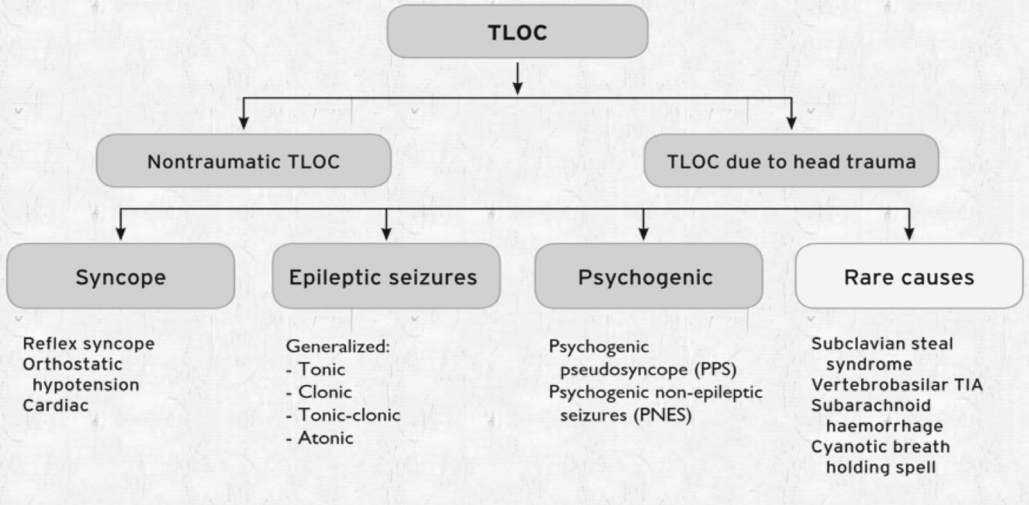
Definition of Syncope

~~" Syncope is *a sudden loss of consciousness associated with the inability to maintain postural tone followed by spontaneous recovery.*"~~

"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.

Syncope in the context of transient loss of consciousness



Classification of syncope

Reflex (neurally-mediated) syncope

Vasovagal:
- Mediated by emotional distress: fear, pain, instrumentation, blood phobia.
- Mediated by orthostatic stress.

Situational:
- Cough, sneeze.
- Gastrointestinal stimulation (swallow, defaecation, visceral pain).
- Micturition (post-micturition.)
- Post-exercice.
- Post-prandial.
- Others (e. g., laugh, brass instrument playing, weight lifting).

Carotid sinus syncope

Atypical forms (without apparent triggers and/or atypical presentation).

Syncope due to orthostatic hypotension

Primary autonomic failure:
- Pure autonomic failure, multiple system atrophy. Parkinson's disease with autonomic failure, Lewy body dementia.

Secondary autonomic failure:
-Diabetes, amyloidosis, uraemia, spinal cord injuries.

Drug-induced orthostatic hypotension:
- Alcohol, vasodiators, diuretics, phenotiaz, antidepressants.

Volume depletion:
-Haemorrhage, diarrhoea, voming, etc.

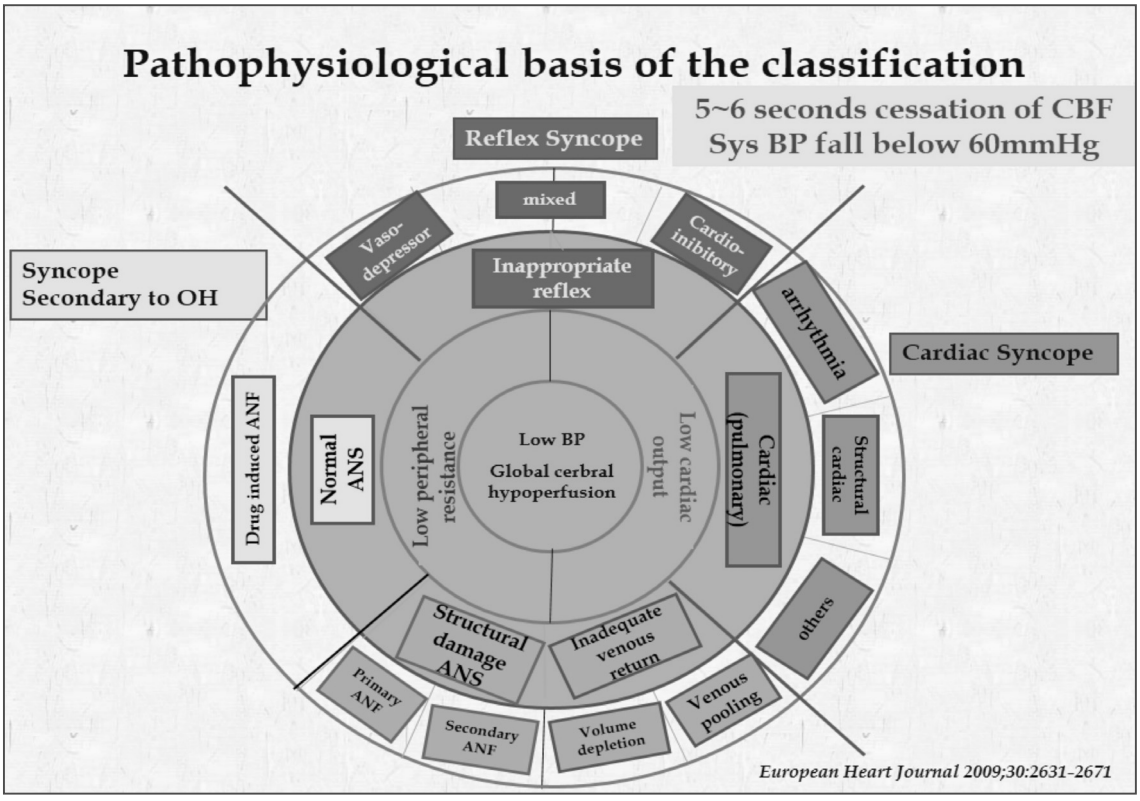
Cardiac syncope (cardiovascular)

Arrhythmia as primary cause:
Bradycardia:
-Sinus node disfunction (including brady-cardia/ tachycardia syndrome).
-Atrioventricular conduction system disease.
-Implanted device malfunction.
Tachycardia:
-supraventricular.
-Ventricula (idiopathic, secondary to structural heart disease or to channelopathies).
Drug induced bradycardia and tachyarrhythmias.

Structural disease:
Cardiac: cardiac valvular disease, acute myocardial infarction/ischaemia, hypertrophic cardiomyopathy, cardiac masses (atrial myxoma, tumor, etc).
Pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valves dysfunction.
Others: pulmonary embolus, acute aortic dissection, pulmonary hypertension.

European Heart Journal 2009;30:2631-2671

Pathophysiological basis of the classification



European Heart Journal 2009;30:2631-2671

Reflex(= Neurally mediated) Syncope

Sudden failure of the autonomic nervous system to maintain BP and sometimes HR at a level sufficient to maintain cerebral perfusion and consciousness.

1. Vasovagal (= neurocardiogenic, = common faint) syncope is the M/C group of reflex (neurally mediated) syncopes.
2. Carotid sinus syndrome
3. Situational syncope

- ***No increased risk for cardiovascular morbidity or mortality***

VVS(common faint)

- Neurally-mediated physiologic reflex mechanism with two components:
  1. Cardioinhibitory (↓ HR) : **caution:** sudden asystole !
  2. Vasodepressor (↓ BP) despite heart beats, no significant BP generated
- Both components are usually present
- Most common form of syncope
  - 8% to 37% (mean 18%) of syncope cases
- **Precipitating event** is often identifiable
  - Stress, trauma, pain, sight of blood, prolonged standing, heat exposure…

VVS(common faint)

Three Phases

- Prodrome (increased sympathetic activity)
  Diaphoresis, epigastric discomfort, weakness, nausea, dizziness
  Lasts about 2 minutes
- Loss of consciousness (sympathetic withdrawn, inc. vagal activity)
  Usually lasts 5-20 seconds
- Postsyncopal phase
  Nausea, dizziness, general sense of poor health
  If present, confusion which lasts no more than 30 seconds

Carotid Sinus Syndrome(CSS)

- Syncope clearly associated with carotid sinus stimulation is rare (≤1% of syncope)
- CSS may be an important cause of unexplained syncope/falls in older individuals
- Prevalence higher than previously believed
- Carotid Sinus Hypersensitivity (CSH)
  - No symptoms
  - No treatment

Situational Syncope

- Urination(micturition)
- Defecation
- Swallowing
- Coughing(tusive) etc.

Mechanoreceptors are present throughout the body (in the bladder, rectum, esophagus, and lungs), and it is thought that the sudden activation of a large number of these receptors also sends afferent signals to the brain, which provokes a similar response

Pathophysiology of Neurally-mediated Syncope

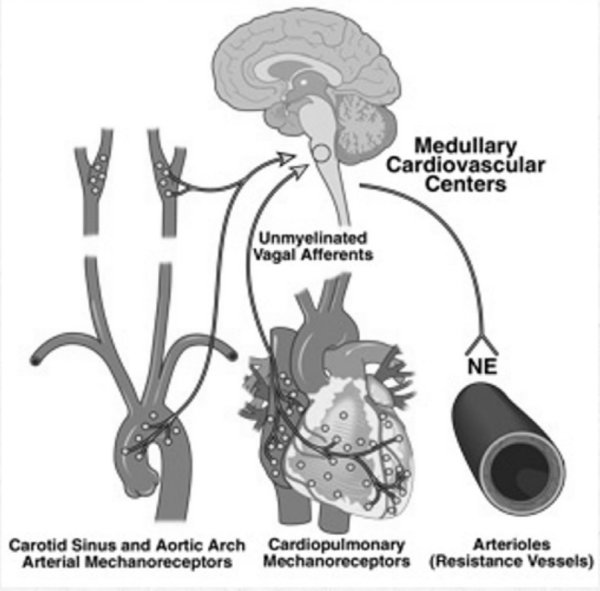
Pathophysiology of Syncope

❖ Decreased cerebral perfusion

❖ Cessation of cerebral perfusion for as little as **3-5 secs** can result in syncope

❖**35% reduction in CBF** will cause syncope

❖ Decreased cerebral perfusion may occur as a result of **decreased C/O or decreased systemic vascular resistance.**



Postulated Mechanism

Predisposition to the condition as a result of **excessive peripheral venous pooling**

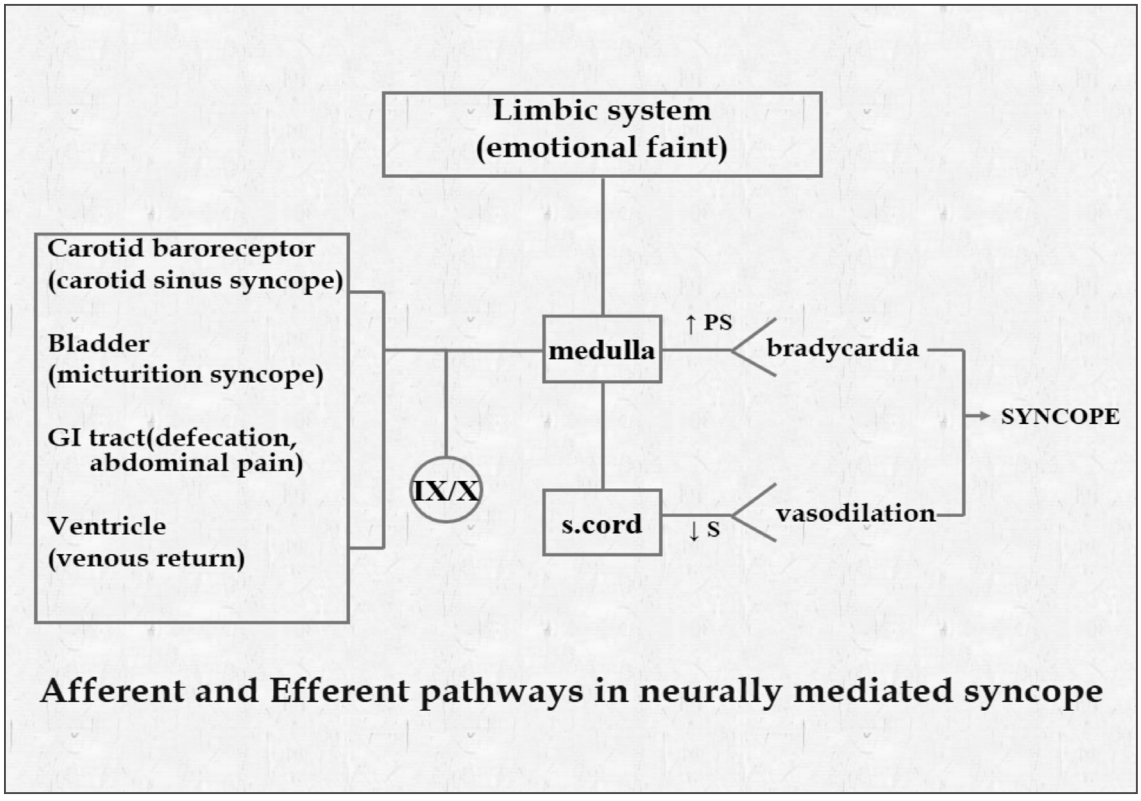
→ Sudden drop in peripheral venous return

→ Cardiac ***"hypercontractile"*** state

→ Activation of mechanoreceptors that normally respond only to stretch

→ Increase in afferent neural traffic to the brain mimics the conditions seen in hypertension

→Provoke an apparent ***paradoxical reflex bradycardia*** and a drop in peripheral vascular resistance.



Afferent and Efferent pathways in neurally mediated syncope

Case vignette I

반복되는 의식소실로 병원에 온 21세 여자
약 1년 전부터 5회의 의식소실

쓰러지기 전 어지럼, 숨막힘, 메스꺼움, 눈 앞이 뿌옇게 흐려짐

사람이 많은 출근 전철 안에 서 있다가, 목욕탕에서 나오던 중

쓰러졌을 때 창백, 늘어짐, 얕은 호흡. 이상 움직임은 없음
40 초 내에 회복 후 무력감 외에 증상 없음
평소 불규칙한 식사와 수면, 스트레스

Case vignette I

P & N/Ex : normal,
P & FHx.: N-C
EKG : normal
Lab. : normal
Orthostatic BP tests : normal

Can we diagnose this case as VVS at this moment?
What else do we evaluate further?
Are autonomic function tests useful?

Recognition of Syncope by History

Can we diagnose reflex syncope by history alone?

Clinical features that can suggest a diagnosis on initial evaluation - Class I, level C

Neurally mediated syncope

Absence of heart disease

Long history of recurrent syncope

After sudden unexpected unpleasant sight, sound, smell or pain

Prolonged standing or crowded, hot places

Nausea, vomiting associated with syncope

During a meal or post-prandial

With head rotation or pressure on carotid sinus
(as in tumors, shaving, tight collars)

After exertion *vs. during exertion*

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Initial evaluation

Initial evaluation of a patient presenting with TLOC consists of

Careful history alone identifies the cause **up to 85%**

P/Ex including

Orthostatic BP measurement

EKG

Based on these findings,

additional examinations may be performed.

Common Precipitations VVS

- Prolonged standing
- Stressful stimuli
  Fear, pain, invasive instrumentation

Common Associated Factors VVS

- Hot weather
- Lack of food
- Rapid early morning rising

Typical Prodromes in NMS

- Light headedness
- Fatigue
- Blurred vision
- Sweating
- Nausea
- Palpitaions

Case vignette II

- 3회의 의식 소실로 온 23세 여자
- 작년 여름, 가을 등산 후, 내원 하루 전 회식 때
- 두통, 온몸 저림, N/V 후 의식 잃는다. 금방 회복
- 좁은 곳에서 검사시
- 사람 많은 곳, 지하철에서 쓰러질 것 같다.
- Lab. EKG, EEG : normal

Common precipitants : postprandial, post-exercise, alcohol, narrow space, crowded, claustered situation

Prodrome : dizzy, paresthesia, N/V, headache

Case vignette I & II

P & N/Ex : normal, P & FHx.: N-C

EKG : normal

Lab. : normal

Orthostatic BP tests : normal

AFT(HUT) : normal

We can diagnose this case as VVS at this moment without further study.

Essential questions

- Triggering patterns
- Prodromal symptoms and signs
- Speed of onset
- Ictal phenomena
- Postictal events

Questions about circumstances just prior to the attack

- **Position** - supine, sitting or standing
- **Activity** - rest, change in posture,
  during or after exercise,
  during or immediately after urination, defecation,
  cough, or swallowing
- **Predisposing factors** - crowded or warm places,
  prolonged standing,
  post-prandial period

Precipitating events - fear, intense pain,
neck movements

"what were you doing at that time?"

most important question

Case III

- 35세 남자
- 야구하다가 공을 눈에 맞은 후 눈에 통증 지속됨.
- 안과에서 진료 후 나오는데 갑자기 어지럼 바로 쓰러짐.
- 1분 이내의 의식소실
- 안와 주위의 통증과 멍
- Normal N/Ex
- Normal EKG
- Normal lab.
- Aschner's test - unevoked

Case IV

기침을 반복하다가 정신을 잃은 54세 남자

약 5년 전부터 만성기관지염으로 약 복용중

쓰러졌을 때 창백, 늘어짐, 얕은 호흡. 이상움직임은 없음

1분 내에 회복.

입원 후 3번 정도 비슷한 증상 반복

기침이 없어지면서 증상도 없음.

Case V

Cough syncope — a diagnosis under pressure

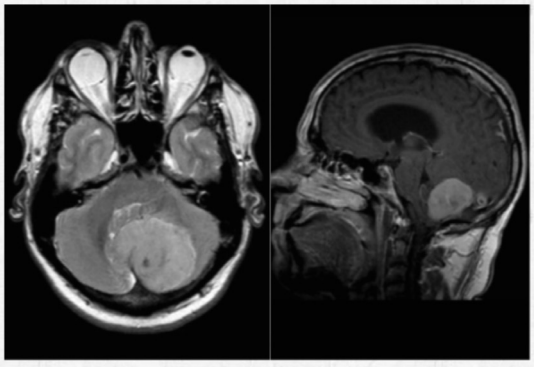
54/M

: Recurrent syncopal attacks with nonproductive coughing rapid recovery

Duration : 2-5 min, no precipitants

Had experienced presyncope, headaches, and syncope triggered by coughing

Valsalva manoeuvre → headache and presyncope



Reflexive tusive syncope와 달랐던 점

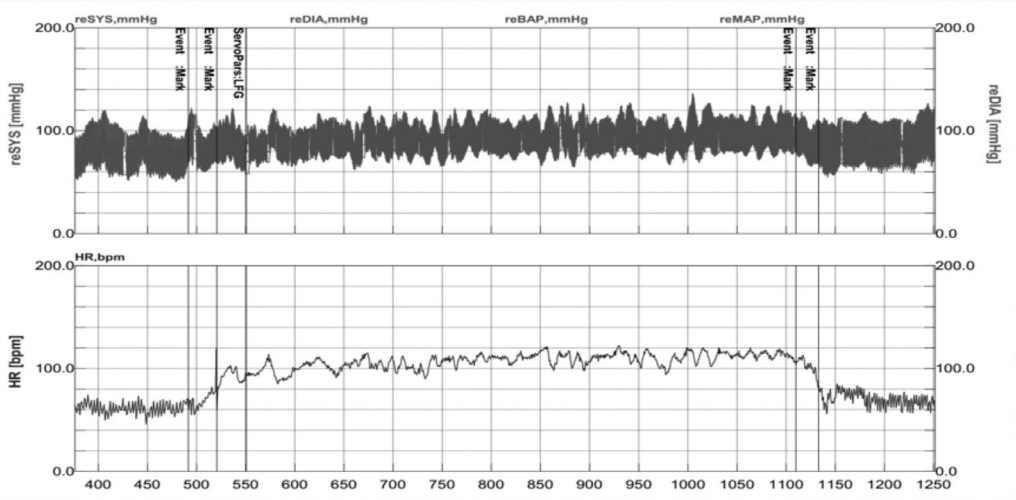
두통 동반, 2-5 min duration,
valsalva maneuver 에 의해 두통과
전실신 증상이 유발됨.

Lancet 2010; 376: 486 Annette Maznyczka, Ian B Squir

Case vignette VI

- 일어설 때마다 어지럽다고 온 23세 여자
- Recurrent dizzy (중학교때부터) → presyncopal attack
  - 화장실에서 배가 아프다가
  - 속 울렁거리고 화장실 가고 싶고 식은땀, 오한
  - 1시간 반정도 지나면 회복
  - TLOC (최근 2번의 attack) ; less than a min.
  - Standing BP → no BP drop
  - EKG : normal

Case vignette VI



Dx.? Postural (orthostatic) tachycardia syndrome, PoTs

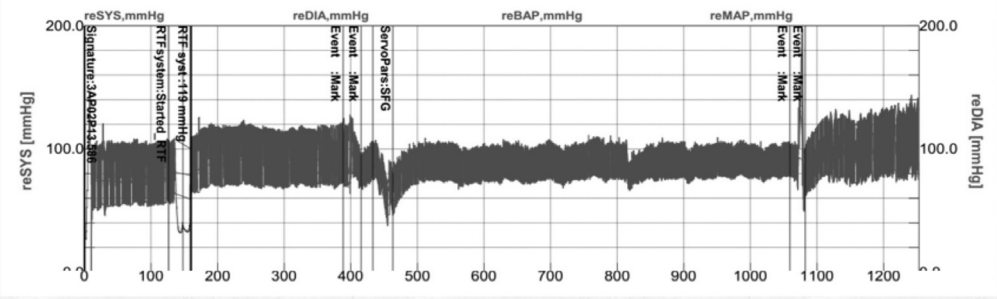
Case VII

새벽에 화장실 가려고 일어나다가 쓰러진 59세 남자

쓰러지기 전 : 눈 앞이 뿌옇게 흐려짐, 다리에 힘이 풀림

약 2년 전부터 전립선비대증으로 Cadura 4mg/day 복용중

쓰러졌을 때 창백, 늘어짐, 얕은 호흡. 이상움직임은 없음



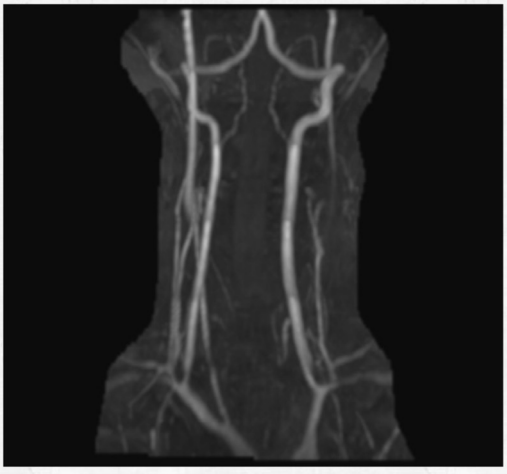
Case VIII

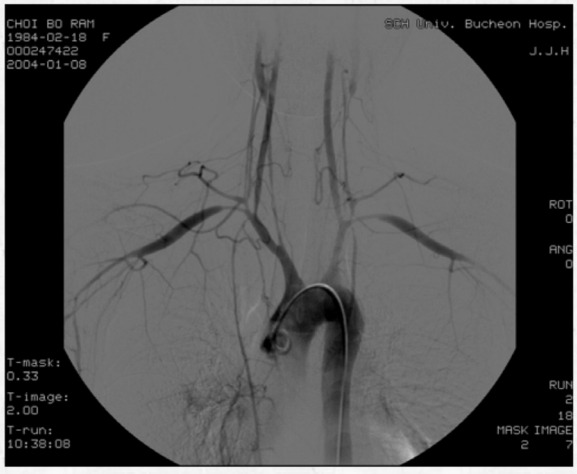
- 19세 여자
- 일어날 때마다 어지럽다. 최근 3번 의식을 잃음. 쓰러지면 창백, 늘어짐, 금방 깨어남. 의식 깨어나면 두통이나 혼돈은 없음.

- ***가위질하다가 팔이 저리고 아프고 어지러웠다.***
- 일어서면 어지럽고 식은땀, 열감,

- **Dyspnea, chest discomfort** for about 2 months
- 평소 소식, 잠을 잘 못잔다.
- Anemia 있어 철분제 드시고 있음.
- **Mild fever 37.8** B.P 95/60 mmHg, HR 104
- Lab. 9.4/30, Plt 449, **ESR 120, CRP 12.3**
- EKG, Chest, Echo, USG, GFS - normal

Case VIII





Caution !!! **Dyspnea, chest discomfort, fever, elevated ESR, CRP**
Arm movement associated

Questions about the background

- **Family history** of **sudden death,**
  congenital arrhythmogenic heart disease
- Previous **cardiac disease**
- **Neurological history** -Parkinsonism, epilepsy, narcolepsy
- **Metabolic disorders** - diabetes, etc.
- **Medication** – antihypertensives, antianginal,
  antidepressant agent, antiarrhythmics,
  diuretics, and QT-prolonging agents
  or other drugs including alcohol

Clinical features that can suggest a diagnosis on initial evaluation - Class I, level C

Cardiovascular syncope

- Presence of **definite structural heart disease**
- FHx. of **unexplained sudden death** or channelopathy
- **During exertion, or supine**
- **Abnormal EKG**
- Sudden onset **palpitation** immediately followed by syncope
- **EKG findings** suggesting arrhythmic syncope
- Bifascicular block : LBBB or RBBB combined with left anterior or left posterior fascicular block
- Other intraventricular conduction abnormalities (QRS duration ≥0.12 s)
- Mobitz I 2nd degree AV block
- Asymptomatic inappropriate **sinus bradycardia(<50 bpm)**, sinoatrial block or **sinus pause≥3** s in the absence of negatively chronotropic medications
- Non-sustained **VT**

Case IX

6년 전부터 반복되는 TLOC로 온 59세 남자. 5회/년 정도의 발작.

사지가 뻣뻣하다가 떨었다. → myoclonic movements 의심.

뇌파와 뇌 MRI는 정상, 항경련제 복용중.

돌연사의 가족력은 없음.

처음 발생: 2006년 앉아서 일하던 도중 갑자기 쓰러짐.

최근 한달 사이에 20회 이상의 의식소실. 하루에 4번 이상 쓰러져 입원함.
주로 앉아 있을 때 쓰러짐.

쓰러지기 바로 전 가슴에서 치밀어 오르는 느낌 외에 다른 Prodrome은 없음.

피로하거나 과음 후에는 항상 발작.

Factors Strongly Suggesting Epilepsy

- Tongue biting
- Turning of the head
- Muscle pain
- Duration of loss of cons(>5min.)
- Cyanosis
- Postictal confusion
- Incontinence may occur in about 20% of syncope
  → no definite exclusion of syncope

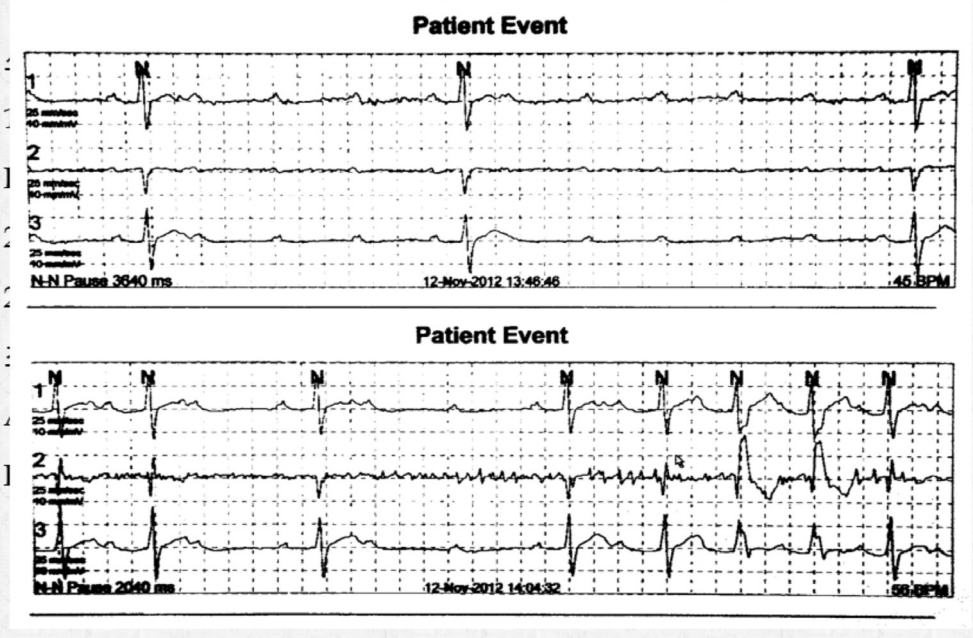
Factors Strongly Suggesting Syncope

- Prolonged sitting or standing
- Sweating before loss of cons.
- Nausea
- History of presyncope
- Paleness

Myoclonic Jerks in Syncope

- Prolonged asystole of 10 to 14 seconds or a prolonged period of a very low BP may cause myoclonic jerk. – m/c in cardioinhibitory
- Usually not rhythmic and not synchronous in the extremities
- Never occur before falling.

Case IX



Essential Tests for Syncope

Limitation of all Diagnostic Tests of Syncope

- Syncope is a transient symptom and not a disease
- Patients are usually asymptomatic at evaluation
- Diagnosis focused on physiologic states that could cause loss of consciousness
- No gold standard (***avoid shotgun approach !***)

Initial evaluation

Initial evaluation should answer 3 key questions;

Is it a syncopal episode or not?

Has the etiological diagnosis been determined?

Are there data suggestive of a high risk of cardiovascular events or death?

The EKG

- Guidelines recommend EKG in the evaluation **of all patients** with syncope.
- Exception: **young healthy** patients with an **obvious cause of syncope**
- Abnormal EKG in 90% of patient with cardiac syncope
- Only 6% of patients with reflex mediated syncope have abnormal EKG.
- **Syncopal patient with negative cardiac history and normal EKG—unlikely to have a cardiac cause**

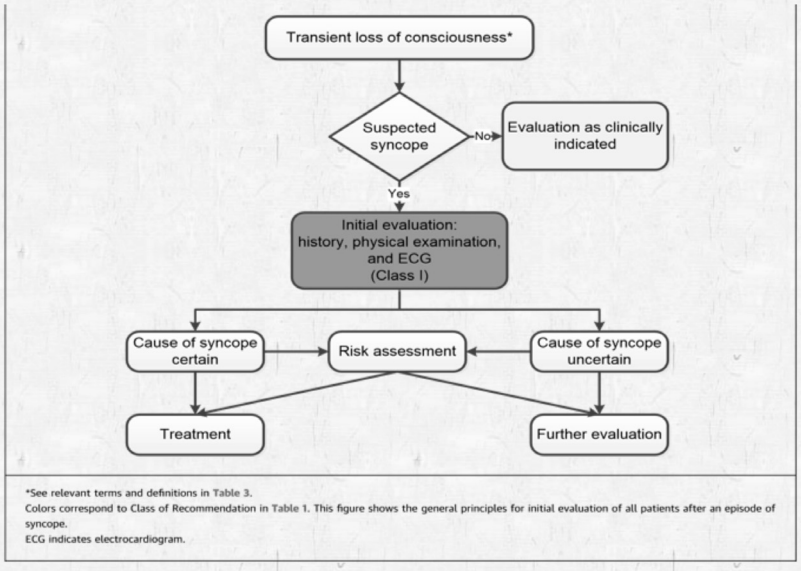
Recommendations carotid sinus massage (CSM)

- **Method**
  - Massage, 5-10 seconds
  - Don't occlude
  - Supine and upright posture (on tilt table)
- **Outcome**
  - **3 second asystole** and/or **50 mmHg fall in systolic BP with reproduction of symptoms** = Carotid Sinus Syndrome
- **Absolute contraindications**
  - **Carotid bruit**, known significant carotid arterial disease, previous CVA, MI last 3 months
- **Complications**
  - Primarily neurological
  - Less than 0.2%
  - Usually transient

Recommedations
Active standing

- **Indications:**
  - manual intermittent determination with sphygmomanometer of BP **supine** and during **active standing for 3 min** is indicated as initial evaluation when **OH** is suspected.
  - Continuous beat-to- beat non invasive pressure measurement may be useful in case of doubt.
- **Diagnostic criteria:**
  - The test is diagnostic when there is symptomatic fall in SBP from baseline value **≥ 20 mmHg or DBP ≥ 10 mmHg** or a decrease of SBP to < 90 mmHg.
  - The test should be considered diagnostic **even when there is an asymptomatic fall of** SBP from baseline value ≥ 20 mmHg or DBP ≥ 10 mmHg or a decrease of SBP to < 90 mmHg.

Initial evaluation of suspected syncope



*See relevant terms and definitions in Table 3.
Colors correspond to Class of Recommendation in Table 1. This figure shows the general principles for initial evaluation of all patients after an episode of syncope.
ECG indicates electrocardiogram.

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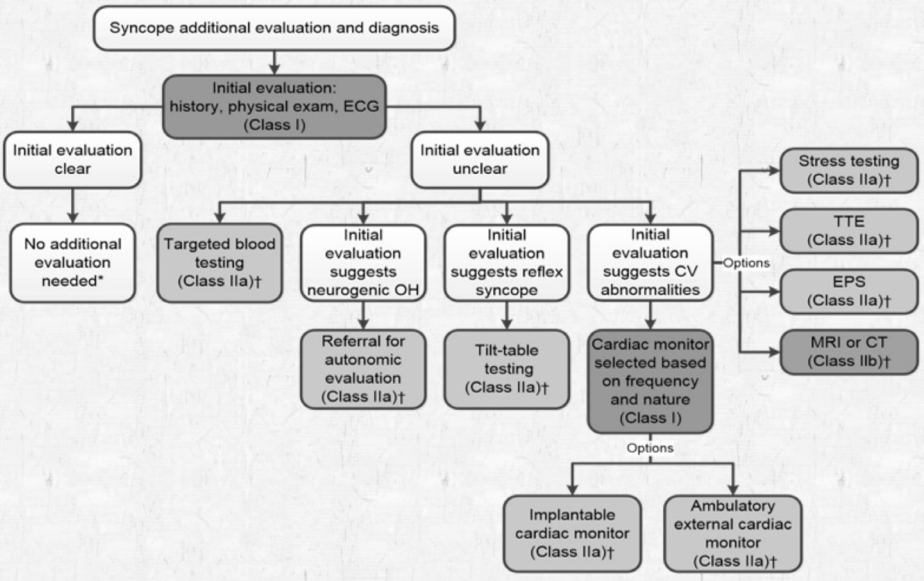
Additional evaluation & diagnosis of syncope



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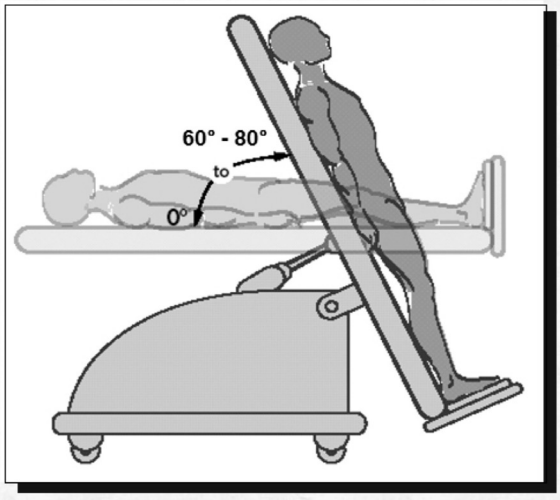
Head-Up Tilt testing

- **Indications:**
  - **Unexplained single syncopal episode in high-risk settings***
  - **Recurrent episodes in the absence of organic heart disease, after cardiac causes of syncope have been excluded.**
  - To demonstrate **susceptibility to reflex syncope.**
  - Should be considered **to discriminate between reflex and OH syncope.**
  - Maybe considered for differentiating syncope with jerking movements from epilepsy.
  - Maybe indicated for evaluating patients with recurrent unexplained falls.
  - Maybe indicated for evaluating patients with frequent syncope and psychiatric disease.
  - Isoproterenol tilt testing is contraindicated in patients with ischemic heart disease.

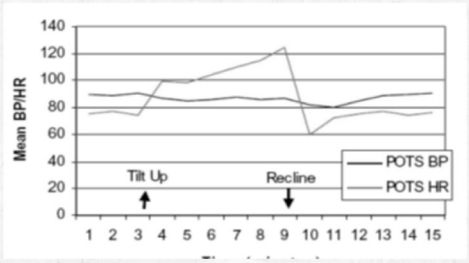
VVS Diagnosis

- History and physical exam, EKG and BP
- Head-Up Tilt (HUT) – Protocol:
  - Fast > 2 hours
  - EKG and continuous blood pressure, supine, and upright
  - Tilt to 70˚, 20 minutes
  - Isoproterenol/Nitroglycerin if necessary
  - End point
    – Loss of consciousness

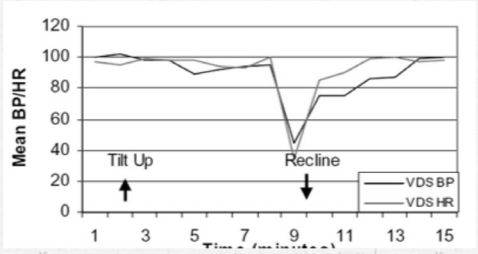


Benditt D, et al. *JACC*. 1996;28:263-275.
Brignole M, et al. *Europace*, 2004;6:467-537.

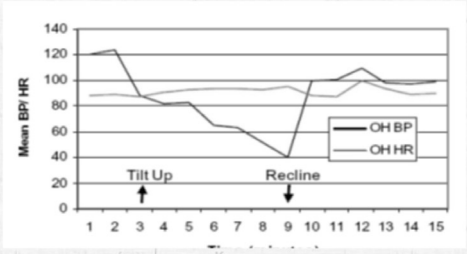
Tilt Table Test in Orthostatic Syndrome



POTS

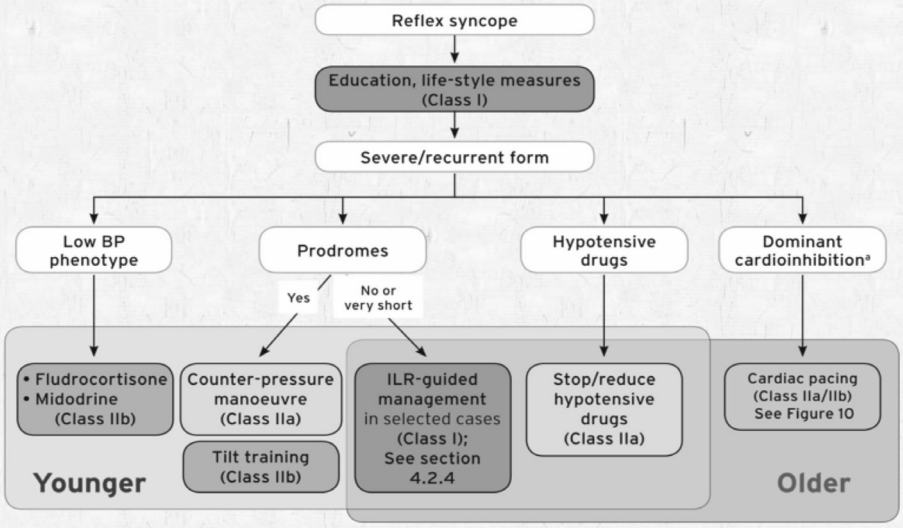


Vasovagal syncope



Orthostatic Hypotension

Management of reflex syncope



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Treatment of Syncope

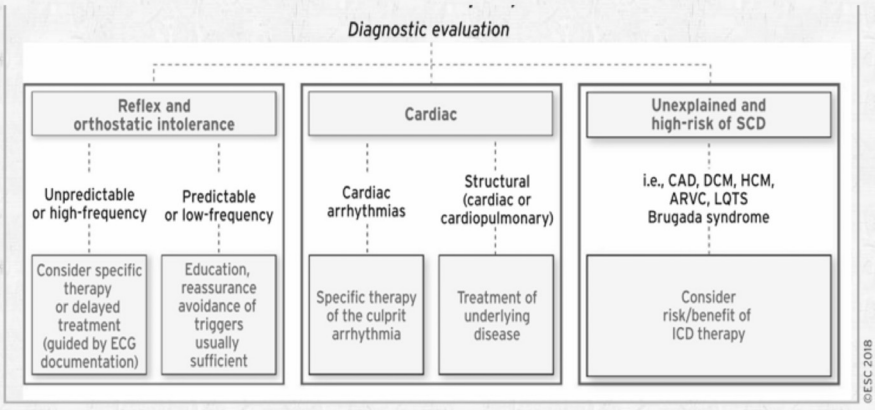

Figure 8 General framework of treatment is based on risk stratification and the identification of specific mechanisms when possible. ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; DCM = dilated cardiomyopathy; ECG = electrocardiographic; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter defibrillator; LQTS = long QT syndrome; SCD = sudden cardiac death.

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Management of OH

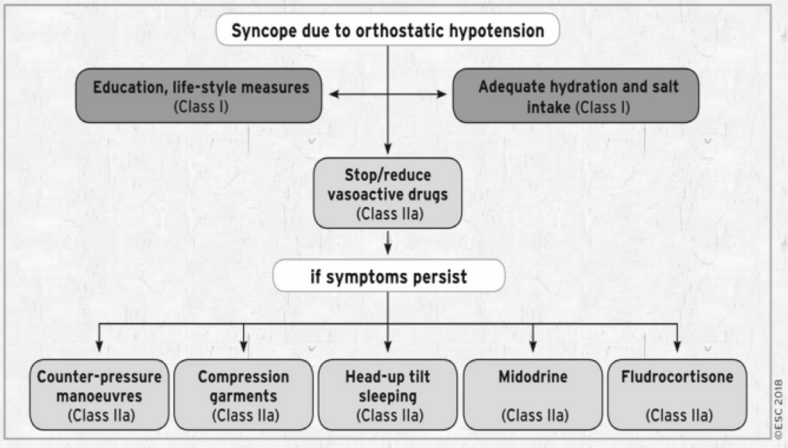

Figure 12 Schematic practical guide for the treatment of orthostatic hypotension.

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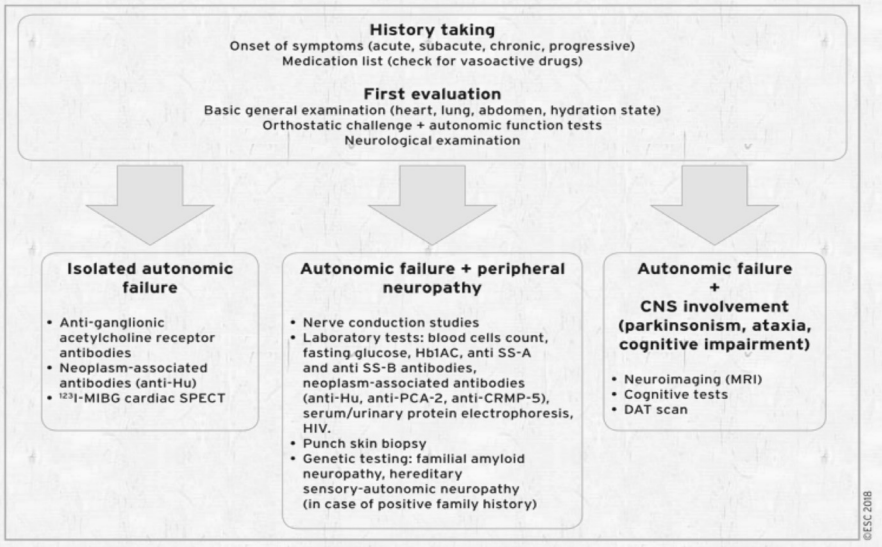
Diagnostic work-up of cardiovascular autonomic failure



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In patients with severe forms of reflex syncope, one or more of the following additional specific treatments according to the clinical features may be selected:

- Midodrine or fludrocortisone in young patients with low blood pressure phenotype.
- Counter-pressure maneuvers (including tilt training if needed) in young patients with prodromes.
- Implantable loop recorder guided management strategy in selected patients without or with short prodromes.
- Discontinuation/reduction of hypotensive therapy targeting a systolic blood pressure of 140 mm Hg in older hypertensive patients.
- Pacemaker implantation in older patients with dominant cardioinhibitory forms.

In patients with OH, one or more of the following additional specific treatments may be selected according to clinical severity:

- Education regarding lifestyle maneuvers.
- Adequate hydration and salt intake.
- Discontinuation/reduction of hypotensive therapy.
- Counter-pressure maneuvers.
- Abdominal binders and/or support stockings.
- Head-up tilt sleeping.
- Midodrine or fludrocortisone.

- The diagnostic process should be reevaluated and alternative therapies considered if the above rules fail or are not applicable to an individual patient.
- Even though guidelines are based on the best available scientific evidence, treatment should always be tailored to an individual patient's need and be patient centered.

Conclusions

- Syncope is a TLOC due to decreased CBF.
- Most common causes: reflex(vasovagal), cardiac (cardiac arrhythmia), and OH.
- Patients with cardiac syncope are at increased risk of death.
- Hx. and P/E are the most important to identify the cause.
- Orthostatic BPs & EKG should be done on all pts.
- Tilt table test can diagnose vasovagal syncope.
- Holter, Echo, EST, EP considered in patients at high risk for cardiac syncope.
- Patients remain undiagnosed in about 30% of cases. – risk stratification