Syncope in the older persons
Epidemiology and Assessment

Dr Chi-Fai Ko
Dept of Medicine & Geriatrics
North Lantau Hospital
Inter-hospital Geriatrics Meeting 27 Jan 2015
Dizziness/fall/syncope syndrome

- Overlap of symptoms
- Newcastle centre: half of 65 patients referred to syncope clinic had mixed symptoms

Figure 1. Number of patients presenting with dizziness, falls or syncope and overlap in presenting symptoms.

Transient loss of consciousness (TLOC)

- Encompass all disorders characterised by transient, self-limited, non-traumatic LOC
- Syncope vs Non-syncopal episodes
Syncope

- Global cerebral hypoperfusion occurs when SBP < 50-60 mmHg at heart level

- Syncope happens if persist for > 5s

- If persist > 15s, myoclonic jerks and urinary incontinence may result

- Sudden onset, brief, loss of postural tone, spontaneous recovery

- Usually recover within 5 min, unless being kept at a erect or sitting position
Prospective evaluation and outcome of patients admitted for syncope over a 1 year period

J.-J. Blanc¹, C. L’Her¹, A. Touiza¹, B. Garo², E. L’Her² and J. Mansourati¹

Departments of ¹Cardiology and ²Emergency, Hôpital Universitaire de Brest, Brest, France

Eur Heart J 2003;23:815-20

Prospective study in Emergency Dept Jun 99 – Jun 2000

37475 attendance

454 TLOC (1.21%), mean age 57 (SD 23)

285/454 (62.8%) admitted, 169/454 (37.2%) discharged from ED direct

288/454 syncope (63%), 56/454 non-syncopal TLOC (12%), 110/454 unexplained (24%)

Causes of syncope:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>NMS</td>
<td>76%</td>
</tr>
<tr>
<td>OH</td>
<td>7%</td>
</tr>
<tr>
<td>Cardiac</td>
<td>16%</td>
</tr>
<tr>
<td>Multifactorial</td>
<td>2%</td>
</tr>
</tbody>
</table>
Causes of syncope

• Neurally mediated syncope (NMS) / Reflex syncope
  • Vasovagal
  • Situational
  • Carotid sinus hypersensitivity

• Orthostatic hypotension
  • Autonomic failure
  • Drug induced
  • Volume depletion
  • Post- prandial

• Cardiac
  • Structural
  • Arrhythmia

Adopted from ESC Guidelines for Diagnosis and Management of Syncope 2009
Reflex syncope

• Cardiovascular effector mechanisms that are normally useful in controlling the circulation become overactive
  – vasodilatation
  – bradycardia

• 2 groups of prodromal symptoms
  – Autonomic activation
  – Retinal and cerebral hypoperfusion

Bezold-Jarisch Reflex

Tilt → Venous Return → Small Ventricle → Reflex → Catechols

- ↓ BP
- ↑ Catechols
- ↓ Inotropy
- ↓ Contractility

Reflex → Vagal Afferent → Brain Stem → Vagal Efferent

- ↓ HR
- ↑ Vasodilation
- → ↓ BP Syncope

Sympathetic Withdrawal
Table 2 Sequence of symptoms and signs in prodromal phase of syncope

<table>
<thead>
<tr>
<th>Abrupt syncope with acute standstill of perfusion of the brain and retina</th>
</tr>
</thead>
<tbody>
<tr>
<td>• After approximately 6 s: darkened vision (black out), staring, ‘freeze’</td>
</tr>
<tr>
<td>• 7–13 s: fixation in the midline or upwards turning of the eyes, loss of muscle tone, loss of consciousness</td>
</tr>
<tr>
<td>• After approximately 14 s muscle jerks</td>
</tr>
</tbody>
</table>

Gradual onset syncope with autonomic activation and symptoms of hypoperfusion

Autonomic activation

• Sweating
• Facial pallor
• Nausea
• Pupillary dilatation
• Palpitations
• Yawning\(^a\)
• Hyperventilation

Symptoms of hypoperfusion

• Brain: light-headedness, unclear thinking
• Retina: blurred vision, loss of peripheral and colour vision (grey out), darkened vision (black out)
• Shoulders: coat hanger pain
• Angina pectoris
• Hypotensive TIA\(^b\)

\(^a\) Precise mechanism unclear.
\(^b\) Extremely rare, usually resulting from the combination of occlusive carotid artery disease and orthostatic hypotension.
Orthostatic hypotension

• Inability of the autonomic nervous system to adapt to the demands of stress
  – Up-right
  – Post-exercise (standing still after exercise)
  – Post-meal
• Defective vasoconstriction and excessive venous pooling
• Prodromal symptoms due to retinal and brain hypoperfusion
Non-syncopal TLOC

- Disorders with partial or complete LOC but without global cerebral hypoperfusion
  - Epilepsy
  - Metabolic disorder
  - Intoxication
  - Vertebrobasilar TIA

- Disorders without impairment of consciousness
  - Cataplexy
  - Drop attacks
  - Falls
  - Functional (psychogenic pseudosyncope)
  - TIA of carotid origin

  ESC Guidelines for the diagnosis and management of syncope 2009
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.

Soteriades et al. 2002;347:878-85

Evaluated the incidence, specific causes and prognosis among women and men participating in Framingham Heart Study from 1971-1998

7814 participants followed for average of 17 years, 822 reported syncope
Followed for all cause mortality, MI or death due to CHD, fatal or non-fatal stroke
Mean age 51.1 (SD 14.4), Range 20-96
56% of participants with syncope saw a doctor or visited a hospital for evaluation

Incidence 6.2 per 1000 person-years
10-year cumulative incidence of syncope 6%

21.6% had recurrence of syncope during the study period
Risk of recurrence is esp high among participant with cardiac syncope, HR 30 (95% CI 14.9 to 60.3)
**Figure 1.** Incidence Rates of Syncope According to Age and Sex.

The incidence rates of syncope per 1000 person-years of follow-up increased with age among both men and women. The increase in the incidence rate was steeper starting at the age of 70 years. Syncope rates were similar among men and women.
Fig 1 Causes of syncope by age.

- Cardiac structural disease
- Orthostatic hypotension
- Arrhythmia
- Neurally mediated syncope

Cases with syncope (%)

Age group (years)

<40
40-60
>60

Parry S W, Tan M P BMJ 2010;340:bmj.c880

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### Table 1. Causes of Syncope According to Sex and the Presence or Absence of Cardiovascular Disease at Base Line.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cardiovascular Disease Absent (N=599)</th>
<th>Cardiovascular Disease Present (N=223)</th>
<th>Total Sample (N=822)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>6.5</td>
<td>3.8</td>
<td>26.7</td>
</tr>
<tr>
<td>Unknown*</td>
<td>31.0</td>
<td>41.7</td>
<td>31.0</td>
</tr>
<tr>
<td>Stroke or transient ischemic attack</td>
<td>1.7</td>
<td>2.5</td>
<td>9.5</td>
</tr>
<tr>
<td>Seizure</td>
<td>7.3</td>
<td>3.3</td>
<td>6.9</td>
</tr>
<tr>
<td>Vasovagal</td>
<td>24.1</td>
<td>24.5</td>
<td>11.2</td>
</tr>
<tr>
<td>Orthostatic</td>
<td>9.5</td>
<td>10.9</td>
<td>6.9</td>
</tr>
<tr>
<td>Medication</td>
<td>7.3</td>
<td>6.5</td>
<td>4.3</td>
</tr>
<tr>
<td>Other†</td>
<td>13.0</td>
<td>6.8</td>
<td>3.5</td>
</tr>
</tbody>
</table>

*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.
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>
<thead>
<tr>
<th>Cause of Syncope</th>
<th>Hazard Ratio (95% Confidence Interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adjusted for Age and Sex</td>
</tr>
<tr>
<td>Any cause</td>
<td></td>
</tr>
<tr>
<td>Death from any cause</td>
<td>1.43 (1.25–1.64)†</td>
</tr>
<tr>
<td>Myocardial infarction or death from coronary heart disease</td>
<td>1.47 (1.15–1.88)‡</td>
</tr>
<tr>
<td>Fatal or nonfatal stroke</td>
<td>1.19 (0.87–1.62)</td>
</tr>
<tr>
<td>Cardiac</td>
<td></td>
</tr>
<tr>
<td>Death from any cause</td>
<td>2.41 (1.78–3.26)†</td>
</tr>
<tr>
<td>Myocardial infarction or death from coronary heart disease</td>
<td>3.56 (2.29–5.55)†</td>
</tr>
<tr>
<td>Fatal or nonfatal stroke</td>
<td>2.67 (1.43–4.98)‡</td>
</tr>
<tr>
<td>Unknown</td>
<td></td>
</tr>
<tr>
<td>Death from any cause</td>
<td>1.36 (1.13–1.65)‡</td>
</tr>
<tr>
<td>Myocardial infarction or death from coronary heart disease</td>
<td>1.43 (1.00–2.03)§</td>
</tr>
<tr>
<td>Fatal or nonfatal stroke</td>
<td>0.72 (0.43–1.22)</td>
</tr>
<tr>
<td>Neurologic (including seizure)</td>
<td></td>
</tr>
<tr>
<td>Death from any cause</td>
<td>1.98 (1.45–2.72)†</td>
</tr>
<tr>
<td>Myocardial infarction or death from coronary heart disease</td>
<td>1.02 (0.48–2.17)</td>
</tr>
<tr>
<td>Fatal or nonfatal stroke</td>
<td>3.12 (1.82–5.36)†</td>
</tr>
<tr>
<td>Vasovagal or other†</td>
<td></td>
</tr>
<tr>
<td>Death from any cause</td>
<td>1.17 (0.95–1.44)</td>
</tr>
<tr>
<td>Myocardial infarction or death from coronary heart disease</td>
<td>1.16 (0.80–1.68)</td>
</tr>
<tr>
<td>Fatal or nonfatal stroke</td>
<td>0.93 (0.57–1.52)</td>
</tr>
</tbody>
</table>

*The values are adjusted for age, sex, smoking status, presence or absence of hypertension, systolic blood pressure, presence or absence of diabetes, total cholesterol level, heart rate, reported use or nonuse of cardiac medications (including antihypertensive medications), and presence or absence of a history of cardiovascular disease (myocardial infarction, coronary heart disease, stroke, congestive heart failure, atrial fibrillation, and intermittent claudication).

†P<0.001.
‡P<0.01.
§P<0.05.
†This category includes vasovagal, orthostatic, medication-induced, and other, infrequent causes of syncope.
Assessment of syncope

• Benign vs Sinister (non-cardiac vs cardiac)

• Identify the underlying cause

• The need for hospitalisation
Cardiac syncope has poorer prognosis

• Cardiac syncope has a significantly higher all cause mortality, myocardial infarct, death due to CHD, and stroke

• Syncope of cardiac cause carries a much higher recurrence rate, HR 30

• Is it a way to identify syncope of cardiac cause?
Patients with syncopal episodes referred to Syncope Clinic
Jan – July 1999
Age 18 and above

Total 341 patients
Mean age 61 (SD 20)

Presence of suspected or certain heart disease is determined by history, physical exam and ECG abnormalities
Cardiac cause identified in 23% of patients, NMS (incl OH) 58%, unexplained 18%

The presence of suspected or certain heart disease was the only independent predictor of a cardiac cause of syncope, OR 16 (95% C.I. 5 to 48), with a sensitivity of 95% and specificity of 55%.

The absence of heart disease excluded cardiac cause of syncope in 97% of the patients

Among patients without heart disease, palpitation before LOC was the only independent predictor of cardiac cause.
Conclusion

• The presence of suspected or certain heart disease after the initial evaluation is a predictor of a cardiac cause of syncope.

• When heart disease is absent, a cardiac cause is unlikely, unless palpitations precede syncope.
Identifying the underlying cause

Transient loss of consciousness

Initial evaluation

- History, physical examination, supine & upright BP, standrad ECG

- Syncope

- Non-syncopal attack

Certain diagnosis

- Cardiac likely
  - Cardiac tests
    - +
    - -

- Neurally-mediated or orthostatic likely
  - Neurally-mediated tests
    - +
    - -

Suspected diagnosis

- Frequent or severe episodes
  - Neurally-mediated tests
    - +
    - -

- Single/rare episodes
  - No further evaluation

Unexplained syncope

Confirm with specific test or specialist's consultancy

Treatment

Re-appraisal

<table>
<thead>
<tr>
<th>Cause</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurally mediated syncope</td>
<td>• Absence of cardiac disease</td>
</tr>
<tr>
<td>• Vasovagal</td>
<td>• Long history of recurrent syncope</td>
</tr>
<tr>
<td>• Carotid sinus syncope</td>
<td>• After sudden or unexpected sight, sound, smell or pain</td>
</tr>
<tr>
<td>• Situational syncope</td>
<td>• Prolonged standing in hot, crowded places</td>
</tr>
<tr>
<td></td>
<td>• Syncope associated with nausea and vomiting</td>
</tr>
<tr>
<td></td>
<td>• During or immediately following a meal</td>
</tr>
<tr>
<td></td>
<td>• With head-rotation, pressure on carotid sinus (as in tumours, shaving, tight collars)</td>
</tr>
<tr>
<td></td>
<td>• After exertion</td>
</tr>
<tr>
<td>Syncope due to orthostatic hypotension</td>
<td>• On standing</td>
</tr>
<tr>
<td>• Autonomic failure</td>
<td>• Temporal relationship with start of medication or changes of dosage</td>
</tr>
<tr>
<td>• Drug- and alcohol-induced orthostatic syncope</td>
<td>• Prolonged standing, especially in hot, crowded places</td>
</tr>
<tr>
<td>• Volume depletion</td>
<td>• Presence of autonomic neuropathy or Parkinsonism</td>
</tr>
<tr>
<td></td>
<td>• After exertion</td>
</tr>
<tr>
<td>Cardiac syncope</td>
<td>• Presence of structural heart disease</td>
</tr>
<tr>
<td>• Sinus node dysfunction</td>
<td>• During exertion or when supine</td>
</tr>
<tr>
<td>• Atrioventricular conduction system disease</td>
<td>• Preceded by palpitation</td>
</tr>
<tr>
<td>• Paroxysmal supraventricular and ventricular arrhythmias</td>
<td>• Family history of sudden death</td>
</tr>
<tr>
<td>• Inherited syndromes (e.g. long QT syndrome, Brugada syndrome)</td>
<td></td>
</tr>
<tr>
<td>• Implanted device malfunction</td>
<td></td>
</tr>
<tr>
<td>• Drug-induced pro-arrhythmias</td>
<td></td>
</tr>
<tr>
<td>Cerebrovascular syncope</td>
<td>• Arm exercise</td>
</tr>
<tr>
<td>• Vascular steal syndromes</td>
<td>• Differences in blood pressure or pulse in both arms</td>
</tr>
</tbody>
</table>

Ageing effect on the symptomatology of syncope

Among 458 consecutive patients with unexplained syncope, the clinical features of cardiac and reflex syncope were very similar in patients > 65.

The diagnosis of the cause of syncope was possible on the basis of the history alone in 26% younger and 5% older patients (p <0.0001)

Myoclonic movements, effort syncope, and supine position during LOC were the most specific diagnostic features for a cardiac cause of syncope in older patients

Among 1606 consecutive patients with tilt +ve VVS, older patients were less likely to give a typical history and therefore clinicians need to have a high index of suspicion when evaluating the older patients presenting with collapse or unexplained falls

Less likely to report total LOC, near LOC, or palpitations and more likely to present with unexplained falls. The typical provoking factor of prolonged standing, posture change and hot environments were also less common in older patients
Witness account is important

Parry et al studied the prevalence of retrograde amnesia in patients presented with syncope or fall with CICSH as the sole cause of symptom

Consecutive patient age > 60 referred to Falls and Syncope service in Newcastle

34 patients with recurrent syncope with no history of falls
34 patients with unexplained falls with no syncope

Apply CSM to all

<table>
<thead>
<tr>
<th></th>
<th>Fall group [34]</th>
<th>Syncope group [34]</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOC during CSM</td>
<td>22 (64%)</td>
<td>15 (44%)</td>
</tr>
<tr>
<td>Amnesia for LOC</td>
<td>21 (95%)</td>
<td>4 (27%)</td>
</tr>
</tbody>
</table>

Parry et al. JACC 2005;45:1840-3
High Diagnostic Yield and Accuracy of History, Physical Examination, and ECG in Patients with Transient Loss of Consciousness in FAST: The Fainting Assessment Study


504 presented with TLOC to Academic Medical Centre Amsterdam, mean age 53 (SD 19)
Assess the yield and accuracy of initial evaluation
After evaluation, divided into 3 gps
Certain diagnosis, highly likely (>80% certain) diagnosis, no diagnosis
Proceed to further work-up as appropriate and followed for 2 years
Expert committee to determine the final diagnosis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Yield</th>
<th>accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Certain</td>
<td>24%</td>
<td>93%</td>
</tr>
<tr>
<td>Certain + highly likely</td>
<td>63%</td>
<td>88%</td>
</tr>
</tbody>
</table>
UK study

Two months after adopting a standardized diagnostic protocol, a prospective outcome analysis of all patients (421) presenting with syncope for 12 month period 11/2000-10/2001

Compared with a retrospective study of all patients (660) presenting with syncope during 1998

Diagnostic yield before the use of protocol 71% vs 78% (the yield after), p = 0.003

The cost of investigation and hospital stay per patient and cost per diagnosis rose significantly after implementing the protocol.
### Table 8  Risk stratification at initial evaluation in prospective population studies including a validation cohort

<table>
<thead>
<tr>
<th>Study</th>
<th>Risk factors</th>
<th>Score</th>
<th>Endpoints</th>
<th>Results (validation cohort)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>S. Francisco Syncope Rule</strong></td>
<td>Abnormal ECG; Congestive heart failure; Shortness of breath; Haematocrit &lt; 30%; Systolic blood pressure &lt; 90 mmHg</td>
<td>No risk = 0 item; Risk = ≥1 item</td>
<td>Serious events at 7 days</td>
<td>98% sensitive and 56% specific</td>
</tr>
<tr>
<td><strong>Martin et al.</strong></td>
<td>Abnormal ECG; History of ventricular arrhythmia; History of congestive heart failure; Age &gt; 45 years</td>
<td>0 to 4 (1 point each item)</td>
<td>1-year severe arrhythmias or arrhythmic death</td>
<td>0% score 0; 5% score 1; 16% score 2; 27% score 3 or 4</td>
</tr>
<tr>
<td><strong>OESIL score</strong></td>
<td>Abnormal ECG; History of cardiovascular disease; Lack of prodrome; Age &gt; 65 years</td>
<td>0 to 4 (1 point each item)</td>
<td>1-year total mortality</td>
<td>0% score 0; 0.6% score 1; 14% score 2; 29% score 3; 53% score 4</td>
</tr>
<tr>
<td><strong>EGSYS score</strong></td>
<td>Palpitations before syncope (+4); Abnormal ECG and/or heart disease (+3); Syncope during effort (+3); Syncope while supine (+2); Autonomic prodrome* (-1); Predisposing and/or precipitating factors† (-1)</td>
<td>Sum of + and - points</td>
<td>2-year total mortality</td>
<td>2% score &lt; 3; 21% score ≥ 3</td>
</tr>
</tbody>
</table>

This table shows several different studies that have analysed the impact of different clinical data on the follow-up of patients presenting with syncope. Overall, the presence of abnormal ECG, increased age, or data suggestive of heart disease imply a worse prognosis at 1–2 year follow-up.

*Nausea/vomiting
†Warm-crowded place/ prolonged orthostasis/fear–pain–emotion.
ECG = electrocardiogram

ESC Guidelines on diagnosis and management of syncope 2009
Individual patient data meta-analysis

Current prediction tools did not show better sensitivity, specificity, or prognostic yield compared with clinical judgment in predicting short-term serious outcome after syncope
Diagnostic tools used in the algorithm

Cardiac tests
- 24-hour ambulatory ECG monitor (Holter monitor)
- echocardiography
- exercise test
- loop recorder
- EPS

Neurally mediated tests
- head-up tilt test (HUTT)
- carotid sinus massage (CSM)
- 24-hour ambulatory BP monitor (ABPM)
- autonomic function tests
Holter Monitoring in Syncope: Diagnostic Yield in Octogenarians

Michael Kühne, MD, Beat Schaer, MD, Christian Sticherling, MD, and Stefan Osswald, MD

Retrospective study done in Swiss
Patients aged 80 and over, presented with syncope and Holter done, individuals not included if no further diagnostic exam was required after the initial evaluation according to current guidelines)

475 Holter identified over 10-year period

53/475 (11.2%) had a diagnostic Holter study (correlation between symptoms and arrhythmia)
[5.8% in 522 control subjects age <80, p=0.003]

26/475 (5.4%) had an abnormal Holter result (e.g, asymptomatic non-sustained VT)

46/475 (9.7%) excluded arrhythmia as a cause of syncope (reported syncope or pre-syncope without significant arrhythmia)

Higher diagnostic yield (~20%) in 3 subgroups by multivariate analysis:

  - male OR 2.07 (1.14 to 3.77)
  - aged ≥ 90 OR 2.42 (1.16 to 5.08)
  - structural heart dis OR 3.18 (1.66 to 6.09)

91/422 (22%) had Holter repeated within 1 year of index Holter : none were diagnostic of syncope
A Prospective Randomized Comparison of Loop Recorders versus Holter Monitors in Patients with Syncope or Presyncope

Sivakumaran et al. AJM 2003;115:1-5

Canadian
100 patients with syncope or pre-syncope
divided into 2 groups

One gp received holter x 48 hrs
Another gp received ext LR x 1/12
Then cross-over

<table>
<thead>
<tr>
<th></th>
<th>confirm</th>
<th>exclude</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>LR</td>
<td>1/49</td>
<td>30/49</td>
<td>31/49 (63%)</td>
</tr>
<tr>
<td>Holter</td>
<td>0/51</td>
<td>12/51</td>
<td>12/51 (24%)</td>
</tr>
</tbody>
</table>

LR => Holter
Holter => LR

Overall diagnostic yield for Holter 22% vs
56% of external LR, p < 0.0001
The role of HUTT in diagnosis of syncope

Gold standard in diagnosing vasovagal syncope
Developed by Prof Rose Anne Kenny in Westminster Hospital, London (1719-1992)

Westminster Protocol (passive tilt x 45min at 60-70°)

Italian Protocol (passive tilt x 20min, followed by TNG spray and further 15min tilt)

specificity 97% in elderly

More sensitive in picking up orthostatic hypotension

In a group of 326 participant aged > 65
mean duration of SBP & DBP reduction are 23s and 17s respectively
Sit to stand (17.2%) vs HUT (58.6%)

Able to pick up delayed OH

Help to diagnose psychogenic pseudosyncope
The role of CSM and CSH in diagnosis of syncope

CSH has been thought to be a common cause for symptom for patients presented with dizziness, falls and syncope (25-45%). And CSM has been recommended to be done in all syncopal patient aged > 40

McIntosh et al. Age Ageing 1993;22:53-58

Later study showed that CSH is present in 39% of asymptomatic people aged > 65


Pacing of CI-CSH in patients with recurrent unexplained falls had no significant effect on the number of subsequent falls

Parry et al. Heart 2009;95:405-9

CSH may be a age-related physical finding and co-incidental with syncope