THE NORTHERN HOSPITAL

SYNCOPE

"Napoleon's Farewell to Josephine", (Alternative title: "My Destiny and France Demand It") Oil on canvas, private collection, Laslett John Pott, (1837-1898)

"...Napoleon finally worked up the courage to tell his wife that he was abandoning her. It was a terrible scene. According to the comte de Baussett, the prefect in attendance, her piercing cries could be heard throughout the palace. When Baussett arrived Josephine was collapsed on the floor. The two of them - Napoleon carrying a candle, Baussett tripping over his ceremonial sword – half carried, half dragged the fainting woman to her apartments. But Josephine was not as desperately overcome, as Baussett first feared. On the way to her chambers she whispered to him, "You are holding me too tightly"

Andrea Stuart, The Rose of Martinique.

The most important aspect to the assessment of any patient who has suffered a "syncopal" episode is to distinguish a serious cause from a benign one...such as Josephine's performance for the comte Baussett and Napoleon.

SYNCOPE

Introduction

Syncope may be defined as **transient loss of consciousness** due to **cerebral hypoperfusion**, characterized by a **rapid onset, short duration**, and **spontaneous complete recovery**.

The term "**presyncope**" is sometimes used for collapse without loss of consciousness. It is used to indicate symptoms and signs that occur **before unconsciousness** in syncope. It is a state therefore that may resemble the **prodrome** of syncope, but which is not followed by a loss of consciousness.

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

LOC due to *trauma* is a different entity and so *not* included in a discussion of true "syncope".

This syncope definition rests on **pathophysiology** (i.e. cerebral hypoperfusion) because no set of clinical features encompasses **all** forms of "syncope" while also excluding all **epileptic** seizures and **psychogenic** TLOC events.

This strict definition, however, is not useful in the ED setting where patients present with "collapse" and assessment must involve a consideration not only of syncope in the pure sense, but also of other potentially serious conditions that may result in collapse.

The most important decision to be made in the ED concerning patients who present with "collapse" is distinguishing a benign physiological response (such as a simple "faint" or "vasovagal reaction") from a more serious, or even life-threatening pathological condition.

In many cases a clear / definitive diagnosis will not be possible in the ED and so it will be important to "risk stratify" patients when planning work-up and disposition.

The commonest causes of **collapse** include:

1. Vasovagal
2. Cardiac, (arrhythmias)
3. Seizure
4. Drug related.
5. Hypovolemia (from any cause)

See also separate documents on:

- **Vasovagal (in Clinical Presentations folder)**
- **Postural Orthostatic Tachycardia Syndrome (POTS) (in CVS folder)**
- **Postprandial Hypotension (in CVS folder)**
- **Orthostatic Hypotension (in CVS folder)**
- **Carotid hypersensitivity syndromes (in CVS folder)**

Pathophysiology

Causes of collapse (which include true "syncope") include:

1. Vagal responses:
    - Emotional responses, fear, pain, shock (in the *lay* sense of the term)
    - Carotid hypersensitivity syndromes
2. Reduced venous return:
    - Valsalva related:
        - ♥ Micturition, cough, heavy lifting or straining of any cause in general.
    - Vasovagal reactions
        - ♥ Venous pooling.
        - ♥ Abrupt standing
3. Hypovolemia, of **any cause.**
4. Seizure
5. Cardiac

- Arrhythmia, (so called “Stokes-Adams” attacks)
    - ♥ As a *general* rule arrhythmias (or conduction delays) that result in collapse will depend on the ventricular rate, usually **< 40** or **> 150.**
- Acute coronary syndrome.
- Acute aortic dissection
- Outflow obstruction:
    - ♥ Aortic stenosis
    - ♥ HCM

6. Respiratory:

- Pulmonary embolism:

  Larger size pulmonary embolism is a recognized cause of syncope.

  These will usually display other signs of pulmonary embolism, such as **dyspnea, tachycardia, chest pain, hypotension** or **reduced oxygen saturation** or signs of a **DVT.**

  It is unclear whether **smaller** PEs (which would not result in hemodynamic obstruction) can be a cause of syncope. Postulated mechanisms have included various vasodepressor or cardio-inhibitory mechanisms or arrhythmias, but this is currently unknown.

  An *aggressive* search for PE in **all** patients with syncope is likely to pick up many small incidental and clinically unimportant cases of PE. [2]

  There is insufficient current evidence to suggest that *routine* investigation is necessary for PE in patients with first episode syncope *without signs or symptoms* of PE or DVT and /or *significant risk factors for PE*, or who otherwise to do not warrant admission to hospital.

7. Drug related, in particular:

- Diuretics
- Beta blockers
- Calcium channel blockers
- Any agent in general capable of causing hypotension.

8. Autonomic dysfunction/ neuropathies:

- Neuropathies, including:
    - ♥ Diabetes mellitus.
    - ♥ Postural orthostatic tachycardia syndrome (**POTS**) - (not to be confused with Pott's Disease which is *tuberculosis of the spine*).
    - ♥ Neurodegenerative conditions:

      Examples include:

        - ♥♥ Parkinson's disease.
        - ♥♥ Dementia with Lewy bodies
    - ♥ Paraneoplastic autonomic neuropathies
- Addison's disease
- Neurocardiogenic syndrome:

  This rare condition is due to an abnormal mechanoreceptor response to assuming an upright posture.

  It occurs within 15 minutes of assuming an upright posture.

  There is a paradoxical inhibition of sympathetic activity and enhancement of parasympathetic activity. This results in an abnormal bradycardic and hypotensive response.

  Specialized "tilt table" testing is required to make this diagnosis.
- Postprandial hypotension.

9. Cerebrovascular:

- TIA:
    - ♥ Syncope due to TIA is **uncommon**

      When it occurs it is usually due to **vertebrobasilar insufficiency** (with ischemia of the reticular activating system of the brainstem).

      Other brainstem features are needed to make the diagnosis with any confidence. These include vomiting, vertigo, visual disturbances and ataxia.
- SAH

- Subclavian steel syndrome
- Colloid cyst causing acute obstruction of the foramen of Monro (uncommon)

10. Hyperventilation

11. Hypoglycemia:

    - Hypoglycemia is a common cause of confusion and coma, but an unusual cause for syncopal symptoms.

12. Hysterical, (i.e. conversion reaction or psychogenic).

Clinical Assessment

Important points of history:

An accurate history from a *reliable witness* is the most critical aspect of assessing the patient who presents with syncope.

In the majority of cases the witness (if any) will not be present in the ED and phone contact will need to be made to gain a more reliable first hand account of the event.

1. Asses the features of the syncopal episode itself:

   Presyncopal features:

   - Complex auras are suggestive of seizure.
   - "Vasovagal" syncopal episodes are suggested by prolonged immobility, (especially in a warm environments), or visual or auditory disturbances.
   - Straining is suggestive of "valsalva" type syncope.
   - Shortness of breath is suggestive of **hyperventilation syndrome** or **pulmonary embolism**
   - **Cardiac** or **aortic** causes are suggested by **palpitations, chest pain** or extreme suddenness (i.e without any warning) of the collapse.
   - **Headache**, (may suggest **ICH/ SAH**)
   - Consumption of a large meal in the elderly (may suggest **postprandial hypotension**).

   Syncopal features:

   - Did the patient injure himself or herself in the collapse, especially with respect to head injury?

- A seizure is suggested by tonic / tonic-clonic / or clonic movements. Lip / tongue biting and urinary incontinence are also suggestive of seizure.

- A prolonged ictal (altered conscious state) period is more suggestive of seizure.

- Skin colour changes:

  - ♥ Extreme **paleness** during the attack, especially with subsequent extreme "flushing" is suggestive of a cardiac cause.

  - ♥ **Blueness** is suggestive of seizure or a respiratory cause or cardiac cause.

Post syncopal features:

- Rapid improvement on lying flat is suggestive of vasovagal type syncope.

- A prolonged period of confusion is strongly suggestive of seizure.

- Focal neurological deficit indicates TIA /stroke, occasionally it may be due to Todd's paresis.

Further important features on history include:

2. Have any **new medications** been prescribed for the patient or has there been any recent alteration in dosages.

3. Has there been any recent volume loss:

   - Fluid loss (vomiting/ diarrhea)

   - Blood loss, (hematemesis / melena)

4. Does the patient have a past history of **seizures**?

5. Has the patient been drinking alcohol or taking drugs or does the patient have a past history of drug or alcohol abuse?

6. Does the patient have a **pacemaker** or other **cardiac device**?

7. Is the patient high risk for pulmonary embolism?

8. High risk past history, (see below)

Important points of examination:

1. Assess airway, breathing and circulation, vital signs.

2. Check glucose and pulse oximeter readings

3. Assess for the possibility of anemia or blood loss, (**including melena**)

4. Asses for signs of dehydration.

5. Check for early signs of hypovolemia by checking for any **postural blood pressure drop**, (orthostatic hypotension)

   In general terms:

   - Look for a fall in systolic pressure to less than 90 mmHg or a fall of greater than 25 mmHg, particularly with reproduction of symptoms.

     An associated reflex tachycardia is suggestive of hypovolemia, whilst a lack of tachycardic response is suggestive of autonomic dysfunction or drug effect.

   - A sustained tachycardic response to standing with symptoms, but without accompanying hypotension may indicated **postural orthostatic tachycardia syndrome (POTS)**

6. General examination, in particular for:

   - Neurological deficits

   - Irregular pulse or evidence of aortic stenosis or HCM.

Risk Stratification:

There are **no** reliable syncope algorithms or protocols for assessing risk.

There is strong consensus from several studies that currently available **risk stratification scoring systems** have **not** shown better sensitivity, specificity, or prognostic yield compared with **clinical judgment** in predicting short-term serious outcomes after syncope. Therefore, these should **not** be used in **isolation** to perform risk stratification in the ED.

Risk will be determined by the presence or absence of known high risk features. The greater the number of high risk features, the greater will be the risk of a serious underlying pathology.

Well recognized high and low risk features may be summarized as follows:

Parameter	**High Risk**	**Low Risk (without other high risk features)**
Syncopal event	Chest pain	Typical “vagal” prodrome

	Dyspnea Abdominal pain Headache Syncope during exertion or when supine Sudden onset palpitations followed by syncope *History of structural heart disease or abnormal ECG, with:* ● No warning (or very short prodrome < 10 seconds) ● Family history of sudden cardiac death at young age. ● Syncope in sitting position.	Sudden painful or emotional stimulus Prolonged standing (especially in a hot environment) Precipitated by a valsalva action Standing suddenly from a supine or sitting position. Head rotation or carotid sinus pressure.
Past history	Structural heart disease (including previous MI) Coronary artery disease Cardiac failure with low LV ejection fractions.	Absence of structural heart disease. Long past history (years) of recurrent syncope with the same characteristics of the current episode.
Examination	Hypotension < 90 mmHg Features suggestive of GIT bleed Persistent bradycardia in awake patient & in the absence of physical training. Undiagnosed systolic heart murmur	Normal examination.
Investigations	Abnormal in general (e.g. Low Hb) Abnormal ECG (see below*).	Normal including normal ECG

High Risk ECG Findings: *

Specific **high risk** findings on ECG include:

1. Acute ischaemic changes.
2. Conduction blocks:
    - Complete heart block
    - Second degree, mobitz II heart block
3. Ventricular rates < 40:
    - Slow AF (< 40)
    - Persistent sinus bradycardia (< 40)
4. Sinoatrial block / pauses > 3 seconds
    - In an awake state, and in the absence of physical training
5. New bundle branch block:
    - Including alternating left and right BBB
6. Sustained and Non-sustained VT
7. Dysfunctioning pacemaker of AICD
8. Brugada Type I pattern
9. Prolonged QTc > 460 ms.
10. History consistent with *arrhythmic syncope* **and** the following changes:
    - First degree heart block with **markedly** prolonged PR interval
    - Second degree Mobitz I block
    - Asymptomatic mild inappropriate sinus bradycardia (40 - 50)
    - Asymptomatic mild inappropriate slow AF (40 - 50)
    - Paroxysmal SVT
    - Paroxysmal AF
    - Pre-excited QRS complex.

- Atypical Brugada patterns (type II & III).
- Short QTc (< 340 ms)
- Negative T waves in right pre-cordial leads
- Epsilon waves characteristic of ARCV

Investigations

These will be largely directed by the clinical findings on history and examination and the index of clinical suspicion for any given pathology.

The following will need to be considered:

Blood tests:

The degree of investigation is tempered by the clinical presentation and the index of clinical suspicion for any given condition:

The following may be considered according to the history and examination findings:

1. FBE:
   - Especially for **anemia**
2. CRP
   - If significantly elevated without clear reason, suspect occult/ early sepsis.
3. U&Es / glucose
   - Hypokalemia or hypokalemia may suggest an arrhythmia
   - Magnesium / calcium.
4. Troponin (if ACS suspected).
5. D-dimers (if PE suspected)

Others such as blood alcohol, as clinically indicated.

ECG:

In *particular* look for:

1. Arrhythmias.
2. WPW

3. Ischemia

4. Prolonged QT

5. ARCV

6. Brugada's syndrome.

7. High grade (so high risk) conduction blocks:

    - Trifasicular heart block / complete heart block
    - Mobitz II second degree heart block
    - Alternating left and right BBB

CT brain/ CT cerebral angiogram:

Indications for CT scan / CT angiogram include:

1. First seizures.

2. *Secondary trauma* sustained during the syncopal episode.

3. Suspected TIA or stroke syndrome (in addition to CTA and CT perfusion scan).

4. Altered conscious state / confusion.

CT Pulmonary angiogram/ V/Q Scan:

When pulmonary embolism needs to be excluded.

Management

If no obvious cause has been found after clinical assessment and investigation the most critical issues relate to disposition, which will be guided by the risk factor profile of the patient and the degree of clinical suspicion for any given condition.

Disposition:

Lower threshold for admission include:

1. Syncope unwitnessed.

2. Significant risk factors, (see above).

3. Elderly; especially without good home supervision.

Suspected cardiac cause:

If the cause is thought to be **cardiac** (and syncopal symptoms were significant and / or the patient is high risk for cardiac events) then the patient should be admitted for monitoring, and cardiology review.

Note that there is no place for "elective" outpatient holter monitoring in patients with significant CVS disease or whose symptoms have resulted in **collapse** or **hypotension** where there is a high risk of **malignant arrhythmia.**

These patients must be admitted for *immediate* 24 hour monitoring either in the SSU, CCU or other telemetry units, in order to rule out an ischemic event and / or malignant arrhythmia.

High risk factors for a cardiac cause include:

- Age
- Known electrophysiological abnormalities, or previously documented malignant arrhythmias.
- Diabetes
- A newly abnormal ECG
- Elevated troponin level.
- Significant depression of ventricular function, documented on echocardiogram
- Documented IHD including past STEMI/ non-STEMI/ abnormal cardiac functional study or abnormal angiogram.

Patients with pacemakers or other cardiac devices:

There must be a high index of suspicion in these patients for arrhythmia and / or cardiac device malfunction.

Patients with pacemakers with unexplained collapse should be admitted until such time as their pacemaker can be checked.

Note that most devices can be checked for a record of significant arrhythmia over an extended period of weeks, an extremely useful additional piece of diagnostic information for these patients!

Suspected drug related cause:

These patient should be admitted for drug medication review and observation.

A Short Stay Unit (or similar) may be appropriate.

Vasovagal:

Even if the cause is considered to be "benign", admission should still be considered in **elderly patients** or those with significant co-morbidities.

This is particularly important when:

- Episodes have been recurrent.
- Significant injuries have occurred.
- There is no adequate supervision at home.

A period of observation and physiotherapy assessment for safe mobility may be appropriate.

Disposition:

High risk patients should ,clearly, be admitted for ongoing monitoring and investigation according to the index of suspicion for any given underlying pathology.

Low risk patients, without any concerning features on history, examination or investigation may be reassured and discharged from the ED, with GP review as appropriate.

Patients may be considered for discharge providing:

1. They do not have significant clinical risk factors, including:
    - CVS risk factors, (as above)
    - Initial hypotension
    - Initial history of shortness of breath
    - Witnessed seizure activity
    - History of seizures, especially when the event is un-witnessed
    - Severe co-morbidities.
2. Investigations, have been normal.
3. Observations and clinical findings are normal.
4. The patient's medications have been reviewed.
5. In the case of the elderly, the home environment is safe.

If there is any doubt, a 12 - 24 hour period of observation in a SSU is advisable

References:

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2. Paolo Prandoni et al. Prevalence of Pulmonary Embolism among Patients Hospitalized for Syncope. The Pulmonary Embolism in Syncope Italian Trial (PESIT), NEJM 2016; 375:1524 - 31.

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Dr J.Hayes
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