Barts Health NHS
NHS Trust

# STANDARD OPERATING PROCEDURE FOR PATIENTS PRESENTING WITH SYNCOPE

| TRUST CORE GUIDELINES | |
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**Summary**
**The purpose of the ED assessment of syncope is to make a firm diagnosis where possible and identify those who may have cardiac cause and refer these for further Ix/Mx. This group has a 20-30% 1 year mortality.**

**Definition**

Syncope is a symptom not a disease. It is defined as below and is one of many causes of transient loss of consciousness.

'Syncope is... defined as a transient, self-limited loss of consciousness, usually leading to falling. The onset of syncope is relatively rapid, and the subsequent recovery is spontaneous, complete, and usually prompt. The underlying mechanism is a transient global cerebral hypoperfusion.'

Transient Loss Of Consciousness

Transient Loss Of Consciousness (TLOC) has four features:

- Loss of consciousness,
- A transient
- Self-limited nature
- Not be due to an external cause (excludes concussion)

**Patients do not present with syncope, they present with TLOC. There are many causes of TLoC, including syncope, as follows:**
**Epilepsy, SAH, AAA, GI bleed, Stroke, TIA, ACS, STEMI, PE, metabolic disturbance (**hypoglycaemia, hypoxia, hypo/er capnia, hyperkalaemia induced bradyarrythmia**).**
**Concussion and toxicological causes of impaired consciousness are classified**

**separately. This SOP deals only with syncope. Other causes of TLoC should be considered in initial assessment and the relevant SOP followed.**

**Epidemiology**
Syncope accounts for 3% of ED visits and 1-6% of hospital admissions. It is a disease of all age groups occurring in around 15% of children under 16, 23 % of adults over 70 years and with a lifetime prevalence of 42%. The incidence increases with age and is recurrent in 30%. Around 75% patients are currently admitted in UK (2006).

**Pathophysiology**
All causes of syncope involve a partial or complete reduction in cerebral perfusion. Average CBF (Cerebral Blood Flow) is 15% CO and 50ml/100gm tissue /min and this is largely dependent on systemic arterial pressure. The most important determinant of CO is venous filling. Any factor interfering with filling or CO or vascular tone may impair CBR and consciousness. A 6-8 second absence of CBF or a fall in SBP to 60 mmHg or a 20% fall in oxygen delivery may result in syncope.

**Causes**

| | | |
|---|---|---|
| Vasovagal | 50-66% | (8-37%) |
| Situational | 5% | (1-8%) |
| Carotid sinus hypersensitivity | 1% | (0-4%) |
| Psychiatric | 2% | (1-7%) |
| Orthostatic | 8% | (4-10%) |
| Medication related | 3% | (1-7%) |
| Neurological | 10% | (3-32%) |
| Cardiac - Arrhythmia | 14% | (4-38%) |
| - Organic | 4% | (1-8%) |
| Unknown | 34% | (13-41%) |

**CLASSIFICATION**

**1.Reflex or Neurally Mediated Syncope**

This group is characterised by a sudden failure of autonomic tone with a fall in blood pressure and /or bradycardia. All involve an exaggerated parasympathetic nervous system tone and / or fall in sympathetic nervous system tone. These may be broadly classified into:
-Vasovagal
-Carotid sinus hypersensitivity,
-Situational syncope and neuralgic syncope.

**1a. Vasovagal Syncope**
Vasovagal is common and preferred name for neurocardiac or neurocadiogenic syncope. It is
a benign and self-limiting condition. It accounts for 50-66% of all syncope. Diagnosis is clinical but may be challenging. The key diagnostic investigation is by tilt table in these

cases.
It involves an exaggerated neurocardiac response to various stimuli. On standing around

500ml of blood immediately leaves the thorax for lower capacitance vessels. In the following 10 minutes another 700ml of fluid enters the interstitium. Blood pressure is preserved by
vasoconstriction and a small increase in pulse rate with pressure / volume changes detected in aortic and carotid baroreceptors and to a lesser extent the cardiopulmonary receptors. The exact mechanism is not yet known.
The key to diagnosis is history from the patient and a witness.
Myoclonic jerking is common – in one series of induced syncope 90% were followed by myoclonic jerks and are frequently misdiagnosed as seizure activity. Myoclonic jerks should last < 30 seconds and resolve on the patient becoming horizontal. OF note, onset is AFTER LOC . By comparison , jerking COINCIDES with altered consciousness in epilepsy.'Palpitations' are also common but are should be of gradual onset, regular and < 100bpm, as this is usually awareness of normal heart beat.
Key points from history:
Prodrome, diaphoresis prior to event, pallor, blurred vision, nausea, clear precipitant – emotional stimulus/pain/standing/missed meal, LOC is usually brief (30-120 sec and post event confusion absent or short lived (<5 mins). Post syncopal exhaustion is common.
NICE suggests remembering key points by the P's – posture, provoking event, prodrome, and pallor.

**The Elderly**
Sycnope is more difficult to evaluate in the elderly due age related changes, multiple pathology, multiple medications and cognitive impairment (20% over 80yrs). Amnesia for the LOC is common and history less reliable. These factors make the diagnosis of VVS challenging.
Post prandial hypotension accounts for up to 8% of syncopal episodes and upto 50% of syncope within 2 hours of meals.

Postprandial Hypotension

Diagnosis

In all elderly patients presenting with syncope/falls its relationship with meals should be ascertained. Sometimes it may be difficult for elderly people to remember how long before the syncope/fall they had their meal. The best way is to plot approximate timing of each of the syncope/falls against their usual times of breakfast, lunch and supper. If the Syncope/fall seem to have occurred with in 90 minutes of their usual meal times it is likely that it could be due to postprandial hypotension. This could be tested by measuring Blood pressure before the meals and then every 15 minutes after a mixed meal (carbohydrate and protein) for 90 minutes while seated. A 20mm Hg drop in systolic pressure confirms postprandial hypotension. Ambulatory blood pressure monitoring with documented meal times is an alternative investigation.
Treatment;
At present there is a lack of enough data on effective treatment.
Nonpharmacological:
Nonpharmacological interventions are advised initially and include having small sized

meals, avoiding hot, high carbohydrate meal, avoiding alcohol and antihypertensive medications around meal time, adequate fluid intake and exercising legs after meals.
Pharmacological:
Evidence for caffeine is inconclusive. Acarbose has been shown to be beneficial in one study. Use of Octreotide, a somatostatin analogue should be reserved for severe symptomatic cases. It is expensive and comes in an injectable form.

**1b. Carotid Hypersensitivity**
The syncopal episode results from an exaggerated reflex response to carotid sinus stimulation – for example putting on a scarf or tie, or shaving. There are two components – increased parasympathetic tone causing bradycardia, increased PR interval, AV block and reduced sympathetic tone resulting in vasodilation and hypotension.
Diagnosis is suggested by history and made by observing a fall of 50mmHg in systolic pressure or a 3 second ventricular pause following from 5-10 seconds of carotid sinus pressure. It is rare under 50 years and is recognised in up to 45% of elderly with syncope, falls or dizziness. Diagnostic testing is part of the ED work up and should take place in resus on all with suggestive history. If a ventricular pause is demonstrated referral for pacing be made as an inpatient.

**1c. Situational syncope**
Cough/sneeze etc stimulates local lung, GIT or GU receptors which stimulate vasodepressor centre and reduce SNS and increase PNS tone causing syncope. The diagnosis is made on history.

**1d. Neuralgic Syncope**
Pain in throat stimulates similar reflex to above via brain stem and reduces SNS but increases PNS tone. Diagnosis is from history.

**2. Orthostatic Syncope**

Postural hypotension is defined as a fall SBP by 20 mmHg or to < 90mmHg associated with

postural change. Postural syncope occurs when this is associated with TLoC. It occurs when there is volume depletion or failure of the autonomic nervous system response to maintain blood pressure by increasing sympathetic tone in response to venous pooling. Volume depletion may occur with blood loss, fluid loss or Addison's disease. Autonomic failure may occur as a primary disease (pure autonomic, multisystem atrophy, Parkinson's) or secondary (diabetes or amyloid).

However the most common cause is drug related, most commonly anti-hypertensive.
It effects around 5% of the general population and around 7-17% in the acute care setting.

It increases with age due to reduced baroreceptor sensitivity and polypharmacy.
Diagnosis is suggested by history and finding postural hypotension. 10% of patients take 2-5 minutes for diagnostic changes in BP. The patient should lie quietly for 5 mins, then stand for BP/pulse at 1 min and if preserved to be repeated with symptoms or at 5 minutes.

**3. Cardiac**

Cardiac causes of syncope may be arrythmogenic or resultant from structural heart disease. 1 year mortality is 15-35% in this group as compared to a background of 0-6%. The incidence of sudden death is 24 % in the cardiac group. However if the cases are matched for similar degrees of cardiac disease these differences are not bourne out – so it is the presence of cardiac disease not the cause of syncope, which determines prognosis.

Any tachy or brady arrhythmias may impair cardiac output. Common causes include:
Sinus node dysfunction
AV conduction failure
Supra ventricular arrhythmias
Ventricular arrhythmias
Inherited tachyarrhythmias
Brady arrhythmias
Pacemaker malfunction

In structural heart disease the heart's ability to meet demand is impaired. Examples include:
Valvular disease
Ischaemia
Obstructive cardiomyopathy
Atrial myxoma
Aortic dissection
Pericardial tamponade
Pulmonary hypotension
PE

Cardiac syncope has a 1-year mortality of 20-35% and thus it is key to identifying this group. Any patient suspected of having cardiac syncope should be monitored in ED and referred for IP or OP monitoring – see SOP flow sheet. If structural heart disease is suspected echo is the diagnostic mode of choice.

**4. Steal Syndromes**

Cerebral supply is reduced by a vessel supplying an arm and part of the brain. This is very rare and the diagnosis suggested by history and confirmed by imaging

**5. Psychiatric**

These are not really causes but associations. Many psychiatric conditions predispose to neurally mediated syncope – anxiety, panic, and depression. Rarely syncope may be the

manifestation of a somatization disorder. Psychiatric causes of TLoC are common and diagnosis is suggested by recurrent episodes, usually in young adults, accompanied by lack of serious harm and with normal examination, ECG, echo and Holter monitoring. Presentation may be bizarre, often with prolonged ictus. Psychiatric drugs also predispose to arrhythmias and orthostatic syncope.



**ED work up**

In streaming – a brief history and focussed examn, ECG, VBG, urine dip (HCG) and lying and standing BP/P – after lying flat for 5 mins take BP/P at 1 and 5 mins after standing. Patients in whom cardiac syncope is suspected should be transferred to a monitored bed. FBC U&E/glucose/troponin have low yield in syncope with only 2-3% of patients having causal findings in these basic bloods and are indicated only when there are specific clinical features. Hb is useful to identify high-risk groups. They are not required on all pts – class 1 recommendation European task force. The ECG is a simple cheap test which will identify causal factors in around 7% but will help direct further risk stratification and investigation in many more. BHCG performed in all women of childbearing age. Mild headache is very common in vaso-vagal syncope and CT head is not indicated unless there is clear suspicion of a non-synopcal cause of TLOC – such as SAH or CVA, in which case this pathway is not appropriate.
Patients with a clear history of vaso-vagal or situation or neuralgic syncope who have made a full recovery may be discharged (with appropriate advice – see below), or if still symptomatic transferred to CDU - class 1 recommendation.
Patients with? cardiac syncope or ?carotid sinus hypersensitivity are transferred to a monitored bed, the latter for diagnostic testing.
Patients with 2nd degree or complete heart block, Bifascicular or trifascicular block, dynamic ST segment changes, SVT, fast AF (>130), GCS <=13, SBP < 90-100mmHg, respiratory distress/acidosis are transferred to resus.

**Further ED work up**
Hx Ex and the above base line tests will allow the rest of the work up to be determined. The most important further work up is a complete history from the patient and a witness, and full examination. The key 3 questions for the clinician are

- is this case of TLoC syncope
- is there a clear diagnosis?
- Is this possibly cardiac syncope

In those where an underlying disease process is suggested by symptoms such as chest pain, palpitations or breathlessness or signs such as a murmur, focal neurology, or GI bleeding, suspicion will dictate investigation and urgency. However in many patients seen in the ED with syncope the aetiology of this will not be evident. In this group further investigation is

dictated by risk. This is discussed in detail below. The major risk is the presence of structural cardiac disease – whether or not this is the cause of syncope.

**? Cardiac syncope**. This group of patients all require monitoring in the ED, usually in monitored area not resus. This group is divided into high risk, requiring IP investigation

and intermediate risk that require OP echo and Holter (+/- loop recorder).



**High risk** – require IP assessment under general medicine or direct transfer to Bart's electrophysiology cardiology. Any of: an undiagnosed systolic murmur - suspected AS/HCOM, FH sudden death < 50 years old, syncope occurring whilst supine or during exercise (note syncope occurring post exercise is almost always a simple vaso-vagal), new dyspnoea, evidence of cardiac failure, chest pain and ECG evidence of any of the following: second degree or complete heart block (refer to Bart's electrophysiologists), abnormal QTc – as a guideline, but age/sex specific > 450 < 350, ventricular arrhythmia, HR < 50 with no cause evident drug/electrolyte cause (suggestive of SSS in elderly), bifascicular block, trifasicular block (refer to Bart's electrophysiologists), BBB, dynamic STT changes s/o IHD; ECG evidence of WPW, Brugada, epsilon wave or features of RV dysplasia associated with syncope should be discussed with the electrophysiologist at Bart's. Suspected pacemaker malfunction should be discussed with Bart's electrophysiologists and may be transferred to their clinic.

**Intermediate risk** – this group require senior input to determine whether IP or OP investigation is indicated. Admission is likely if any of the following are present: P< 50bpm, PR bleed, Hg < 10, chest pain, ECG – Q wave in any lead but III, SpO2 < 94%. If discharged then OP echo and 48 hour Holter is warranted. Follow up is with cardiology

**Carotid sinus hypersensitivity** is diagnosed by provocative testing. It is not performed in the presence of a bruit or known cerebrovascular disease. The patient is supine on a monitor and continuous BP measures taken. Pressure is applied lightly for 5-10 secs at the anterior margin of sternocleidomastoid at level cricoid cartilage. This is repeated on the contralateral side in case of a negative result. It may be repeated after atropine administration to look for the vasodilatory response (either a bradycardiac or vasodilatory response or both may be seen). CS hypersensitivity is defined as a fall in SBP > 50mmHg or a Ventricular pause > 3 secs – and these patients are considered for IP pacing. Unfortunately many elderly patients have an abnormal response and no syncope and by performing the study in the supine position only some cases will be missed (false negatives) – hence some clinicians repeat it with the patient upright – often on a tilt table. However those with CSH and no inducible syncope may be at risk of future syncope and ventricular pauses. The European Task force recommends CSM be carried out in those pts with unexplained syncope but be avoided in those with a risk of carotid artery disease related stroke. They further recommend it should be carried out in erect and supine positions.

**Undifferentiated patients**

This question is best answered by careful history and examination but the answer may not be apparent initially or indeed until there has been investigation and follow up. Around 30-50% of all patients fall into this category.

**Unexplained syncope**

This is a common sequelae. The key is to identify those at risk of cardiac syncope, as the most important factor in the prognosis is the presence of structural cardiac disease – even if this is not the direct cause of the syncopal episode. If the patient has heart disease then

this is an independent predictor of a cardiac cause of syncope with sensitivity of 95% and specificity of 45%. If the patient does not have evidence of heart disease (normal examination and ECG) then a cardiac cause of syncope is ruled out 97% of the time.
This group should be further evaluated in the ED on a monitored bed. IP referral is suggested by P<50 at any time, PR bleed, Hb<10 gm/d, chest pain, ECG showing new Q wave in any lead but III, SpO2 < 94% air, new dyspnoea, age > 65 (with no clear diagnosis and no prodrome) and any patient in whom significant injury has resulted from syncope. The time required for IP monitoring – 6, 12, 24, 48 hours - is not known. There is little evidence that monitoring beyond 24 hours as an IP is useful.

**Patient with evidence of structural heart disease**
In the group not having the features listed above but with a history of heart disease, pulmonary hypertension, an abnormal ECG, evidence of heart murmur, syncope with no prodrome or > 2 episodes syncope then referral for OP echo and 48 hour Holter is warranted (European Guidelines class 1 recommendation), followed by EPS, EST or loop recorded if the former are non diagnostic as judged by the cardiologist. Any patient in a high-risk profession – pilots, professional drivers, and doctors – should be referred for Holter and echo.

The utility of echo as a screen has a low yield – i.e. if there are no symptoms or exam / ECG findings to raise the question of cardiac disease the chance of a significant finding on echo are remote.

**Patients with no Probable structural heart Disease** (no history of heart disease, normal exam and ECG)
This group are managed as outpatients. The likely diagnosis is vaso-vagal syncope. A normal physical exam and ECG makes a cardiac cause of syncope unlikely – although it does not rule out some arrhythmias such as atrial tachycardias. In the patient with no known heart disease and a normal ECG the likely cause of syncope is vaso-vagal and tilt table best explores this. NICE recommends a Holter be performed first.

**Is it syncope?**

The diagnosis may not be straightforward. Common areas for confusion are as follows:
**? Epilepsy** - The commonest difficult differential diagnosis is epilepsy. Over half of all cases of syncope involve some myoclonic jerks after onset of syncope, but these are usually short-lived, often irregular. Seizure is more likely if the time of unconsciousness was over 5 minutes, the prodrome included aura, incontinence of urine occurred, there was a period of post collapse confusion, the tongue is bitten laterally (anterior tongue biting is not specific and is common in vaso-vagal), the posture included lateral head turning or repetitive symmetrical jerking, muscle pain is present and automatisms. This group are referred to either CDU for review by the epilepsy nurses or referred to first fit clinic for expert opinion. The diagnosis may be difficult and require video EEG and tilt table studies for a definitive answer. EEG should not be requested from the ED.

**? TIA** - Syncope is rarely the result of a TIA and then only ever posterior circulation TIAs

that will almost invariable be associated with other signs



**Vertigo associated collapse** - Vertigo involves a sense of motion and does not involve impaired consciousness. The latter may suggest posterior circulation TIA. The physical examination may elicit focal neurological signs, nystagmus, a positive Dix-Hallpike manoeuvre or altered hearing.

**Headache associated with TLoC**. This is a common accompaniment of vaso-vagal syncope. SAH of sufficient size to produce LOC is very unlikely to leave no abnormal findings. CT is rarely indicated in TLoC but is the initial investigation if ICH or SAH is considered.

**Clinical Pointers**
There are several clinical pointers to help guide the clinician: -

| | |
|---|---|
| Prodrome of light-headedness, diaphoresis, nausea, blurred vision, palpitations pallor, paraesthesia - all resolve when supine | vaso-vagal syncope |
| ppt fear, smell, sight, sound<br>post exercise, standing ,prolonged sitting<br>Post syncopal exhaustion | vaso-vagal syncope |
| Obvious ppt – cough, micturition | situational syncope |
| Head turning, pressure on neck (shaving) | CS hypersensitivity |
| Headache | migraine, SAH, seizure |
| Vertigo, diplopia, dysarthria | TIA, subclavian steel<br>Basilar migraine |
| Assoc arm exercise | subclavian steel |
| During exercise<br>[post exercise is vagal benign) | HCOM, pul HT, MS<br>AS, IHD |
| Confusion, LOC > 5 mins<br>Average vasovagal is < 60secs | seizure |
| BP different left/right | aortic dissection, subclavian steel |
| Ppt position change – turn over, lie down | atrial myxoma/thrombus |
| FHx +ve sudden death | Brugada, long QT, RV dysplasia |

syndromes or HCOM

No prodrome arrythmia

**Treatment**

All patients not admitted should receive a full explanation of likely diagnosis, trigger avoidance and follow up.

**Vaso-vagal**
Full education on;
1 Trigger avoidance - avoid stressors like heat, temp change, dehydration, alcohol, tight clothing.
2 Early evasive action – attempt to make yourself horizontal when prodrome occurs,
3 Physical counter manoeuvres -forearm, calf tensing.
4 Hydration – 1L till lunch time, then enough to keep urine clear.

If recurrent refer to general medical OP for ? salt/fluid advice, exercise training and medication (fludrocortisone, etilefrine, SSRI, β-Blockers, anticholinergics)
Orthostatic hypertension - hydrate if dehydrated, preferably orally, consider medication change in conjunction with GP if OP mx, consider CDU or IP treatment if risk of injury or complex medications.

**Pacing**
Class 1 – syncope with CHB, bi/trifacicular block or CSM hypersensitivity with V pause > 3 secs (no causal drugs)

**Driving**
All patient with a suspected cardiac syncope, cough syncope, seizure or TLoC with no clear diagnosis referred for further evaluation should be advised not to drive until evaluated by the specialist team

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