

## CARDIAC CHEST PAIN

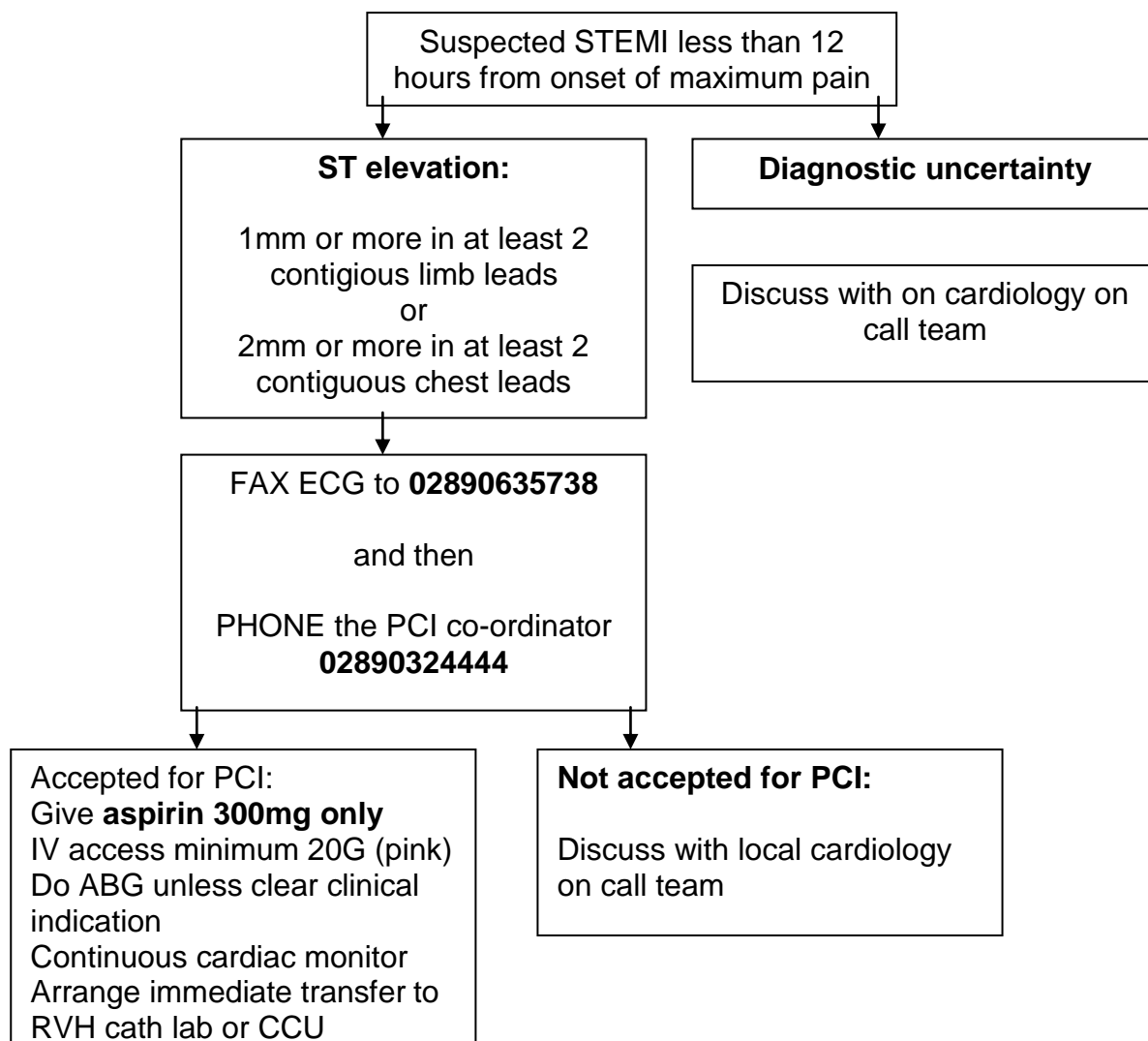
ACUTE ST ELEVATION MYOCARDIAL INFARCTION\* and ACUTE CORONARY SYNDROME (ACS) are caused by instability/rupture of atheromatous plaques in the coronary circulation. Identifying ST Elevation MI rapidly is the first goal so that definitive treatment can be achieved as early as possible to limit myocardial damage.

The ACS spectrum ranges from unstable angina (USA) to non-ST elevation myocardial infarction (NSTEMI). ACS is extremely common amongst ED patients and it is associated with high risk of cardiac arrest, peri-arrest arrhythmias, acute ST elevation myocardial infarction and acute LVF.

*\*Rarely acute MI may be caused by another cause such as cocaine abuse*

### 1. ST Elevation MI

All patients with a STEMI should be referred to RVH for consideration of primary PCI. NIAS have a bypass protocol so most patients that they attend who have a STEMI will go directly to RVH.



## **2. Non- ST Elevation MI and other Acute Coronary Syndromes**

### **History:**

Cardiac sounding chest pain  
 Atypical pain – especially females and diabetic  
 Cardiac risk factors  
 Previous MI / IHD

### **Examination:**

CVS and RS examination  
 BP in both arms and peripheral pulses if possibility of dissection  
 thoracic aorta

**Ischaemic ECG changes:** (repeat ECG in 30 mins if suggestive history but normal initial ECG)

1. LBBB = assume anterior myocardial infarction if new
2. Profound ST depression V1-3 +/- Tall R-wave in V1 = posterior myocardial infarction
3. Tall peaked T-waves with early slurring of ST segment= ?hyperacute ischaemic ECG: GET ADVICE
4. Any other ST segment depression(“NSSTTW”) = assume Acute Coronary Syndrome unless present on old ECGs

### **Management:**

All patients with suspected ACS should receive the following treatment (unless contra-indicated e.g. on warfarin).

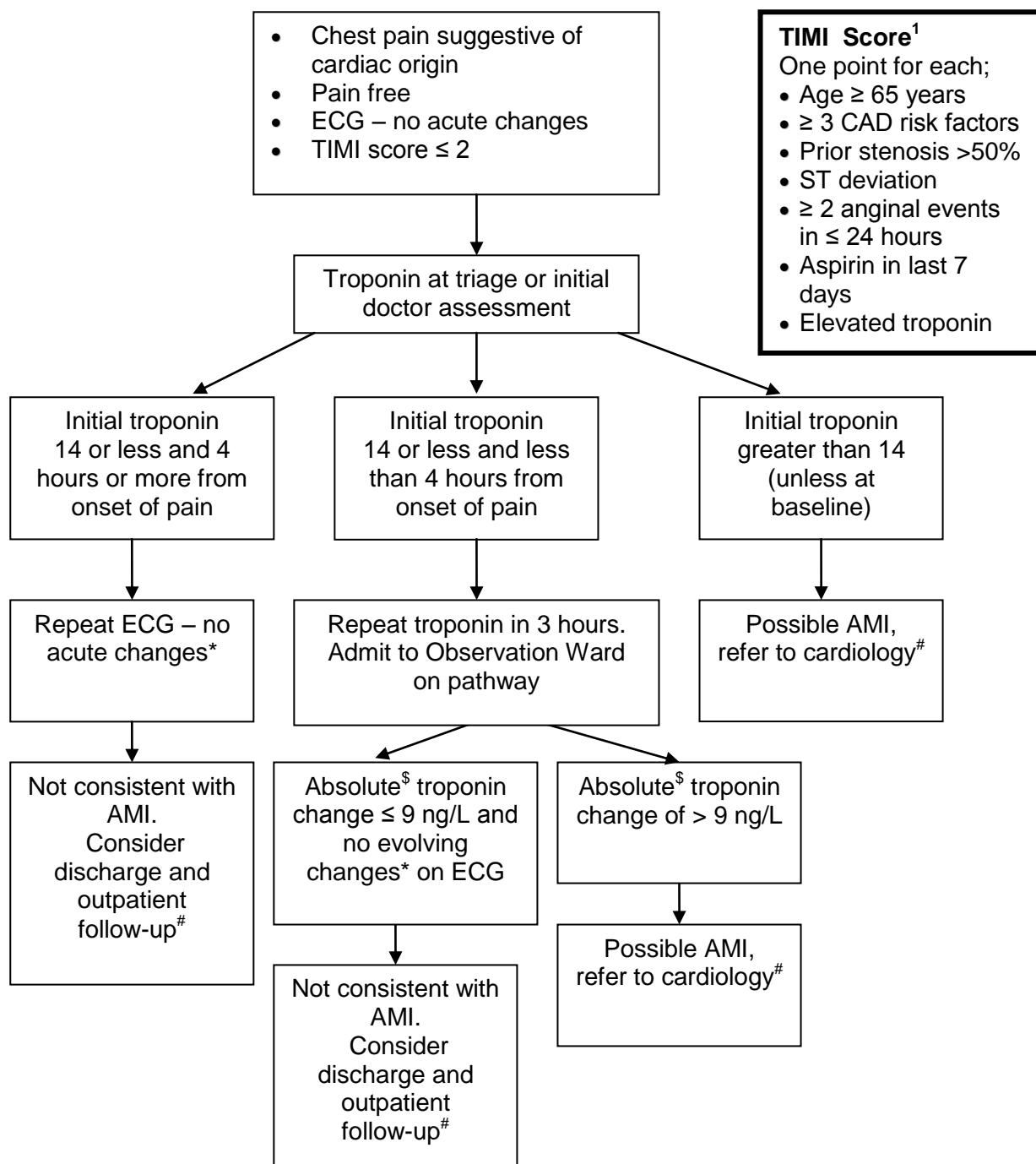
- 100 % oxygen NRRM
- Continuous ECG monitor, SaO<sub>2</sub>, NIBP
- GTN spray
- Pain relief as required e.g. diamorphine +/- metoclopramide
- Chewable Aspirin 300 mgs orally
- Enoxaparin (Clexane) 1mg/kg subcutaneously bd.
- Admit under cardiology

## **3. Low Risk Cardiac Chest Pain**

Patients with history suggestive of cardiac chest pain but who are low risk (TIMI score 2 or less) who are pain free are suitable for ED / Observation Ward management.

See flow diagram on next page for appropriate management.

**Assessment of low risk patients with cardiac sounding chest pain presenting to Antrim ED**



**References**

1. Antman EM, Cohen M, Bernink PM, et al. The TIMI Risk Score for Unstable Angina/Non–ST Elevation MI: A Method for Prognostication and Therapeutic Decision Making. *JAMA*. 2000;284(7):835-842

**Notes**

\* if acute ischaemic changes refer to cardiology

§ Absolute change equates to difference between the original and second troponin results, this may be positive or negative. Absolute change of 9 ng/L is considered clinically significant

# it is the responsibility of the discharging doctor to follow-up results and complete discharge letter

#### **4. Rapid Access Chest Pain Clinic**

The Rapid Access Chest Pain Clinic is designed to provide a 'one stop' service for patients presenting with a recent onset of chest pain thought to be stable angina or very low risk unstable angina. ED doctors on the advice of the ED consultant can refer patients directly to RACPC.

#### **5. Not all Chest Pain is Cardiac**

You will see many other types of chest pain. Common causes of chest pain include:

- Musculoskeletal pain is the commonest – take a good history!--did the patient undertake strenuous activity e.g. gardening?, do certain movements hurt? Is there a tender costo-vertebral junction suggesting an acutely subluxed rib at the back (common and self-limiting) – *but remember that ~15% of patients with acute MI have marked chest wall tenderness!*
- Upper GI – GORD, Acute Cholecystitis, Pancreatitis
- PE (use Canada Score)
- Stress, Hyperventilation
- Chest infection
- Rib fractures e.g. cough fracture
- Herpes zoster (dermatomal)

### **TRANSIENT LOSS OF CONSCIOUSNESS (TLOC)**

Significant causes of TLOC must be excluded before patients can be safely discharged home. The following is a summary of the NICE 2010 Clinical Guidelines for the Management of TLOC in adults and young people.

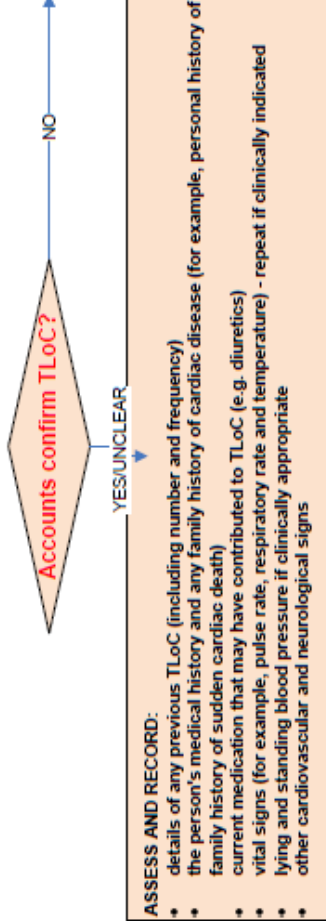
<p><b>Box A</b></p> <p>Ask the person who has had the suspected TLoC, and any witnesses, to describe what happened before, during and after the event. Try to contact by telephone witnesses who are not present. Record details about:</p> <ul style="list-style-type: none"> <li>• circumstances of the event</li> <li>• person's posture immediately before loss of consciousness</li> <li>• prodromal symptoms (such as sweating or feeling warm/hot) appearance (for example, whether eyes were open or shut) and colour of the person during the event</li> <li>• presence or absence of movement during the event (for example, limb-jerking and its duration)</li> <li>• any tongue-biting (record whether the side or the tip of the tongue was bitten)</li> <li>• injury occurring during the event (record site and severity)</li> <li>• duration of the event (onset to regaining consciousness)</li> <li>• presence or absence of confusion during the recovery period</li> <li>• weakness down one side during the recovery period.</li> </ul>	<p><b>Box B</b></p> <p>If an automated interpretation is not available, the unreported 12-lead ECG should be reviewed by a healthcare professional trained and competent in identifying the following abnormalities.</p> <ul style="list-style-type: none"> <li>• Inappropriate persistent bradycardia.</li> <li>• Any ventricular arrhythmia (including ventricular ectopic beats).</li> <li>• Long QT (corrected QT &gt; 450 ms) and short QT (corrected QT &lt; 350 ms) intervals.</li> <li>• Brugada syndrome.</li> <li>• Ventricular pre-excitation (part of Wolff-Parkinson-White syndrome).</li> <li>• Left or right ventricular hypertrophy.</li> <li>• Abnormal T wave inversion.</li> <li>• Pathological Q waves.</li> <li>• Atrial arrhythmia (sustained).</li> <li>• Paired rhythm.</li> </ul>	<p><b>Box C</b></p> <p>ECG abnormality (as specified in Box B)</p> <ul style="list-style-type: none"> <li>• Heart failure (history or physical signs)</li> <li>• TLoC during exertion</li> <li>• Family history of sudden cardiac death under 40 years and/or inherited cardiac condition</li> <li>• New or unexplained breathlessness</li> <li>• Heart murmur</li> </ul> <p>Consider referring within 24 hours for cardiovascular assessment, as above, anyone aged older than 65 years who has experienced TLoC without prodromal symptoms.</p>	<p><b>Box D</b></p> <p>Make a diagnosis of <b>uncomplicated faint</b> when:</p> <ul style="list-style-type: none"> <li>• There are no features that suggest an alternative diagnosis.....AND</li> <li>• there are features suggestive of uncomplicated faint such as:             <ul style="list-style-type: none"> <li>• Posture - prolonged standing or similar episodes which have been prevented by lying down.</li> <li>• Provoking factors (such as pain or a medical procedure).</li> <li>• Prodromal symptoms (such as sweating or feeling warm/not before TLoC).</li> </ul> </li> </ul> <p>Make a diagnosis of <b>situational syncope</b> when:</p> <ul style="list-style-type: none"> <li>• there are no features from the initial assessment that suggest an alternative diagnosis.....AND</li> <li>• syncope is clearly and consistently provoked by straining during micturition (usually while standing) or by coughing or swallowing.</li> </ul>
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Use clinical judgement to determine appropriate management and the urgency of treatment if there is:

- a condition that requires immediate action
- the person has sustained an injury as a result of TLoC or
- they have not made a full recovery of consciousness

Take patient and witness account of the suspected TLoC [box A]  
 Include paramedic records in your information gathering

Manage according to non-TLoC presentation



**12 LEAD ECG:**

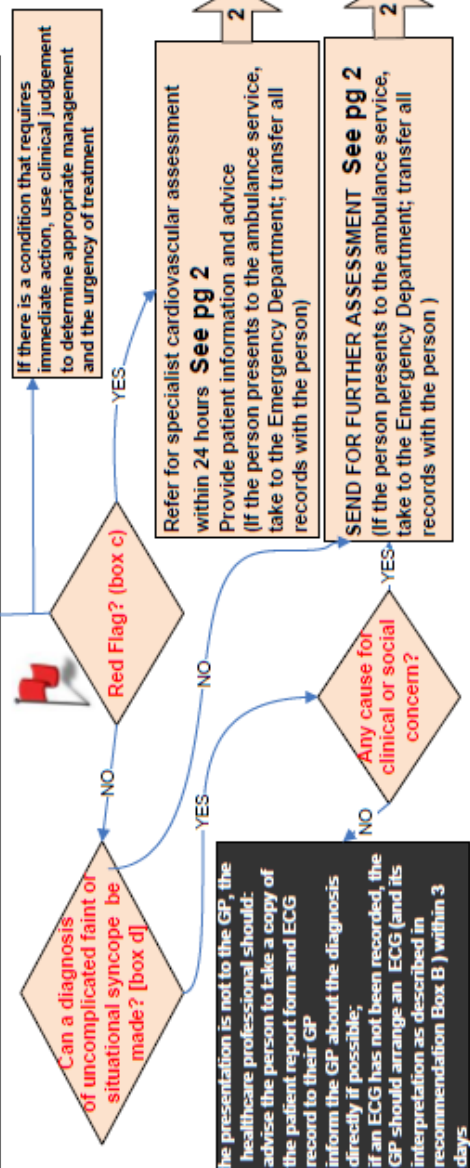
Record a 12-lead ECG using automated interpretation. 12-lead ECG – Treat as a red flag if any of the following abnormalities are reported on the ECG printout:

- conduction abnormality (e.g. complete right or left bundle branch block or any degree of heart block)
- a long or short QT interval, or
- any ST segment or T wave abnormalities

If automated ECG unavailable take manual 12 lead ECG (box b)

**ADDITIONAL TESTS:**

- if there is suspicion of an underlying problem causing TLoC, or additional to TLoC, carry out **relevant** examinations and investigations (for example, check blood glucose levels if diabetic hypoglycaemia is suspected, or haemoglobin levels if anaemia or bleeding is suspected).
- do not routinely use electroencephalogram (EEG) in the investigation of TLoC (see pg. 2 Suspected Epilepsy box)



## Further Assessment and Referral



**Suspected orthostatic hypotension** on the basis of the initial assessment when:

- there are no features suggesting an alternative diagnosis, and
- the history is typical

Yes

Measure lying and standing blood pressure (with repeated measurements whilst standing for 3 minutes)

NO

**Orthostatic hypotension is confirmed?**

YES

If orthostatic hypotension is confirmed, consider likely causes, including drug therapy, and manage appropriately (for example, see 'Falls: the assessment and prevention of falls in older people' [NICE clinical guideline 24]).

Refer all people with TLoC (apart from the exceptions below) for a specialist cardiovascular assessment by the most appropriate local service. Exceptions are:

- uncomplicated faint
  - situational syncope
  - orthostatic hypotension
- and people whose presentation is strongly suggestive of epileptic seizures.

Advise people waiting for specialist cardiovascular assessment.

- What they should do if they have another event.
- If appropriate, how they should modify their activity (for example, by avoiding physical exertion)
- They should not drive prior to seeing cardiovascular assessment

**Suspected epilepsy** - Refer people who present with one or more of the following features (that is, features that are strongly suggestive of epileptic seizures) for an assessment by a specialist in epilepsy; the person should be seen by the specialist within 2 weeks (see 'The epilepsies: the diagnosis and management of the epilepsies in adults and children in primary and secondary care [NICE clinical guideline 20]).

- A bitten tongue.
  - Head-turning to one side during TLoC.
  - No memory of abnormal behaviour that was witnessed before, during or after TLoC by someone else.
  - Unusual posturing
  - Prolonged limb jerking (note that brief seizure-like activity can often occur during uncomplicated faints)
  - Confusion following the event
  - Prodromal déjà vu or jamais vu (see glossary)
- Consider that the episode may not be related to epilepsy if any of the following
- Prodromal symptoms which on other occasions have been abolished by sitting or lying down.
  - Sweating.
  - Prolonged standing that appeared to precipitate TLoC
  - Pallor during the episode

- **EEG should not be used routinely in the investigation of TLoC [see CG20]**
- **Offer advice to people waiting for a specialist neurological assessment for their TLoC [see CG20]**

## Specialist cardiovascular assessment

### HISTORY AND EXAMINATION

Carry out a specialist cardiovascular assessment as follows.

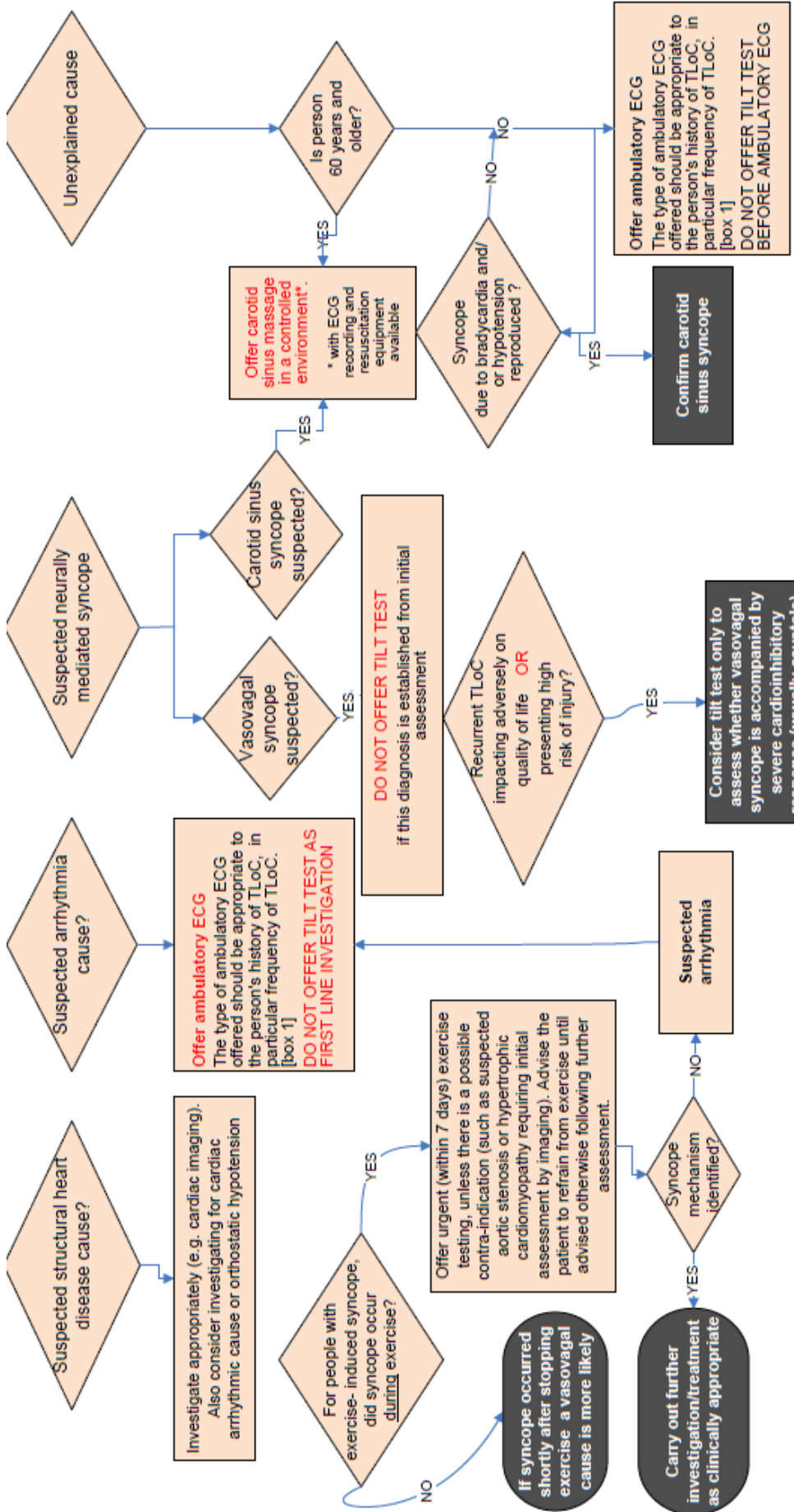
- Reassess the person's:
  - detailed history of TLoC including any previous events
  - medical history and any family history of cardiac disease or inherited cardiac condition
  - drug therapy at the time of TLoC and any subsequent changes.
- Conduct a clinical examination, including full cardiovascular examination and, if clinically appropriate, measurement of lying and standing blood pressure.
- Repeat 12-lead ECG and examine previous ECG documentation.

On the basis of this assessment, assign the person to one of the following causes of suspected syncope:

- suspected structural heart disease
- suspected cardiac arrhythmia
- suspected neurally mediated, or
- unexplained.

Offer further testing see page 3 or other tests as clinically appropriate.

3



**BOX 1**

For people who have:  
 • TLoC at least several times a week, offer Holter monitoring (up to 48 hours if necessary). If no further TLoC occurs during the monitoring period, offer an external event recorder that provides continuous recording with the facility for the patient to indicate when a symptomatic event has occurred.  
 • TLoC every 1-2 weeks, offer an external event recorder\*. If the person experiences further TLoC outside the period of external event recording, offer an implantable event recorder.  
 • TLoC infrequently (less than once every 2 weeks), offer an implantable event recorder. A Holter monitor should not usually be offered unless there is evidence of a conduction abnormality on the 12-lead ECG.  
 \*Excludes event recorders that do not perform continuous ECG monitoring (and therefore are not capable of documenting cardiac rhythm at the moment of TLoC).  
 When offering a person an implantable event recorder, provide one that has both patient-activated and automatic detection modes. Instruct the person and their family and/or carer how to operate the device. Advise the person that they should have prompt\*\* follow-up (data interrogation of the device) after they have any further TLoC.  
 \*\*The timing of the follow-up is dependent on the storage on the device and the condition of the person.

**If the cause remains uncertain or the person has not responded to treatment**

- Consider PNES or Psychogenic pseudo-syncope if a person has persistent TLoC and if, for example,
  - the nature of the event changes over time
  - there are multiple unexplained physical symptoms
  - there are unusually prolonged events
 Refer for neurological assessment
- Advise people who have experienced TLoC to try to record any future events (for example, a video recording or a detailed witness account of the event) particularly if the diagnosis is unclear or taking a history is difficult
- If, after further assessment the cause of TLoC remains uncertain or the person has not responded to treatment, consider other causes of TLoC, including the possibility that more than one pathology may co-exist, for example total arrhythmias

## ACUTE LEFT VENTRICULAR FAILURE

Recognition:

- Sudden onset of dyspnoea or sudden deterioration?
- Previous cardiac history?
- Pallor/sweating?
- Pulmonary crepitations?
- Hypotension/ clammy?

### Treatment

- Inform senior ED doctor immediately
- Consider arrhythmia or MI as cause – monitor, 12-lead ECG
- Give oxygen 100%\*
- **Diamorphine 2.5-5mg iv** +/- metoclopramide 10mg iv
- Administer **Furosemide 40-80 mg iv** (repeat if necessary)
- Consider iv **GTN** (only if SBP >110)
- Consider **CPAP** if respiratory distress
- Notify Cardiac doctor ASAP +/- ICU

## THROMBO-EMBOLIC DISEASE

- IT IS ESSENTIAL TO UNDERSTAND THE DIFFERENCES IN APPLICATION AND NORMAL RANGE BETWEEN D-DIMER TESTING FOR PULMONARY EMBOLISM AND DVT

### 1. Pulmonary Embolism

MASSIVE PULMONARY EMBOLISM IS A CLINICAL DIAGNOSIS MADE IN THE PRESENCE OF SHOCK, RIGHT HEART STRAIN AND SEVERE HYPOXIA WITH RISK FACTORS FOR THROMBOEMBOLIC DISEASE AND NO OBVIOUS ALTERNATIVE DIAGNOSIS (EG MI). GIVE O<sub>2</sub> via NRRM and CONSIDER IMMEDIATE LYSIS (PREFERABLY AFTER CARDIAC ECHO IN RESUS) – SEEK SENIOR ADVICE

Assessment for acute sub-massive pulmonary embolus follows the 'rule in-rule out' method outlined below. A scoring system devised by a team of Canadian physicians forms the basis for our assessment. Although this system incorporates D-dimer testing and is supported by diagnostic imaging, your clinical assessment of the likelihood of PE as opposed to some other diagnosis is crucial.



### **a) Step One: Clinical Assessment**

**History:** Acute pulmonary embolus is often a difficult diagnosis as signs are often non-specific or unreliable. Patients often complain of dyspnoea, pleuritic chest pain or collapse with shock in the absence of other causes.

97% of patients have one of the following

1. Dyspnoea
2. Tachypnoea (Respiratory rate >29/min)
3. Pleuritic chest pain

But you must consider if another diagnosis is more likely.

**Examination:** of the cardiovascular system, chest and legs may confirm your suspicion of PE but physical findings are more often useful in suggesting an alternative diagnosis (see below).

#### **Investigations:**

- **ECG:** should be taken, mainly to exclude acute MI or pericarditis. In PE tachycardia is the most common finding, non-specific ST-T wave abnormality is common, S1Q3T3 is rare.
- **PACXR:** should be also requested. Once again, it is often more helpful in identifying an alternative diagnosis such as pneumothorax, LVF or chest infection. It is normal in 10-20% of patients with PE (note that a normal CXR with hypoxia and significant dyspnoea supports a diagnosis of PE). Most of the remaining patients have non-specific findings like atelectasis / small effusion / elevated diaphragm, cardiomegaly. Occasionally, specific findings like a pulmonary infarct will be seen (wedge shaped, Hampton's hump) or an area of oligaemia identified distal to a dilated vessel (Westermark sign).
- **ABG / O<sub>2</sub> sat:** should always be measured. Low O<sub>2</sub> saturation or P<sub>O</sub><sub>2</sub> increases suspicion in the absence of alternative diagnosis but normal oxygenation does not exclude PE. Comparing ABGs with and without O<sub>2</sub> mask is not helpful.

### **b) Step Two: Measure Canadian Score**

Clinical features of DVT	3.0
Recent immobility or surgery	1.5
Active cancer	1.0
Hx of DVT / PE	1.5
Haemoptysis	1.0
Resting heart rate >100/min	1.5
PE <i>as likely as or more likely than</i> an alternative diagnosis	3.0

SCORE	Pre-test Probability of PE
<2	low
≥ 6	Medium to high risk

### **c) Step Three: Investigation to Rule In or Rule Out PE**

**RULE OUT:** Patients with a low pre-test probability score can have a d-dimer test to rule out PE. IF their D-Dimer is < **250ng/ml**, PE can be excluded and an alternative diagnosis should be sought.

**RULE IN:** Patients with a medium or high pre-test probability OR a D-dimer > **250mg/ml** will probably require radio-isotope scan or CTPA scan to rule in PE irrespective of D-dimer result. Stable patients with a low PESI score may be investigated in the Observation Ward. Patients with a high PESI score are high risk and should be admitted for investigation and management under the medical inpatient team.

### **d) Management**

All patients suspected of having a PE after the above assessment should be treated with **enoxaparin 1.5 mk/kg SC** (reduced in renal impairment as per BNF).

Next their PESI score should be calculated as an indication of risk of complications. Those patients with a PESI score of 85 or less can be investigated through the Observation Ward Pathway. Those with a PESI score higher than 85, a confirmed diagnosis of PE or clinical picture suggestive of a massive PE should be admitted under the respiratory team.

### **PESI Score**

Clinical indicator	Points
Age	+ age (in years)
Male sex	+ 10
Cancer	+ 30
Heart failure	+ 10
Chronic lung disease	+ 10
Pulse > 110 bpm	+ 20
Systolic BP < 100 mmHg	+ 30
Respiratory rate ≥ 30 breaths/min	+ 20
Temperature < 36 <sup>0</sup>	+ 20
Altered mental state	+ 60
Arterial saturation < 90%	+ 20

## **2. Acute Life Threatening Pulmonary Embolism**

**RESUSCITATION** — When a patient presents with suspected PE, the initial focus is on stabilizing the patient.

**Respiratory support** — Supplemental oxygen should be administered if hypoxemia exists. Severe hypoxemia or respiratory failure should prompt consideration of intubation and mechanical ventilation.

**Hemodynamic support** — Hemodynamic support should be instituted promptly when a patient presents with PE and hypotension, defined as a systolic blood pressure <90 mmHg or a drop in systolic blood pressure of  $\geq 40$  mmHg from baseline.

Intravenous fluid administration is first-line therapy. Clinicians should be wary of administering more than 500 to 1000 mL of normal saline during the initial resuscitation period.

If the patient's hypotension does not resolve with intravenous fluids, intravenous vasopressor therapy should promptly follow.

**THROMBOLYSIS** — Thrombolytic therapy accelerates the lysis of acute PE and improves important physiologic parameters, such as RV function and pulmonary perfusion. However, no clinical trial has been large enough to conclusively demonstrate a mortality benefit. Thrombolytic therapy is associated with an increased risk of major hemorrhage, defined as intracranial hemorrhage, retroperitoneal hemorrhage, or bleeding leading directly to death, hospitalization, or transfusion.

Persistent hypotension due to PE (ie, massive PE) is the most widely accepted indication for thrombolytic therapy.

**A 50 mg bolus of alteplase is recommended.**