

GUIDELINES FOR THE
MANAGEMENT OF
PATIENTS
ON THE
CORONARY CARE UNIT

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CCU ADMISSION CRITERIA

1. Assessment, diagnosis and treatment of patients with acute myocardial infarction and other acute coronary syndromes
2. Patients with haemodynamically significant brady- or tachy-arrhythmia
3. Acute cardiogenic pulmonary oedema with haemodynamic compromise
4. Cardiogenic shock
5. Drug overdose associated with life-threatening arrhythmia
6. Confirmed pulmonary embolism with haemodynamic compromise, being considered for thrombolysis

CONSULTANT ALLOCATION ON CORONARY CARE UNIT

- Patients admitted to the unit are automatically allocated the Consultant on call by bed bureau on the patient front sheet
- The whiteboard consultant is deliberately left blank at this stage to enable the appropriate Consultant to add in their name when they see the patient on the ward round
- If a patient admitted is currently under the care of a particular Cardiologist they may be transferred **only** when the registrar / SHO has been able to discuss this with the consultant in question and they have accepted responsibility (may be on holiday etc)
- Occasionally, a patient may be admitted after the ward round and be deemed fit to be transferred to the ward before a Consultant has actually seen the patient. In this instance the patient will be allocated to the Consultant Cardiologist on call for that day by the registrar who has seen the patient. **NB The patient is not allocated to the Consultant doing the ward round that day.** Patient details and ward number will be recorded on the CCU whiteboard. The consultant on the CCU ward round the next day will see the patient on the ward and take over their care.
- Patients may be admitted to cardiology wards after review by the Chest Pain Outreach nurses (e.g. from A&E, MAU etc). In these cases, the patient name is added to the CCU board as above, for review on the next day ward round by the consultant performing the CCU round.
- On CCU w/round it is imperative that medical and nursing staff document a consultant change / allocation in the patient pathway so that there is no discrepancy when transferred to the ward.
- The interventional Cardiologist should give the CCU / medical staff explicit instructions if they wish the patient to be under their care.
- In most cases the consultant will be different to that stated on the hospital system and patient front sheet. The clerk on CCU will make changes on CAMIs after the w/round on a daily basis.
- Both the ward clerk and the nursing staff should ensure that the Patient Front Sheet is changed and identifies the correct Consultant on discharge from CCU. They should also ensure that the patient consultant is identified during handover to the nurses on the Cardiology wards. **Both the ward clerks and staff on the Cardiology wards should ensure that they are aware of the correct consultant on receiving the transferred patient.**

PRE-HOSPITAL TRIAGE - CHEST PAIN ? CARDIAC

Patients referred to hospital with suspected myocardial infarction should be assessed in the community with 12-lead ECG performed by paramedic crew. ECG should be faxed to Telemedicine Receiving Station in CCU. ECG will be reviewed as soon as possible (target 3 minutes) by the shift co-ordinator who will then be contacted by the paramedic to discuss patient's history. A paramedic triage form will be completed and used to help direct the patient to appropriate setting, according to the triage algorithm. Any ECG demonstrating acute ST elevation MI should prompt blue-light transfer and direct admission to CCU.

On some occasions there may be technical problems with the teled transmission of the ECG. In these cases, the co-ordinator should obtain as much information as possible about the patient and ECG (e.g. magnitude of ST elevation). If the paramedic crew feel that the ECG shows pathological ST elevation in a patient with a compatible history, direct admission should be arranged even if the ECG cannot be viewed. The co-ordinator should alert the catheter lab interventionist during office hours or the on-call interventionist out of hours. The interventionist will make a decision on the basis of available information, whether the patient should be directed to CCU or direct to the catheter lab and also whether the on-call team should be activated out of hours.

On receipt of an ECG demonstrating acute ST elevation in a patient with compatible history, the shift co-ordinator should bring the ECG to the catheter lab interventionist or call the on-call interventionist out of hours. The on-call registrar should be alerted, but the co-ordinator need not wait to speak to the registrar prior to contacting the consultant when a diagnostic ECG has been obtained. The ideal is to have the catheter lab ready to receive the patient directly, bypassing CCU and minimising delay.

LBBB is less specific for infarction and patients should be reviewed in CCU before any decision is made to activate catheter lab staff.

GUIDELINES FOR MANAGEMENT OF PATIENTS WITH SUSPECTED MYOCARDIAL INFARCTION

Patients with pre-hospital ECG documentation of ST elevation myocardial infarction (STEMI) or acute ECG changes representing myocardial ischaemia should be admitted direct to CCU (or the cardiac catheter lab in confirmed ST elevation).

Patients with suspected cardiac chest pain without ST elevation on initial ECG should be admitted to Chest Pain Observation Bay (CCU).

An initial assessment, including 12 lead ECG, will be made by a senior CCU nurse. Patients with typical presentation of acute myocardial infarction presenting within 12 hours of onset of major symptoms and diagnostic ST elevation on ECG should have primary PCI – contact catheter lab interventionist or on-call interventionist out of hours whether patient has arrived or not. During office hours, the interventionist working in the catheter lab that day should be contacted immediately.

Regardless of initial nurse management, patients should be seen by the on call cardiology SHO/Registrar within 5 minutes. NB Nursing staff will be able to give opiates via Patient Group Directive.

The cardiology CCU SHOs are responsible for all admissions. The names and bleep numbers of the responsible SHOs are displayed on the CCU whiteboard at all times.

A rapid assessment should lead to the following categorisation:-

1. Definite MI e.g. typical history + definite ECG changes (ST elevation or LBBB)
2. Uncertain e.g. typical history + non-diagnostic ECG (MI Rule Out Pathway).
3. Definitely not MI e.g. chest infection.

The Chest Pain Triage Algorithm should be used to help with appropriate categorisation.

Admissions and Discharges.

Definite MI and uncertain MI should be admitted to CCU.

Definitely not MI should be referred to a general medical ward/MAU/home. Patients should not be discharged home without the authority of a registrar or consultant.

Patients with an uncomplicated MI can be moved to a cardiology ward approximately 12 hours after admission.

Patients should be prioritised for discharge from CCU, making provision for emergency admissions and avoiding having to move patients out "in a rush".

Cat. A = Fit for discharge from CCU

Cat B = Could be transferred from CCU if essential

Cat C = Must stay in CCU

Prioritisation should be reviewed at least daily and recorded on white board. Consultant responsible for continuing care of patient should be clearly marked at same time.

**GUIDELINES FOR THE MANAGEMENT OF
DEFINITE MYOCARDIAL INFARCTION**

DO NOT DELAY.

Insert a venous cannula, preferably at least a green venflon in left ante-cubital fossa.

Give **ANALGESIA** e.g. morphine 10 mg iv with 5 mg increments every 10minutes as needed.
Max 20mg without senior review. Caution in elderly patients or with low body mass

Give **ANTIEMETIC** e.g. metoclopramide 10 mg iv.

Give **ASPIRIN** 300 mg chewed - unless allergic or already given.
If allergic, give clopidogrel 600 mg po.

Give **OXYGEN** Measure pulse oximetry and aim for SaO₂ > 96%

Immediate **REPERFUSION THERAPY** is main stay of management

ALL PATIENTS WITH STEMI SHOULD BE CONSIDERED FOR **PRIMARY ANGIOPLASTY** -
INFORM CATHETER LAB OR ON-CALL INTERVENTIONIST AND CARDIOLOGY SPR ON
BLEEP 9595.

For primary/ rescue/ salvage PCI patients:

Give **CLOPIDOGREL** 600mg po prior to procedure even if already on long term clopidogrel.
The target time for door to balloon inflation time is 90 minutes

NB patients are usually admitted directly to catheter lab for PPCI. On return to CCU, they need a full clerk-in by SHO.

THROMBOLYSIS

If patient is not eligible for primary angioplasty, consider thrombolysis :

Tenecteplase single weight adjusted iv bolus over 5 seconds, as below:

Patient Weight (kg)	TNKase (mg)	Volume TNKase* to be administered (mL)
< 60	30	6
≥ 60 to < 70	35	7
≥ 70 to < 80	40	8
≥ 80 to < 90	45	9
≥ 90	50	10
*From one vial of TNKase reconstituted with 10 mL SWFI.		

Comcommitant heparin should be administered and continued for 48hrs:

4000U bolus + 800U/hr if weight <67kg - adjusted as per aPTT
5000U bolus + 1000U/hr if weight ≥kg - adjusted as per aPTT

TRANSFER PROTOCOL FOR STEMI (DGH patients)

All patients within the North of England Cardiovascular Network are now being transferred to JCUH or Freeman for primary PCI rather than having thrombolysis on-site.

Referrals /transfers may come from 3 sources:

1. STEMI patients identified by paramedic crew.
These patients will be brought directly to JCUH CCU. The paramedic crew alerts the JCUH CCU shift co-ordinator with telemetered ECG and referral is accepted or re-directed to the normal base hospital if no pathological ST elevation on ECG. The shift co-ordinator should contact the interventionist / SpR / catheter lab team in the normal way.
2. STEMI patients who self present to A&E.
On diagnosis of STEMI, A&E doctor contacts JCUH co-ordinator urgently with faxed ECG. Ambulance control contacted and an emergency “critical care transfer” requested (not “within the hour” urgent transfer). JCUH shift co-ordinator contacts the interventionist / SpR / catheter lab team as above. Patient given CLOPIDOGREL 600mg prior to transfer unless this will incur a delay. Ideally, patient transferred direct to catheter lab.
3. Patients in DGH CCU or other ward with chest pain who develop ST elevation MI after admission.
Senior CCU Nurse contacts JCUH co-ordinator urgently with faxed ECG. Ambulance control contacted and an emergency “critical care transfer” requested (not “within the hour” urgent transfer). JCUH shift co-ordinator contacts the interventionist / SpR / catheter lab team as above. Patient given CLOPIDOGREL 600mg prior to transfer unless this will incur a delay. Ideally, patient transferred direct to catheter lab.

Following PPCI, the patient should be offered the choice of repatriation to the base hospital for the remainder of the in-patient stay or to remain at JCUH until discharge. This will be based on an estimate of the projected discharge date on the post PCI or CCU ward round and at the discretion of the responsible interventional cardiologist.

If transfer is to be arranged, the CCU co-ordinator should contact the base hospital CCU to book bed. It is the responsibility of the base hospital to secure the bed for the anticipated transfer of the patient. On transfer, an immediate discharge summary will be completed by the CCU co-ordinator outlining diagnosis, angiographic findings, treatment, anticipated discharge date and further management recommendations (e.g interval exercise test to assess other lesions). For those patients discharged direct from JCUH CCU, an immediate discharge summary as above will be faxed to the base hospital CCU. This will provide basic information in case the patient is re-admitted.

ANTICOAGULATION

Heparin used for patients with acute coronary syndromes . Use s/c LMWH, eg enoxaparin 1mg/kg bd, unless early percutaneous intervention or CABG is being considered. Post PCI heparin is not usually required unless as prophylaxis against DVT/PE: enoxaparin 20mg od (40mg od if high DVT/PE risk).

Patients with renal failure on dialysis or with creatinine > 400 have unpredictable heparin clearance and should have half-dose LMWH 60units/kg.

Unfractionated heparin iv should be used in patients with intra-aortic balloon pumps in situ or when patients awaiting CABG become unstable and may require urgent surgery.

All in patients should be considered for DVT prophylaxis as per trust protocol - dalteparin 5000 IU / day until discharge.

INTRAVENOUS ANTI-PLATELET THERAPY (gp IIb/IIIa receptor inhibitors)

Most patients who undergo infarct PCI or other high risk PCI will have intravenous anti-platelet therapy for 12 hours post procedure. Most patients will receive abciximab (ReoPro), commenced in the catheter lab.

Patients may be transferred in from other units on either tirofiban (Aggrastat) or eptifibatide (Integrelin). Occasionally patients with high risk features who are not immediately suitable for treatment in the catheter lab will be medically stabilised with tirofiban in CCU prior to angiography. (see protocol below)

A small minority of patients will develop severe thrombocytopenia and platelet counts are mandatory at 4 and 12 hrs post commencement of infusion. Pseudo-thrombocytopenia due to platelet clumping is common and should be excluded by a further sample in citrate.

TIROFIBAN PROTOCOL

Concentrate must be diluted before use.

Preparation and Dosage

Withdraw and discard 50ml from a 250ml bag of 0.9% NaCl.

Withdraw contents of 1 vial (50mls) tirofiban concentrate and add to bag, making a total of 250ml at a concentration of 0.05mg/ml or 50mcg/ml.

Give an INITIAL LOADING INFUSION at a rate of 0.4mcg/kg/min for 30 mins as indicated in the table.

Set IVAC rate and VTBI to run for 30 mins only.

After 30 mins, continue with MAINTENANCE INFUSION at a rate of 0.1mcg/kg/min as indicated in the table for at least 48 hours, not exceeding 108 hrs (or 12 hours post PCI on instruction of interventionist).

Weight (kg)	≤37	38-45	46-54	55-62	63-70	71-79	80-87	88-95
Loading dose (mcg/30min)	400	500	600	700	800	900	1000	1100
IVAC rate (ml/hr)	16	20	24	28	32	36	40	44
IVAC VTBI (total vol over 30 mins)	8	10	12	14	16	18	20	22
Maintenance dose (mcg/hr)	200	250	300	350	400	450	500	550
IVAC rate (ml/hr)	4	5	6	7	8	9	10	11

Weight (kg)	96-104	105-112	113-120	121-128	129-137	138-145	≥146
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Loading dose (mcg/30min)	1200	1300	1400	1500	1600	1700	1800
IVAC rate (ml/hr)	48	52	56	60	64	68	72
IVAC VTBI (total vol over 30 mins)	24	26	28	30	32	34	36
Maintenance dose (mcg/hr)	600	650	700	750	800	850	900
IVAC rate (ml/hr)	12	13	14	15	16	17	18

Tirofiban is usually administered with unfractionated heparin until PCI performed, then for 12 hrs post PCI. If patient already on LMWH, this may be continued rather than iv unfractionated heparin. Severe renal failure (creatinine clearance <30ml/min): reduce dosage by 50%.

OTHER THERAPY

Give **NITRATES** e.g. buccal suscard 2 - 10 mg, routinely (it reduces pain) unless hypotensive (systolic <90).

Aim to keep systolic blood pressure 100-120 mm Hg without reflex tachycardia or too great a fall from 'baseline'. Buccal nitrates 2 mg - 10 mg for the first 6 hours then discontinue.

Consider **BETA-BLOCKERS** especially if hypertensive, tachycardic, continuing pain, not suitable for reperfusion therapy etc. Give metoprolol 5 mg - 10 mg iv then continue orally. Beta-blockers should not be used in patients with significant acute pulmonary oedema.

Routine tests:

FBC

Random cholesterol (on admission or <24 hrs from onset of pain)

troponin T (on arrival and the shorter of >10hrs after admission or 12 hrs after pain onset, or at 6 am if second TnT required overnight)

K⁺ and glucose

Chest X-ray – on admission or as soon as is practical thereafter

MIRO (Myocardial Infarction Rule out)–

Troponin T at admission and 10-12 hours from onset of symptoms

CONTINUING TREATMENT

Patients with an uncomplicated infarction can be discharged to a step-down bed or referring centre 12-24 hours after admission. All MI patients should be on the following therapy unless contra-indicated.

1. ASPIRIN 75 mg od indefinitely.
If GI intolerance, add lansoprazole 30mg od. If allergic, clopidogrel 75mg od.
2. CLOPIDOGREL 75mg od for 1 year.
3. BETA-BLOCKERS indefinitely
unless asthma, decompensated heart failure, heart block, heart rate <50 bpm, blood pressure <100 mm Hg.
Usually use metoprolol 25mg bd to initiate and change to once daily agent next day if tolerated (e.g. bisoprolol 5mg)

In stabilised heart failure, carvedilol 3.125mg bd (target 25mg bd) or bisoprolol 1.25mg od (target 10mg)

NB not contraindicated by impaired LV function, COPD or peripheral vascular disease

4. ACE-INHIBITORS indefinitely; start 24h post-MI
e.g. ramipril 2.5–10 mg od, perindopril 2-8mg od.
Titrate upwards to target dose. Avoid or introduce with caution if SBP < 90 mmHg or if creatinine >200
5. STATINS indefinitely
Use atorvastatin 80mg od for all confirmed ACS patients, regardless of initial cholesterol level. Both diltiazem and verapamil have significant interactions with statins and a maximum dose of 20mg simvastatin (or equivalent) should be used with verapamil; 40mg simvastatin or equivalent with diltiazem.
6. GTN. All IHD patients should be given a GTN spray with instructions on use at discharge

OTHER THERAPY in selected patients

1. VERAPAMIL (if beta-blockers contraindicated)
verapamil sustained release 240 mg od. Avoid if impaired LV. NB statin interaction.
2. GLUCOSE
All patients presenting with myocardial infarction + hyperglycaemia requiring GKI or insulin infusion should be referred to on-call diabetes team for consideration of long term insulin therapy.
3. ALDOSTERONE ANTAGONISM
All patients with clinical heart failure complicating MI and echo evidence of moderate or severe LV dysfunction should be considered for eplerenone 25 mg od, started 3-14 days post MI, ideally after ACE inhibitor. Titrate to 50mg within 4 weeks. Renal function should be checked 48 hours and 1 week after initiation or after dose change.

MOBILISATION

Bed rest for first 12 hours. Bedside commode.

Uncomplicated - aim for discharge 48-72hrs post PPCI.

Day 2 mobilise around bay.

Day 3-5 mobilise around ward ± stairs.

Cardiac rehabilitation referral Day 1. Supply Rehabilitation booklets/advice and patient diary in CCU.

Patients with complications should be discharged approximately 3-5 days after they have been stabilised.

FOLLOW-UP POST MYOCARDIAL INFARCTION

UNCOMPLICATED PATIENTS

All patients should have an echocardiogram to assess LV function prior to discharge (if possible). In some cases patient may be discharged prior to echo. In these cases, local hospital should arrange echo rather than patient travelling back to JCUH.

See C2C Network ICD Guidelines for indications for ICD insertion post MI (page 27).

All NSTEMI should have angiogram \pm revascularisation, prior to discharge unless contraindicated. Patients not undergoing angiography should be considered for an exercise ECG pre-discharge.

COMPLICATED PATIENTS

Patients with post-infarct angina/heart failure/arrhythmia should be fully assessed pre-discharge.

MANAGEMENT OF COMPLICATIONS OF ACUTE MI

RECURRENT CHEST PAIN

Remember a 'bruised feeling' is common after an MI.

Pericarditis will be different with postural or pleuritic pain and a rub may or may not be present - treat with paracetamol 1 G qds. If not settling, consider colchicine 500mg qds until symptoms relieved.

Recurrent infarction will be manifested by new ST elevation (or ST re-elevation) and will require iv nitrates, heparin (+ beta-blockers) and consideration of repeat PCI - discuss with cardiologists as matter of urgency.

Recurrent ischaemia will be manifested by ST depression and will require buccal or iv nitrates, beta-blockers, heparin etc. - such patients may require repeat emergency coronary angiography and revascularisation - discuss with cardiologists as matter of urgency.

HEART FAILURE

Mild degrees of pulmonary congestion are common and respond to low dose diuretics e.g. furosemide plus initiation of ACE-I therapy. **N.B.** Watch potassium.

Greater degrees of heart failure e.g. pulmonary oedema require more vigorous treatment - oxygen, iv diamorphine, iv furosemide. If blood pressure allows, give iv nitrates, if blood pressure low then iv dobutamine \pm dopamine.

If there is an associated murmur get an urgent echocardiogram.

If pulmonary oedema is refractory consider CPAP or ventilation. Intra-aortic balloon pump insertion may be required if severe heart failure complicates infarction.

Patients with heart failure post MI need ACE-I treatment and (if renal function and potassium allow) eplerenone.

SHOCK

Shock is a clinical syndrome of hypotension, oliguria and poor peripheral perfusion. When first recognised, the duty cardiology registrar and/or consultant should be notified immediately.

1. Cardiogenic shock early in the course of infarction.

If the patient presents with shock within 24 hours after the onset of infarction consider emergency angiography and revascularisation. All patients with cardiogenic shock should be discussed urgently with the cardiology registrar or consultant.

Certain patient groups have a very poor outlook and may not benefit from PCI. Poor prognostic markers include advanced age >75yrs, failed thrombolysis, previous CABG, previous MI and other serious comorbidity.

2. Shock and pulmonary oedema.

This is usually associated with a large infarct or an infarct in a patient with previous infarctions. Treatment is as for pulmonary oedema plus the addition of inotrope i.e. dobutamine 2.5 µg/kg/min up to 20 µg/kg/min ± dopamine 2 - 5 µg/kg/min. Consider Swan Ganz catheter.

3. Shock plus a murmur.

This raises the possibility of either an acute ventricular septal defect or mitral regurgitation due to papillary muscle rupture which may require surgical treatment - get an urgent echo/ senior cardiology opinion.

4. Right ventricular infarction.

This can produce a clinical syndrome of shock with clear lung fields. All patients with inferior wall STEMI should have right sided ECG leads recorded. In the acute phase (first 12 hours) hypotension and evidence of right ventricular involvement should prompt consideration of IV fluids to raise BP.

RV infarction can be confirmed by insertion of a Swan Ganz catheter and the findings of high right sided pressures with a low wedge pressure. Under these circumstances cautious volume expansion with colloid improves blood pressure and cardiac output. This should be titrated against the wedge pressure.

“Fluid resuscitation” should not be used in anterior infarction or cardiogenic shock without haemodynamic monitoring and senior guidance. Hypotension in this situation is usually due to extensive LV damage and not under-filling.

GUIDELINES OF THE MANAGEMENT OF UNCERTAIN MYOCARDIAL INFARCTION

If in doubt, ask advice.

Remember ST segment elevation may develop rapidly, therefore repeat a 12 lead ECG every 30 minutes if necessary.

Clinical judgement will be required where the 'baseline' ECG is abnormal e.g. previous MI or RBBB etc.

If the pain is ischaemic and the ECG shows ST depression or other changes treat as acute coronary syndrome.

Remember to record an ECG with posterior leads to look for posterior infarction.

GUIDELINES OF THE MANAGEMENT OF ACUTE CORONARY SYNDROME

Insert a venous cannula. Take blood for Troponin T estimation. Obtain ECGs with pain and when pain free.

Give ASPIRIN 300 mg stat and 75 mg od

CLOPIDOGREL If clear ACS (classical symptoms and ECG changes), give 600mg loading followed by 75mg od. Discontinue if diagnosis revised on subsequent investigations. **Do not load all suspected ACS patients with clopidogrel unless diagnosis confirmed by elevated troponin T or significant ECG changes.**

Give NITRATES e.g. buccal nitrates 2 - 10 mg or glyceryl trinitrate iv 10 µg/min - 100 µg/min. Aim to keep systolic blood pressure 100-120 mm Hg without reflex tachycardia or too great a fall from 'baseline'. If infusing nitrates iv use for 4 hours then switch to buccal nitrates 2 - 10 mg for next 8 hours. If the pain has settled change to oral nitrates.

Give BETA-BLOCKERS e.g. metoprolol 5 mg - 15 mg IV, followed by metoprolol 50-100 mg bd or bisoprolol 5-10mg od.
If beta-blockers contraindicated because of asthma or still hypertensive despite beta-blockers, use verapamil or diltiazem (not beta-blocker and verapamil together)

Give HEPARIN give low molecular weight heparin e.g. enoxaparin 1mg/kg bd
If on warfarin in therapeutic range, do not add heparin.
Do not administer LMWH to all suspected ACS patients unless diagnosis confirmed by elevated troponin T or significant ECG changes.

If severe pain

Give ANALGESIA e.g. morphine 10 mg iv with 5mg increments every 10 minutes as needed. Max 20mg without senior review. Caution in elderly patients or with low body mass

Give ANTIEMETIC e.g. metoclopramide iv 10 mg.

If pain does not settle remember to repeat ECG - if ST elevation treat as for MI. If recurrent pain despite above measures consider emergency angiography and revascularisation.

Patients presenting with possible acute coronary syndrome whose pain settles, or whose TnT is normal at 12 hours, can have heparin discontinued 24 hours after last episode of pain.

Patients with pain on mobilisation should be considered for angiography.

Patients whose admission ECG shows ischaemia or with raised Troponin T should be considered for angiography.

- Patients pain-free on mobilisation and without ischaemia on admission ECG and a negative Troponin T should undergo an inpatient stress test - if abnormal they should be considered for angiography.

Reasons for not referring for invasive investigation

high bleeding risk – e.g. anaemia with undiagnosed cause, recent GI/cerebral bleed, recent surgery

high procedural risk – e.g. renal failure, intolerance of antiplatelet drugs, severe cerebrovascular or peripheral vascular disease

poor non-cardiac prognosis – e.g. severe copd, disseminated malignancy

Intensive Medical Therapy

first dose of low mol wt heparin and the 600 mg loading dose of clopidogrel should be given prior to troponin result for those with ischaemic ecg but should be continued only if troponin elevated

low mol wt heparin should be given until patient pain free for at least 48 hrs

clopidogrel 75mg daily should be continued for 12 months

Troponin

Troponin is measured on admission and 12 hours post onset of worst symptoms or 12 hours post admission if onset is unclear .

Current accepted definition of MI is any elevation of troponin associated with at least one of:

- typical symptoms of myocardial ischaemia
- ECG changes indicative of NEW ischaemia

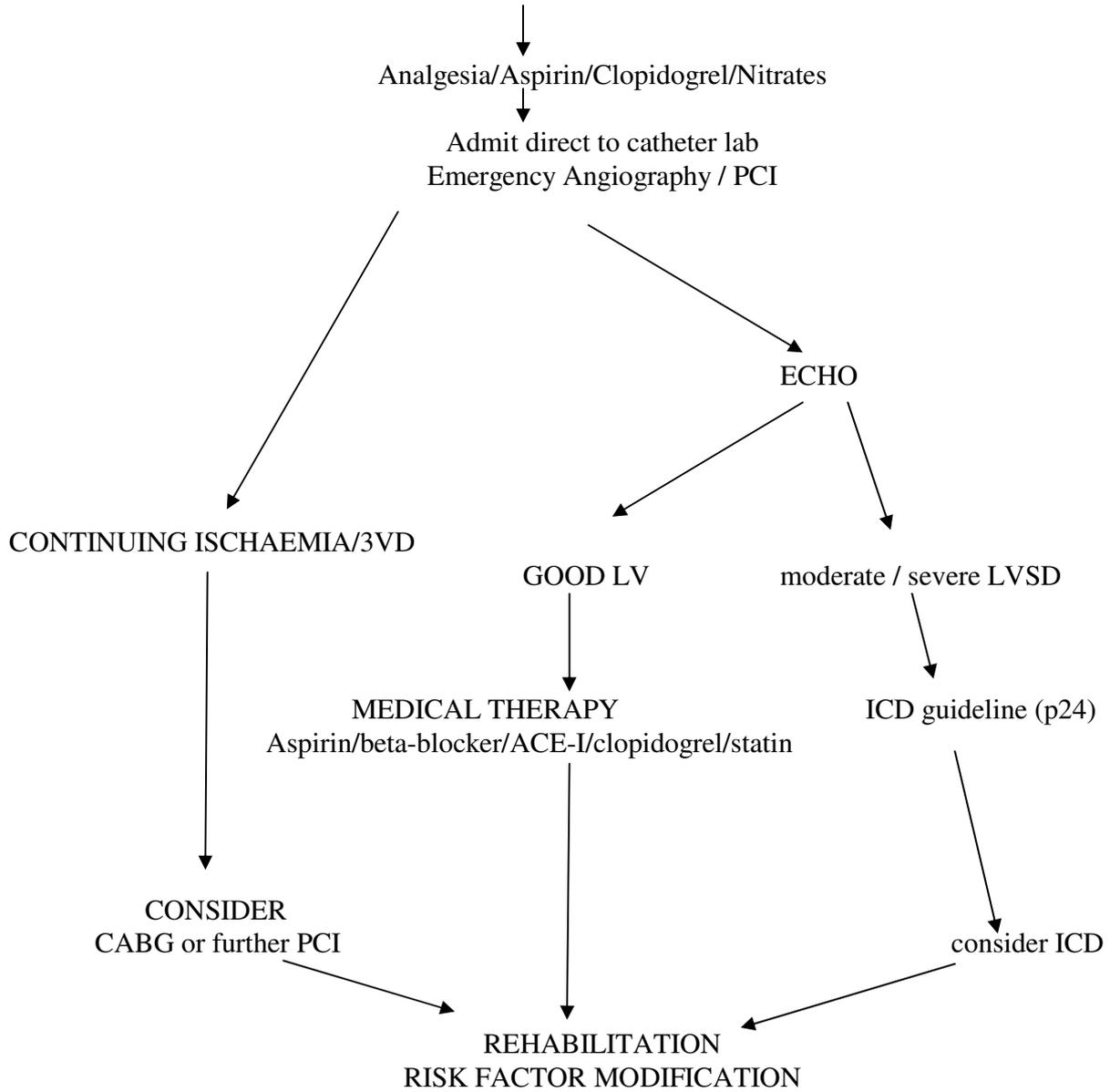
- Development of pathological Q waves on ECG
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

Where diagnosis of MI is entirely dependant on troponin, a minimum of two levels should be compared to look for typical rise or fall, with minimum 20% increase required. This will not be required where diagnosis clinically obvious or confirmatory information is available from ECG, angiography or other imaging modalities.

when TnT is raised consider non-ischaemic causes which include:

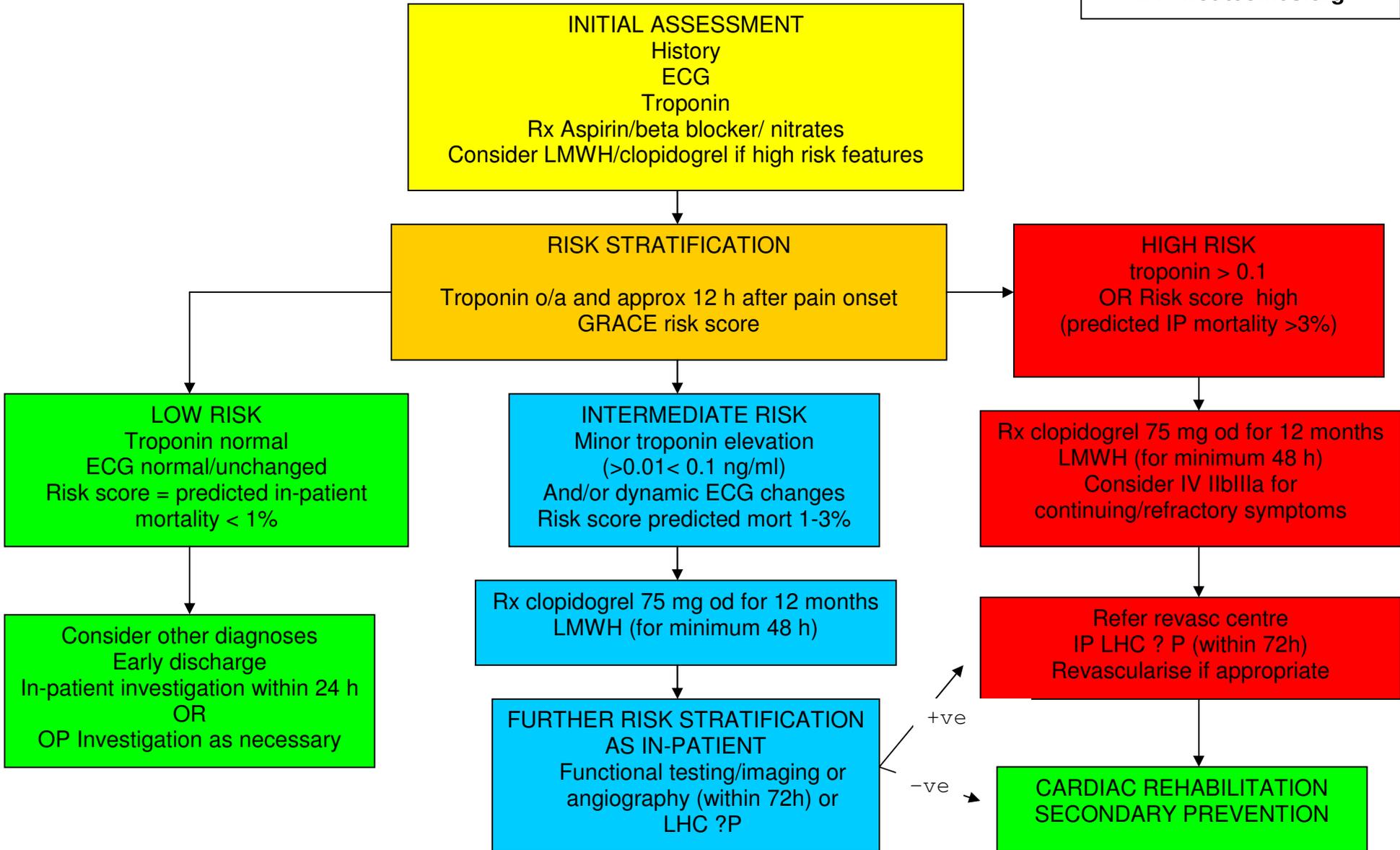
- pulmonary embolism
- myocarditis
- congestive cardiac failure
- dc cardioversion
- tachyarrhythmias (eg vt, fast af/svt)
- renal failure
- septicaemia
- non-cardiogenic shock
- stroke/sah
- pneumonia
- gi bleeding
- chemotherapy

ACUTE ST ELEVATION MYOCARDIAL INFARCTION



**NORTH ENGLAND CARDIOVASCULAR NETWORK
GUIDELINES FOR MANAGEMENT OF ACS 2008**

GRACE risk score
www.outcomes.org



HEPARIN

Use low molecular weight heparin – enoxaparin 1mg/kg bd – for unstable angina and NSTEMI. If coronary angiography planned, stop on day of procedure and ensure at least **12 hours** elapsed since last dose before removing femoral sheath.

If heparin is required after angiography or PCI, use heparin 1000 u/ml strength by infusion pump.

To initiate heparin give 80 units/kg bolus maximum 5000 units then start the infusion based on body weight at 18 units/ kg/hour (hr) average rates indicated below

Commence infusion based on body weight:

- < or = 40kg = 0.7 ml/hr
- 50 kg = 0.9 ml/hr
- 60 kg = 1.1 ml/hr
- > or = 70 kg = 1.25 ml/hr

Measure APTR 4-hours and adjust dose according to results.

APTR	Recommended change infusion rate
> 5.0*	Stop for one hour and reduce dose by 500 units /hr (0.5mls/hr)
4.1 to 5.0*	Reduce rate by 300 units/hr (0.3 ml/hr)
2.7 to 4.0*	Reduce rate by 100 units/hr (0.1 ml/hr)
1.6 to 2.6	Continue same rate
1.2 to 1.5	Increase rate by 200 units/hr (0.2 ml/hr)
< 1.2	Increase rate by 400 units/hr (0.4 ml/hr)

*if patient is bleeding with high APTR, stop heparin infusion for one hour and reassess clinical situation before recommencing infusion.

Recheck APTR 4 hours after dose change.

If therapeutic recheck APTR daily unless any change in clinical condition, bleeding, renal impairment. Patients with renal failure may require more intensive monitoring.

HEPARIN INDUCED THROMBOCYTOPAENIA (HIT)

Both LMWH and UFH may result in HIT. Platelet counts should therefore be checked at baseline and every 48-72 hrs thereafter in patient who remain on either form of heparin.

Type I is generally benign - patients have a transient decrease in platelet count without any further symptoms. This recovers even if heparin continues to be administered. It is not due to an immune reaction and antibodies are not found upon investigation.

Type II HIT is due to an autoimmune reaction with antibodies formed against various platelet factor 4 which form complexes with heparin. Type II HIT develops in about 3% of all patients on UFH and in < 1% of patients on LMWH, and causes thrombosis in 30% to 40% of these patients. Type II HIT is the main contraindication to heparin

The use of heparin in patients with HIT may result in severe arterial and venous thrombosis with a significant associated mortality. If HIT is suspected, an immediate referral to the haematology Consultant or SpR should be made, who will advise on appropriate investigations to confirm diagnosis. After discussion with Haematology medical staff, a serum sample in a plain tube (red top at JCUH,

white at Friarage) should be sent urgently to the coagulation lab with an accompanying phone call so that the sample can be processed on arrival. A result should be available within 1 hour.

If HIT is suspected or confirmed, alternative anti-thrombotic treatments should be used. The heparinoid danaparoid, which inhibits factor Xa and thrombin, is a suitable alternative. The following protocol should be followed in patients suspected of/ or previously diagnosed with HIT.

Danaparoid Administration

Therapeutic Dose:

In general, monitoring of plasma anti-Xa activity is not necessary. However, in patients suffering from renal insufficiency, patients weighing over 90kg, and those with prosthetic valves monitoring is recommended. The need for monitoring will be determined by the Haematologist. There is a standard operating procedure for this test in the laboratory however as this is a specialised test it may not be possible outside normal working hours.

Danaparoid should be administered intravenously as a bolus dose of:

- 1250 units < 55kg
- 2500 units 55-90kg
- 3750 units > 90kg

Followed by an intravenous infusion of 400units/hour for 2 hours then 300 units/hours for 2 hours and then a maintenance infusion of 200 units/hour.

The expected plasma anti-Xa levels are 0.5-0.7 units/ml 5-10 minutes after the bolus, not higher than 1.0 units/ml during the adjustment phase of maintenance infusion and 0.5-0.8 units/ml during the maintenance infusion.

A regimen for the use of danaparoid in patients having alternate day dialysis is given in the table below

IV Bolus	Monitoring
3750 (2500*) units before first and second dialysis;	Anti-Xa 0.5-0.8units/ml
3000 units before third dialysis; then according to predialysis anti-Xa level:	
<0.3 3000 (2000)units	
0.3-0.35 2500 (1500)units	
0.35-0.4 2000 (1500)units	
>0.4 omit	
*Doses in parentheses for patients <55kg	

Thromboprophylaxis

Patients with a previous history of HIT requiring thromboprophylaxis should have HIT antibodies measured if >100 days from diagnosis. If antibodies persist or it is less 100 days from diagnosis then Danaparoid should be used, no monitoring is required.

Danaparoid 750 units subcut twice daily

HIT in patients undergoing PCI

The novel short-acting, direct-thrombin inhibitor bivalirudin is the only alternative to heparin that has been well validated in patients undergoing Percutaneous Coronary Intervention (PCI). In patients undergoing PCI with HIT or with a previous history of HIT, bivalirudin should be used in the catheter lab as an alternative to heparin.

It is anticipated that a diagnosis of HIT will have been confirmed before a patient undergoes PCI as a planned procedure. The management of a patient with a potential diagnosis of HIT requiring PCI as an emergency should be discussed with the Haematologist on call.

Bivalirudin Administration

Dose:

Bivalirudin can be stored at room temperature, when reconstituted should be used immediately. There will be a supply in Catheter Lab 3 drug cupboard (one adult dose) with further stock available in pharmacy.

- ◆ Intravenous bolus dose 0.75mg/kg
- ◆ Followed immediately by an intravenous infusion at a rate of 1.75mg/kg/hr for at least the duration of the procedure. It may be continued for up to 4hrs if clinically warranted.
 - Inject 5 ml of sterile water for injection into the 250 mg Angiox[®] vial.
 - Gently swirl until the powder is completely dissolved and the solution is clear.
 - After reconstitution 1ml contains 50mg bivalirudin.
 - Withdraw 5 ml of the reconstituted Angiox[®] (bivalirudin) solution from the vial and inject it into the 50 ml intravenous infusion bag to give a final concentration of 5mg/ml.
 - **After dilution 1 ml contains 5 mg bivalirudin**

- Determine the correct dose of Angiox[®] (bivalirudin) based on patient weight:

Angiox [®] (bivalirudin) dosing Using 5 mg/ml concentration			
Patient weight (kg)	Bolus dose 0.75 mg/kg irrespective of renal function (ml)	Normal renal function & mild renal impairment (GFR >60 ml/min)	Moderate renal impairment (GFR 30-59 ml/min)
		Infusion 1.75 mg/kg/hour (ml/h)	Infusion 1.4 mg/kg/hour (ml/h)
43-47	6.8	15.8	12.6
48-52	7.5	17.5	14.0
53-57	8.3	19.3	15.4
58-62	9.0	21.0	16.8
63-67	9.8	22.8	18.2
68-72	10.5	24.5	19.6
73-77	11.3	26.3	21.0
78-82	12.0	28.0	22.4
83-87	12.8	29.8	23.8
88-92	13.5	31.5	25.2
93-97	14.3	33.3	26.6
98-102	15.0	35.0	28.0
103-107	15.8	36.8	29.4
108-112	16.5	38.5	30.8
113-117	17.3	40.3	32.2
118-122	18.0	42.0	33.6
123-127	18.8	43.8	35.0
128-132	19.5	45.5	36.4
133-137	20.3	47.3	37.8
138-142	21.0	49.0	39.2
143-147	21.8	50.8	40.6
148-152	22.5	52.5	42.0
153-157	23.3	54.3	43.4

- Withdraw the bolus dose from the IV infusion bag and administer the bolus dose to the patient. The recommended bolus dose is 0.75 mg/kg.
- Set the infusion rate according to patient weight. The recommended infusion rate is 1.75 mg/kg per hour for at least the duration of the procedure. In moderate renal impairment (GFR 30-59 ml/min) the infusion rate should be reduced to 1.4 mg/kg per hour. Angiox[®] (bivalirudin) is contraindicated in patients with severe renal impairment (GFR < 30ml/min) and in dialysis-dependent patients.
- The infusion may be continued for up to 4 hours after the PCI procedure, if clinically warranted
- Routine ACT monitoring is not needed.

Ongoing Care: After care is not affected by the use of Bivalirudin and the standard protocol should be followed. Patients with HIT requiring in going anticoagulation will require danaparoid or lepirudin as advised by Haematology.

STEMI - CRITERIA FOR EMERGENCY TRANSFER FROM OTHER UNITS FOR ANGIOGRAPHY AND REVASCULARISATION

All STEMI patients presenting within 12 hours of symptom onset. In some cases, the time course may be uncertain and individual discussion will be necessary. Immediate transfer for PCI may also be considered in those presenting within 24 hours if still in significant pain.

Cardiogenic shock complicating recent infarction

Recurrent infarction will be manifested by new ST elevation (or ST re-elevation) and will require iv nitrates, heparin (+ beta-blockers) and transfer for PCI.

Post-infarct angina despite therapy with nitrates, beta-blockers, heparin etc. should lead to consideration of angiography and emergency revascularisation

Infarction after recent PCI suggests stent thrombosis. Patients presenting with STEMI within 8 weeks of PCI should be transferred urgently to the catheter lab for further PCI. Thrombolysis should **not** be administered.

All these are guidelines, not absolute rules and there is clearly a need for discretion and clinical judgement. Individual cases should be discussed with the cardiologists.

ARRHYTHMIAS

Bradycardia

Sinus bradycardia is common and unless associated with hypotension or rate <40 bpm requires no treatment - otherwise give atropine 0.6 mg iv.

Heart block with inferior myocardial infarction:

1. 1st degree heart block - observe, no treatment.
2. 2nd degree Mobitz I (Wenkebach) - no treatment.
2nd degree Mobitz II - no treatment unless associated with hypotension, then atropine 0.6 mg iv.
If no response temporary pacing.
3. 3rd degree complete heart block - no treatment unless associated with hypotension, heart failure, broad complex rhythm or rate <40 bpm, then atropine 0.6 mg iv repeated if necessary (maximum 1.2mg) If no response temporary pacing.

Heart block with anterior myocardial infarction:

1. 2nd degree Mobitz II - temporary pacing.
2. 3rd degree complete heart block - temporary pacing.

TEMPORARY PACING

Temporary pacing should be performed by the cardiology SpR or on call SHO supervised by the on call cardiology registrar.

In an emergency consider using the transthoracic pacer whilst preparing for/introducing a transvenous wire. Give the patient adequate sedation/analgesia. Attach pacing pads and increase energy until electrical capture seen on ECG. Ensure mechanical capture is achieved by feeling large volume pulse and auscultating heart sounds.

Isoprenaline infusion 0.5-2.5 µg/min may be used to *temporarily* maintain heart rate while preparing for pacing. (N.B. increased oxygen consumption and myocardial irritability).

Access should be via the internal jugular or right subclavian vein. If the patient is anticoagulated consider femoral vein. Central access should be gained under ultrasound guidance.

Tachycardia

Sinus tachycardia - consider pain, anxiety, excess nitrates, large infarct.

Atrial fibrillation/atrial flutter may be precipitated by hypokalaemia (keep K⁺ 4.5 - 5.5 mmol) or hypomagnesaemia (give i.v. Mg²⁺, bolus 2g in 20mls N saline, 8 mmol, followed by 15g in 500mls 5% dextrose, 60 mmol, infusion over 12 hours). Keep Mg²⁺>1.0mmol/l

- if associated with haemodynamic collapse cardiovert.
- if need to return to sinus rhythm iv amiodarone (**via central line or long line only**)
- if need to control rate beta-blockers or verapamil with or without digoxin.

IV amiodarone may only be given via a peripheral vein in a cardiac arrest or peri-arrest situation. This applies to the bolus dose only – infusions must always be given by central or long line. Central access or a long line is required in anything other than cardiac arrest or peri-arrest scenario.

Ventricular tachycardia -

ALL BROAD COMPLEX REGULAR TACHYCARDIAS ARE VT (until proven otherwise)

Treatment:

1. If VT produces cardiac arrest then cardiovert as per VF resuscitation protocol.
2. If haemodynamically stable **and** diagnosis unclear give adenosine i.v. 12mg followed by 18mg if no effect
3. Ensure K⁺ 4.5-5.5 mmol and give magnesium (give i.v. Mg²⁺, bolus 2g in 20mls N saline, 8 mmol, followed by 15g in 500mls 5% dextrose, 60 mmol, infusion over 12 hours). Keep Mg²⁺>1.0mmol/l.
4. If haemodynamically stable try lidocaine 100 mg iv, then 50mg every 20 minutes to 200mg total.
If successful then give an infusion 2 mg/min for 2 hours then 1 mg/min thereafter.
Beware toxicity with abnormal neurological signs, fits, blurred vision, hypotension etc.
5. If lidocaine unsuccessful seek senior advice.

6. ICD patients – seek senior advice.

CASES REFRACTORY TO THE ABOVE MEASURES - SEEK ADVICE

Ventricular extra systoles - ignore

Non-sustained ventricular tachycardia – Beta blocker unless contra-indicated. Consider treatment with lidocaine (if haemodynamic compromise, frequent, symptomatic and poorly tolerated).

Polymorphic ventricular tachycardia – correct potassium and magnesium; seek advice.

Ventricular fibrillation (see resuscitation).

Idioventricular tachycardia - broad complex tachycardia rate 100-120. A form of parasystole competes with sinus rate, not associated with haemodynamic impairment, a sign of reperfusion, no treatment needed.

Management of Hyperglycaemia on CCU – proven or suspected ACS

1. Measure blood glucose on admission for all patients
2. If known diabetic or lab glucose/BM > 11 mmol/l, start IV insulin (or GKI(target glucose 6 – 10 mmol/l),
 - a. Send HbA1c
 - b. Light diet only
 - c. Stop if MI excluded
 - d. Avoid hypoglycaemia
 - e. Avoid over-treating oral controlled or newly diagnosed diabetic
 - f. Avoid stopping metformin on discharge – proven benefit in reducing CVD events: stop only if severe LVSD or creatinine > 150 µmol/l or eGFR < 35
 - g. Classify diabetes status as below
3. NOT KNOWN TO HAVE DIABETES
 - a. QDS “BM” – before meals and bed
 - b. Fasting lab glucose day after admission
 - c. Average glucose < 10 – follow-up with GP
 - d. Average glucose > 10 – refer to diabetes team: consider sc insulin
4. KNOWN DIABETIC – ALREADY ON INSULIN
 - a. Restart normal insulin regime after 24-48 h or when haemodynamically stable
 - b. QDS “BM” – before meals and bed
 - c. Average BM < 10 – normal follow-up
 - d. Average BM > 10 – refer to diabetes team
5. KNOWN DIABETIC ON > 2 ORAL AGENTS
 - a. Stop glitazone and/or sulphonylurea
 - b. Withhold metformin – restart once safe to do so (usually 48 h post angiogram)
 - c. Start sc insulin after 24-48h or when haemodynamically stable (when insulin infusion is stopped)
 - d. Refer to diabetes team : normal management will be 3/12 insulin then review
6. KNOWN DIABETIC ON DIET or METFORMIN only

- a. Continue diabetic diet – re-educate if needed
 - b. Withhold metformin – restart once safe to do so (usually 48 h post angiogram)
 - c. QDS “BM” – before meals and bed
 - d. Average BM < 10 – normal follow-up
 - e. Average BM > 10 – refer to diabetes team: consider sc insulin
7. INSULIN INFUSION REGIME
- a. Draw up 50 ml of actrapid or humulin S in 50 ml N saline
 - b. Start infusion at 3-6 units/h depending on blood glucose and patient size
 - c. Check BM every hour
 - d. Check potassium, on admission and then 12 hourly
 - e. If potassium < 3.5 or > 5 mmol.l, seek medical advice
8. INSULIN REGIME for new starters
- a. Basal bolus regime
 - b. Total daily dose = 50% of body weight
 - c. 50% given as basal insulin: Humulin I at 2200
 - d. 50% given as three equal mean time doses : Humulin S
 - e. Eg Body weight 80 kg : Rx Humulin I 20 units at 10 pm, Humulin S 7 units tds 20 min before meals

GUIDELINES FOR THE MANAGEMENT OF LIPID DISORDERS

Screening and treating lipid disorders should involve a multiple risk factor strategy. Modifiable risks include smoking, hypertension, obesity, diabetes mellitus, lack of exercise and inappropriate diet including excess alcohol. Unmodifiable risks include a family history of premature vascular disease or personal history of vascular disease, past or present. Drug treatment is of benefit mainly for secondary prevention (via GP) but is also indicated for primary prevention in high risk individuals (via GP) and for patients with severe familial hyperlipidaemias (via lipid clinic).

A PATIENT’S LIPIDS SHOULD BE MEASURED IF

they have vascular disease - angina, myocardial infarction, TIA, stroke or peripheral vascular disease

or

they have signs of hyperlipidaemia - xanthelasmata, xanthoma, premature arcus

or

they have 2 or more risk factors

- family history of premature vascular disease
(1st degree relative with angina or MI <55 years)
- hypertension
- diabetes mellitus
- smoking
- obesity (BMI >30)

MEASUREMENT OF LIPIDS

Cholesterol can be measured from a non-fasting sample and is sufficient to guide treatment in secondary prevention. For all other indications, a fasting sample should be sent for total cholesterol and triglycerides.

MANAGEMENT OF HYPERLIPIDAEMIA

All patients should be given general advice about diet, weight reduction, exercise and smoking. Secondary causes should be excluded by checking blood glucose, thyroid and liver function tests. Hypertension, diabetes and ischaemic heart disease should be treated as per local/regional guidelines.

SECONDARY PREVENTION OF CHD

- Statins are the treatment of choice for lowering cholesterol
- Fibrates are less effective than statins and are therefore not recommended
- Dietary advice is also recommended although trials indicate that diet alone reduces total cholesterol by only 1-5% and that compliance is generally poor.
- Simvastatin is first-line on the basis of long-term clinical outcome data and efficacy

1st line Simvastatin 40 mg od
2nd line Atorvastatin 20 mg od

In patients with ACS / MI, the routine use of atorvastatin 80mg is recommended. Should this dose prove intolerable, reduce dose rather than changing to alternative agent.

Before treatment

- Total serum cholesterol should be measured before initiation of a statin to establish a baseline figure.
- LFTs – it may be possible to initiate in patients with elevated LFT tests although additional LFT monitoring during treatment is necessary
- Warfarin interaction – the effect of warfarin is enhanced by simvastatin. Careful INR monitoring is therefore required
- Other interactions – see BNF

Monitoring during treatment

- LFTs - If simvastatin 40mg used, no routine monitoring required if normal pre-treatment. Stop treatment if serum transaminases rise to and persist at 3 x upper limit of normal.
- Aim for total cholesterol < 4.0mmol/l on therapy. Consider higher dose of more potent statin or additional agent if goal not met on simvastatin 40mg (e.g. atorvastatin, rosuvastatin, ezetimibe).
- Side effects. If CNS side effects on simvastatin, consider changing to atorvastatin
- Myopathy if diagnosed or is suspected and if creatine kinase is >10x upper limit of normal, the statin should be stopped

Dose titration

- Most patients can be initiated on 40 mg simvastatin od. Higher doses of statins (eg 80 mg simvastatin) may be indicated for specific lipid syndromes (eg familial hypercholesterolaemia) under the care of a cardiologist or lipid clinic. The priority is to ensure all secondary prevention patients receive at least 40mg Simvastatin or equivalent.
- If total cholesterol >4mmol/l or LDL cholesterol > 2mmol/l on at least 40mg of simvastatin, consider more potent lipid lowering therapy. Options include the addition of ezetimibe to simvastatin 40mg or a change to more potent statins such as atorvastatin or rosuvastatin. Discuss with consultant.

- Counselling. Patients should be counselled to seek medical advice and stop statin treatment if unexplained persistent generalised muscle pain develops
- Common side-effects are GI, altered LFTs and muscle aches

Coast to Coast Cardiac Network

ICD Implant Guidelines

Implantable Cardioverter Defibrillators (ICDs) are of proven benefit in terms of reducing sudden cardiac death in a number of patient groups. NICE guidance has been published with respect to many of these groups, however, there are as yet some situations that have not been covered by NICE. At a recent meeting of the Network clinical sub-group it was agreed by those present that we would modify the NICE guidelines for use within our Network as outlined below.

ICDs ARE RECOMMENDED FOR PATIENTS IN THE FOLLOWING CATEGORIES:

Secondary prevention – that is, for patients who present, in the absence of a treatable cause, with one of the following:

- having survived a cardiac arrest due to either ventricular tachycardia (VT) or ventricular fibrillation (VF)
- spontaneous sustained VT causing syncope or significant haemodynamic compromise
- sustained VT without syncope or cardiac arrest, and who have an associated reduction in ejection fraction (LVEF of less than 35% or equivalent (moderate to severe LV dysfunction)), no worse than NYHA class III

Primary prevention – that is, for patients who have:

a history of previous (more than 4 weeks) myocardial infarction **or cardiomyopathy** and:

EITHER

- left ventricular dysfunction with an LVEF of less than 30% or equivalent (severe LV dysfunction), no worse than NYHA class III and QRS duration of ≥ 120 ms. **PATIENTS IN THIS GROUP CAN BE REFERRED DIRECTLY FOR CONSIDERATION OF ICD IMPLANT**

OR

- left ventricular dysfunction with an LVEF of less than 35% (moderate to severe LV dysfunction), no worse than NYHA class III and non-sustained VT (4 or more beats at a rate of 120bpm or more (cycle length <500ms)) on Holter monitoring. **PATIENTS IN THIS GROUP SHOULD BE REFERRED FOR CONSIDERATION OF A VT INDUCTION STUDY**

Other patients suitable for consideration of an ICD for primary prevention include those with a familial cardiac condition with a high risk of sudden death, such as long QT syndrome, hypertrophic cardiomyopathy, Brugada syndrome or arrhythmogenic right ventricular dysplasia (ARVD), or those who have undergone surgical repair of congenital heart disease.

ARRHYTHMIAS NOT ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTION

Tachycardia. - 12 lead ECG needed for diagnosis.
- haemodynamic state prime determinant of management.

Irregularly irregular - atrial fibrillation rates 100-180.
if rates 200-300 with broad complexes, consider WPW (pre-excitation).

Irregular broad complexes no cardiac output.
- polymorphic VT - seek advice (after resuscitation).

Regular rate 140-160 narrow complex, usually atrial flutter with 2:1 AV block.
look for 'saw-tooth' flutter waves in leads II, III and aVF
rate 160-200 narrow complex, usually = SVT
rate 160-200 broad complex, usually = VT.

Atrial fibrillation.

Consider the causes e.g. thyrotoxicosis, alcohol, mitral valve disease.

If associated with haemodynamic collapse - synchronised DC cardioversion.

If return to sinus rhythm is desirable/feasible then use amiodarone oral (iv via central or long line).
Other anti-arrhythmics such as sotalol, propafenone, flecainide or procainamide should be used under guidance of a consultant cardiologist.

If control of ventricular rate only needed, use β -blockers or verapamil (+/- digoxin)

If persistent > 24 h, consider heparin.

If chronic consider warfarin.

If paroxysmal consider β -blocker, flecainide, or amiodarone. Anticoagulation with either aspirin or warfarin depends on risk profile for stroke – seek senior advice. Difficult cases should be referred for specialist advice – ablation procedures may be indicated.

If rapid ventricular response refractory to pharmacology, consider AV nodal ablation and permanent pacemaker insertion.

Atrial flutter.

Adenosine may slow ventricular rate to unmask flutter waves and assist diagnosis. As atrial fibrillation but drug therapy to slow rate often unrewarding. Low threshold for cardioversion. Consider overdrive pacing and ablation of flutter circuit.

SVT

Supraventricular tachycardia encompasses atrial tachycardias, AV nodal re-entrant tachycardias, AV re-entrant tachycardia (as seen in Wolff-Parkinson-White syndrome). Careful review of a 12 lead ECG will often clarify the diagnosis. Adenosine may convert to sinus rhythm and/or reveal the diagnosis.

Record ECG during administration of adenosine. (great value in precise diagnosis).

Consider curative ablation rather than chronic drug therapy for the long term management.

VT obtain 12 lead ECG unless cardiac arrest

- if haemodynamically unstable - cardioversion.

- if haemodynamically stable: (consider adenosine with 12 lead ECG)

try lidocaine 100 mg iv - if unsuccessful, seek senior advice.

if lidocaine successful give infusion.

if VT not occurring in setting of acute MI then seek advice on further management.

ADMISSION TO CCU OF PATIENT WITH DRUG OVERDOSE

Patients having taken a drug overdose will need CCU care if

a) they have a serious bradycardia requiring intervention e.g. complete heart block needing pacing

b) they have a serious tachycardia needing intervention e.g. polymorphic VT

c) they are at high risk of a serious arrhythmia eg a tricyclic overdose with a persistent sinus tachycardia of ≥ 120 bpm; eg digoxin overdose

Patients at low risk of arrhythmia can be managed on a medical ward with CCU telemetry or bedside monitoring. Others signs of overdose eg impaired conscious level are not an indication for CCU admission.

DEATHS ON CCU

It is important that patient's GP and/or referring hospital/physician are informed as soon as possible that a patient has died. It is the responsibility of the cardiology SHO on duty for CCU to contact by phone the patients GP and the referring hospitals/physicians within 24 hours of the patient's death. For patients dying over a week-end GPs etc should be contacted on Monday morning.

Please record the date and time of informing the patient's GP in the notes and also if the GP (name) has been informed directly or if a message has been left with a receptionist (name).

If a patient dies within 24 hours of admission or following a cardiac intervention (PCI, pacing, IABP) case should normally be discussed with the coroner - all such deaths should be discussed with Consultant responsible for case as soon as practicable and *always* before contacting Coroner. The Coroner's Office is now staffed during office hours at weekends.

RESUSCITATION STATUS

See trust DNAR policy.

The responsibility for issuing a DNAR order rests with the Consultant or Specialist Registrar, Staff Grade or Associate Specialist responsible for the patient. The DNAR status form must be completed and signed by the by the Consultant in charge of the patient following discussions with the patient/family/other appropriate representative as feasible. For emergency admissions, the form may be completed and signed by a middle grade or equivalent doctor acting as the Consultants authorised deputy and the decision **MUST** be reviewed and signed by the Consultant within 24 hours. The form should not be completed by the House Officer or by a Senior House Officer of less than 2 years experience.

Resuscitation status must be documented on Trust DNAR status sheets and filed in the case sheet. Decisions regarding resuscitation attempts should be binary i.e. attempt or do not attempt CPR. Uncommonly, the consultant in charge may consider an attempt at resuscitation appropriate but ITU care inappropriate. In these uncommon cases, this directive should be documented in the case sheet.

TERMINALLY ILL PATIENTS

It may be inappropriate for some patients in CCU to continue to be managed aggressively because of terminal prognosis (e.g. advance heart failure with no other therapeutic option available). Decisions regarding this change in management status should always be made at consultant level (or SpR if out of hours – to be reviewed within 24 hours by consultant). In these cases, refer to End of Life Care Pathways for guidance on pain control, agitation etc. Guidelines are available via Pathfinder on Trust Intranet or contact Macmillan/Specialist Palliative Care Team for advice.

Where patients have an ICD in situ and have been assigned to palliative treatment, the ICD should be de-activated. During working hours, the pacing / device team in CIU should be contacted and the device re-programmed. Out of hours or in an emergency, ICDs may be de-activated by placing a magnet over the generator. The magnet may be secured in place with an appropriate adhesive dressing until the device can be formally de-activated during working hours.

DEATH IN PATIENT WITH IMPLANTABLE LOOP RECORDER

Some patients with unexplained syncope have an Implantable Loop Recorder (ILR) fitted to elucidate the cause of symptoms. In the unusual circumstance that a patient with an ILR in situ has a cardiac arrest or sudden death, it is important to have the device interrogated to ascertain any arrhythmic cause. This may be important in estimating risk for other family members even if the patient does not survive the arrest. Contact the pacing / device team in CIU to download the information. Out of hours, the body **should not** be released from the hospital for burial / cremation until the device is interrogated.