



One Heart

Course Handbook

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Introduction

One Heart is devised by trainees who have completed their cardiac rotations and is supported by cardiac anaesthetists within their specific fields of expertise. The course is designed to provide the core knowledge and practical skills which are unique to Cardiac anaesthesia and Cardiac ICU (CICU). The ultimate aim is to improve candidates' confidence in the management of commonly-encountered clinical problems. The course is designed to supplement the RCoA/FICM cardiac anaesthesia and ICU specialty training curriculums.

Pre-course reading and small workshops provide the knowledge required to underpin the successful management of patients. High-fidelity simulation scenarios are employed to reinforce the knowledge, skills and attitudes, including human factors, required for safe practice. There is a strong emphasis on the importance of team-working.

One Heart simulation scenarios are run in a slightly different manner to other comparable simulation courses. A 'typical' patient 'journey' is followed through their cardiac surgery pathway by all candidates. Each candidate will have multiple opportunities to practice the core skills and fulfill different roles within the team. There is a specific focus during this 'journey' on specific areas of cardiac anaesthesia and ICU that are commonly misunderstood, poorly practiced or unlikely to have been encountered prior to cardiac rotations. These include:

Temporary Epicardial Pacing (Pacing Boxes)

Thromboelastograph (TEG)

Heparin, Activated Clotting Time (ACT) and Protamine

Post-Operative Bleeding

Cardiopulmonary Bypass

Cardiac Tamponade and Resternotomy

Timetable

0815 - Registration

0830 - Introductory Lecture

0845 - Pre-course Quiz / Assessment

0915 - Fundamentals of Cardiac Anaesthesia

1000 – Sim / Manikin Familiarisation

1015 - The Postoperative Cardiac Patient

1100 – COFFEE (change in to scrubs)

1120 - Pacing Tutorial – S Underwood

1210 - Simulation Scenario 1

1240 – LUNCH

1310 - Simulation Scenario 2

1350 - Simulation Scenario 3

1430 - Simulation Scenario 4

1510 – COFFEE

1525 - Simulation Scenario 5

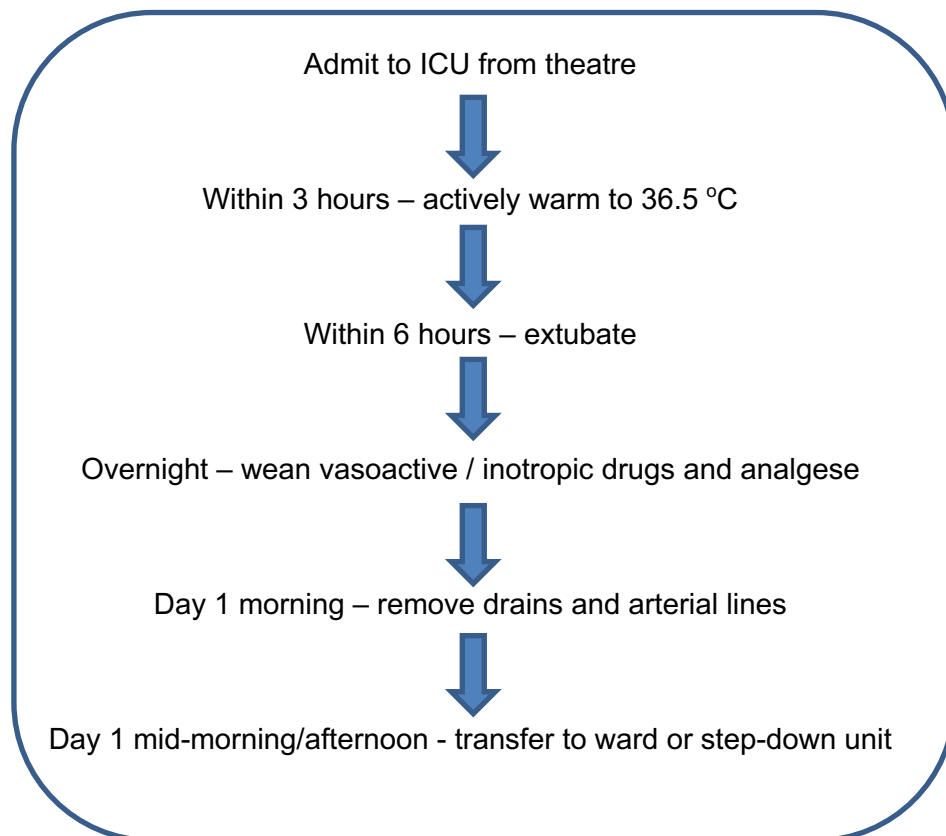
1605 - Post-course Quiz

1630 - Summary and Feedback

1645 - Close

Normal Post-Operative CICU Timeline

Cardiac surgery encompasses a number of operations and a variety of patient types. However, many of the objectives in the post-operative period are common to all and are suitable for protocol driven care by ICU nursing staff. Finer details vary between centres but the general principles are universal across the United Kingdom. The overall ICU goal after uncomplicated surgery is to discharge patients to the ward or step-down care within 24 hours. Protocols outlined below are those used in the Bristol Heart Institute.



Problems Requiring Senior Involvement

Cardiac anaesthesia is a consultant-led and consultant-delivered specialty and therefore the following should definitely be discussed with the responsible anaesthetist:

- Suspected tamponade
- Post-op bleeding requiring blood products or possible return to theatre
- Haemodynamic instability not responsive to initial measures
- Haemodynamic problems with congenital heart disease patients
- Decisions about treatment limitations or withdrawal of treatment
- Respiratory failure requiring re-intubation
- New admission to ICU

Post-Operative Ventilation

Mechanical ventilation is continued from the operating theatre according to protocol and then adjusted to maintain the pH, P_aCO_2 and P_aO_2 within normal limits for the patient. Often the anaesthetist will set the ventilator parameters prior to leaving CICU. Generally, patients will initially be commenced on:

BiPAP or SIMV-VC
 FiO_2 0.50
 V_T 6mls/kg
 PEEP 5cmH₂O

There is frequently a pre-induction arterial blood gas for comparison. Those with a balanced circulation and reactive pulmonary vasculature require extra care to maintain these close to normal to avoid profound falls in cardiac output and hypoxia.

Weaning Ventilation and Extubation

For the uncomplicated cardiac surgical patient, ventilator weaning and extubation is protocol driven by the ICU nurse, without any specific intervention from medical staff.

Sedation is stopped when:

- The patient is haemodynamically stable on little or no inotropic support
- There is no metabolic derangement (pH within normal range, BE < -3, serum lactate <2)
- Core temperature >36.5°C
- There is adequate gas exchange (FiO_2 <0.50, P_aO_2 >75mmHg (10kPa), P_aCO_2 <53mmHg (7kPa))
- Chest drainage is <100mls/hr for 2 consecutive hours

Nurse-led extubation criteria:

- Patient awake, neurologically intact and obeying commands
 - Equal hand grip
 - Arm raise to shoulder height
 - Able to lift head off pillow
- Adequate analgesia
- BM, K^+ and Na^+ normal
- Warm, well perfused and minimal inotropic support
- Respiratory rate 10 – 30/minute
- ABGs acceptable:
 - pH 7.35
 - $PaCO_2$ 35-49mmHg
 - PaO_2 >90mmHg
 - BE -3 to +3
- Spontaneous tidal volume (V_T) >6ml/kg
- PEEP 5cmH₂O
- Pressure support <10cmH₂O
- FiO_2 <0.50

Patients should be extubated onto humidified oxygen, which should be continued for 48-72 hours to prevent desaturation. Patients should be encouraged to deep breathe and cough to aid in lung recruitment and prevent sputum retention.

Post-Cardiac Surgery Respiratory Failure

The major determinant of outcome is cardiac function. However some degree of respiratory failure is common (up to 20% of patients) following cardiac surgery and is a continuing source of morbidity and mortality.

Pathology ranges from sputum retention and atelectasis to Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS). Common causes of respiratory failure after cardiac surgery are shown below:

Common causes of respiratory failure after Cardiac Surgery
Acute Lung Injury
Pump lung – inflammatory response following CPB
Altered lung mechanics following sternotomy
Atelectasis
Pneumonia
Effusion – including haemothorax
Pneumothorax
Cardiac dysfunction and pulmonary oedema
Intrinsic pulmonary pathology

Treatment of respiratory failure is essentially supportive and is aimed at reaching the following goals:

Goals for Treating Respiratory Failure
SaO ₂ > 90% / PaO ₂ > 7kPa
pH > 7.25
RR < 30
Ability to speak in short sentences
Ability to protect airway
Haemodynamic stability

The above goals can be achieved using a stepwise progression through supplemental oxygen -> non-invasive ventilation -> tracheal intubation and invasive ventilation.

Ventilation should follow a lung protective strategy:

FiO₂ < 0.6
 V_T < 6ml/kg
 Permissive hypercapnia
 Minimise peak airway pressure (<35cmH₂O ideally)

Post-operative ECG Changes

ST segment changes are not unusual in the post-op period, particularly with off-pump surgery. The following associations in the context of ST changes need further consideration and investigation:

- Haemodynamic compromise
- Chest pain
- Changes in regional pattern

Vasoactive Drugs and LV Dysfunction

Vasoactive drugs are either usually titrated to haemodynamic parameters or may be maintained at a constant level for a set period. Cardiac output falls immediately after cardiac surgery with cardiopulmonary bypass (CPB), but usually returns to normal over 24 hours when pre-operative LV function is normal. In those with impaired LV function, this may be prolonged.

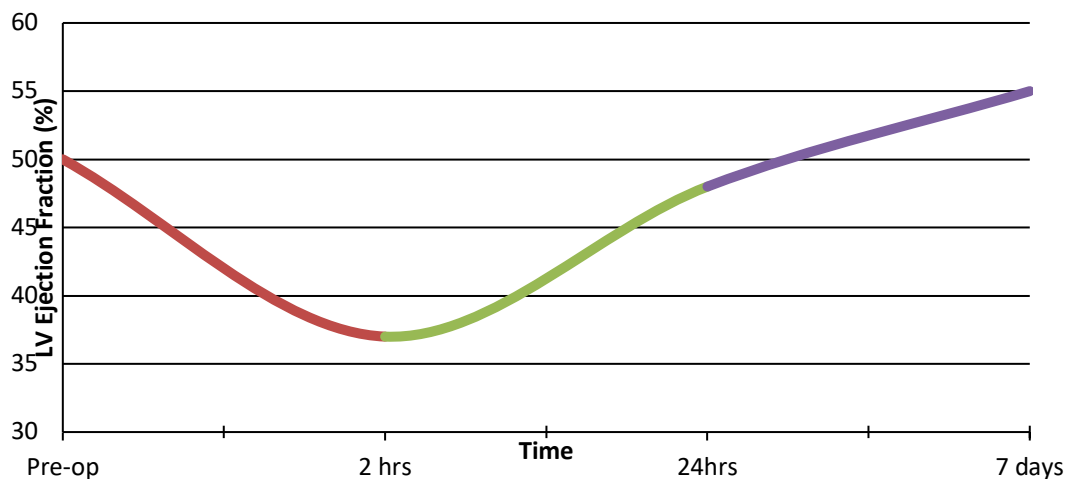


Fig 2. LV ejection fraction following 'on-pump' coronary artery surgery. Adapted from Roberts et al (*Ann Thorac Surg.* 1983 Feb; 35(2): 208-25).

LV dysfunction can occur for a number of remediable reasons and these should be corrected before starting vasoactive medicines. Common causes of LV dysfunction after cardiac surgery are shown below:

Common Reasons for Post-Cardiac Surgery LV Dysfunction
Metabolic abnormalities
Low Ca ²⁺ due to citrated blood
Myocardial ischaemia (thrombosis, occlusion, vasospasm)
Beta-blockers
Prolonged CPB times (>2 hours)
Myocardial stunning from ischaemia-reperfusion injury

The approach to the patient with post-operative LV dysfunction is the same as optimising any patient's cardiac output. Namely, to optimise:

- By remove any precipitating factors
- HR 80-100bpm -> pace if needed
- Rhythm -> maintain sinus rhythm or AV sequential pacing
- Pre-load -> fluid challenge to assess responsiveness
- Afterload -> reduce vasopressors or use of an intra-aortic balloon pump
- Contractility -> using inotropic drugs/intra-aortic balloon pump or ventricular assist devices

RV Dysfunction

RV dysfunction is difficult to manage due to 'ventricular interaction'. Essentially, the failing RV 'bulges' into the LV and impairs LV function and so cardiac output. RV failure accounts for about 20% of circulatory failure after cardiac surgery.

- **Systolic Dysfunction**
 - Myocardial stunning
 - Myocardial ischaemia / infarction
- **RV Volume Overload**
 - Excess fluid administration
 - PV Regurgitation
 - TV Regurgitation
 - ASD
- **RV Pressure Overload**
 - Hypoxia
 - Hypercarbia
 - Acidosis
 - Over-PEEP
 - Acute Lung Injury / ARDS
 - Pulmonary embolus
 - Chronic pulmonary disease
 - PV Stenosis
 - Pulmonary vascular occlusive disease

Fig 4. Causes of post-operative RV dysfunction.

Diagnosis is based on a rising CVP in the face of falling blood pressure and cardiac output. Echocardiography is useful to confirm the diagnosis and exclude tamponade.

Treatment is based on optimisation of:

- **Removal / reversal of precipitating causes**
 - Thromboembolectomy / thrombolysis
 - Relief of Tamponade (often manifests as RV dysfunction)
- **Heart rate** – optimising
- **Heart Rhythm** – maintain sinus
- **Maintaining adequate pre-load**
 - Do not aggressively fluid load patients with RV failure. It rarely improves either cardiac output or blood pressure and reduces the RV perfusion pressure by increasing RVEDP.
- **Maintaining RV perfusion pressure**
 - Intra-aortic balloon pumps, although not specifically indicated for RV failure, will improve RV perfusion pressure and thus may help to support the RV as a bridging method.
- **Augmenting contractility**

- Inotropes will both improve RV perfusion pressure and augment contractility.
- An inodilator (dobutamine / milrinone / enoximone) in combination with low-dose noradrenaline reduces RV afterload as well as improving both contractility and blood pressure.

- **Reducing RV afterload**

- Simple measures such as reversing hypoxia, hypercapnia, acidosis and reducing PEEP to the lowest possible level are often extremely effective.
- Specific treatments include nitric oxide, sildenafil, as well as nebulised or intravenous prostacyclin.

Cardiac Tamponade

This is characterised by falling blood pressure and cardiac output in the face of a rising CVP and tachycardia and is notoriously difficult to diagnose in the post-cardiac surgical patient. It can develop rapidly within the first 24hrs after cardiac surgery, although tamponade physiology can occur in a sub-acute fashion over several days. The physiology, diagnosis and treatment of surgical tamponade differs from medical tamponade for the reasons listed below:

- Rapid onset
- Ability for even small amounts of blood to cause localised compressive effects and significant haemodynamic compromise. This makes for difficult echocardiographic diagnosis. A *negative* TTE or TOE examination should **NOT** prevent return to theatre for suspected tamponade.
- Tamponade due to non-pericardial causes such as oedema of the thoracic structures.
- There is often a period of short-lived fluid responsiveness, which can make distinguishing tamponade from hypovolaemia difficult.

Treatment of tamponade is aimed at maintaining cardiac output. In the post-surgical population return to theatre is required. Percutaneous drainage is not indicated because of the need to identify and control the source of bleeding and because percutaneous removal of localised tamponade is unreliable. If you suspect tamponade liaise with the consultant anaesthetist for CICU and the cardiothoracic surgery registrar.

Post-Operative Bleeding

Post-operative bleeding is a common problem after cardiac surgery. The Cardiac surgical units haematocrit transfusion trigger is 0.23 (Hb 7-8). However, in the bleeding patient it is prudent to maintain the haematocrit around 0.30 (Hb 10), as a level around this is required for maximal clotting. Causes of postoperative bleeding:

- Pre-operative drug induced platelet dysfunction (eg aspirin / clopidogrel)
- Residual heparinisation (including 'pump blood' transfusions)
- CPB induced coagulopathy / fibrinolysis (worse with prolonged CPB time)
- 'Surgical bleeding' – sternal wires / anastomoses / graft side branches

Bleeding should be aggressively treated and investigated along the following lines:

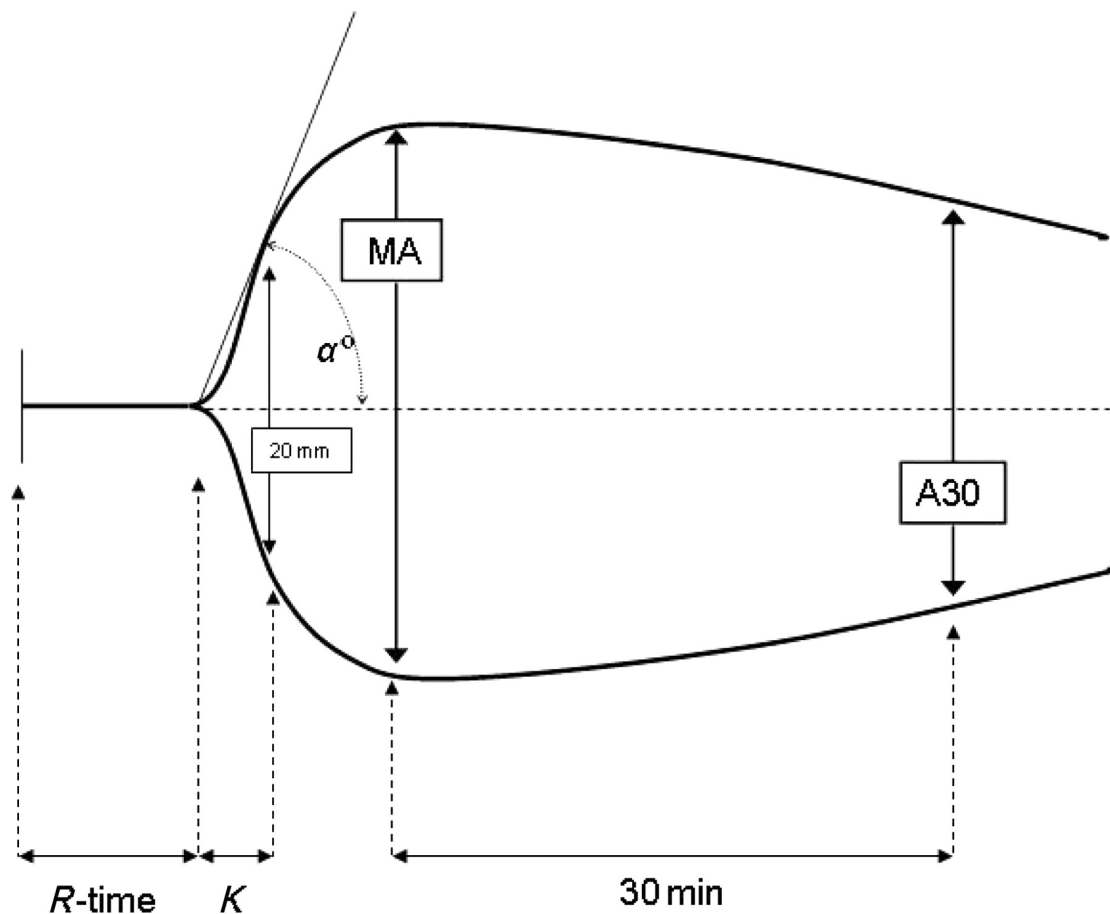
- Appropriate clotting tests: FBC, APTT, PT, Fibrinogen (Fibrinogen must be requested separately to clotting)
- Red cell transfusion to maintain haematocrit
- Transfusion of blood components guided by
 - Coagulation screen
 - Thromboelastograph (TEG) a near patient testing device which provides a dynamic assessment of clot strength. Clopidogrel and aspirin do not affect the TEG and so empiric treatment with platelets will help overcome this problem.
 - Platelets are the most common product used due to CPB induced dysfunction.
 - FFP is often transfused unnecessarily, but *is* useful after long bypass times and in those with pre-existing coagulopathy (warfarin, liver disease).
 - Cryoprecipitate is guided by a fibrinogen assay (Fibrinogen < 1.5). Activated Factor VII is used off licence for patients with life threatening bleeding refractory to other treatments.
- Administration of drugs
 - Protamine: The half-life of heparin is significantly longer than that of protamine and therefore there can often be a degree of residual heparinisation, particularly if the 'pump blood' (blood salvaged from the CPB circuit) is given back to the patient
 - The Cardiac surgical unit often gives anti-fibrinolytics (usually tranexamic acid) as a routine, but if these have not been previously given then a single dose or infusion may be helpful
 - Aprotinin in cardiac surgery has essentially stopped due to an excess mortality in those in whom it was used.
- Surgical re-exploration where appropriate. Approximately 2 - 5% of patients need re-exploration for bleeding and this is associated with a significant increase in morbidity and mortality. As a general rule, the following chest drain losses should prompt re-exploration:
 - >400mls in the first hour
 - >200mls in 2 consecutive hours
 - >100mls in 4 consecutive hours

Other factors to consider include

- Maintaining SBP around 100 – 120mmHg
- Restoration of normothermia
- Addition of PEEP – may reduce bleeding by increasing mechanical intrathoracic pressure, but may also reduce venous return and thus cardiac output in those who are hypovolaemic.
- A chest x-ray or TOE may show large pleural / pericardial collections which necessitate immediate return to theatre regardless of coagulation status.
- If the patient is bleeding – do not extubate them until it has stopped.

Thromboelastograph (TEG)

The thromboelastograph (TEG) is a machine that gives a dynamic measure of clot strength. Because it uses whole blood, you get an assessment of the interactions between platelets, clotting factors and the fibrinolytic system. Whole blood is placed in a cuvette and a pin is lowered into it. Rotational force is applied to the pin and the torque transmitted from the pin to the blood is measured. Initially, there is no torque transmitted as blood is liquid, then a clot forms and the blood and pin move together. This reaches a maximum point and then there is fibrinolysis with the clot becoming less solid and a consequently smaller amount of torque. Two cuvettes may be run at the same time – one with heparinase, which removes the effect of heparin and the two traces may be superimposed. This allows you to see how much effect may be reduced by protamine. Various points in the trace can be quantified.

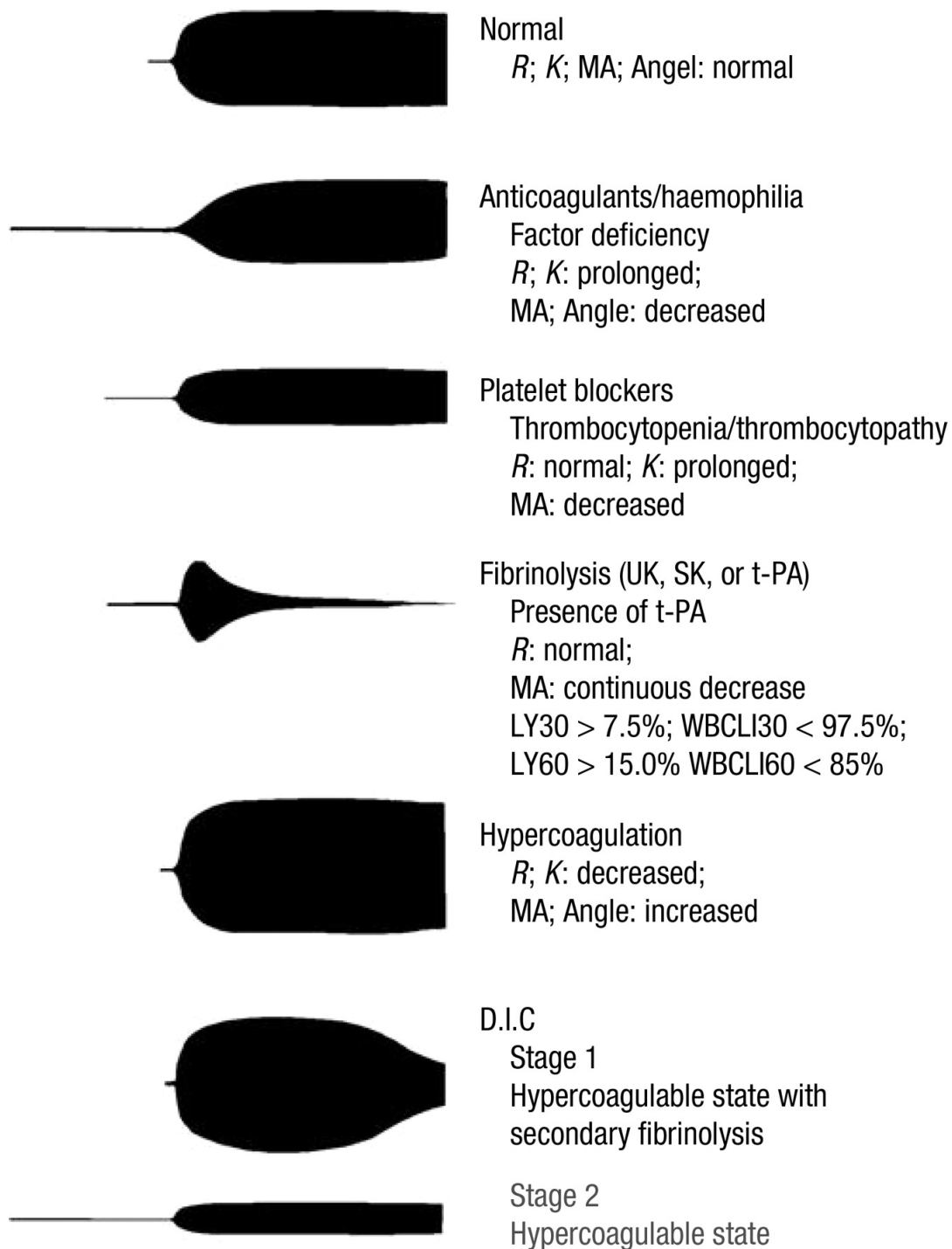


- **R- time (reaction time)** – Time from trace start until amplitude reaches 1mm. Prolonged by anticoagulants including heparin and clotting factor deficiencies.
- **K- time** – Time from initiation of oscillatory movement until amplitude reaches 20mm. Prolonged by thrombocytopenia and low fibrin levels.
- **α-angle** – The angle from R to K. Reduced by thrombocytopenia and low fibrin levels.
- **R + K** – The sum of the R and K-time represents whole blood clotting time.
- **MA** – Maximum amplitude. Represents the point at which the rotational torque is maximum. Reduced by platelet dysfunction, low fibrin levels and heparin.
- **A₃₀** – The rotational torque 30 minutes after MA is reached. It is a measure of clot stability and lysis.

Although the BHI does not have a protocol for treatment of clotting abnormalities seen on a TEG. The following may help you when you are starting out:

- MA < 48mm – give 1 bag of Plts
- MA < 40mm – give 2 bags of Plts
- R 14-21 (with *identical* heparinise trace) – Give 1 FFP
- R 21-28 (with *identical* heparinise trace) – Give 2 FFP
- R > 28 (with *identical* heparinise trace) – Give 4 FFP
- R > 14 (with *normal* heparinise trace) – Give Protamine

Common patterns of clotting dysfunction are shown below:



Anticoagulation

Almost all patients will have a degree of residual coagulopathy on the day of cardiac surgery. The incidences of DVT and PE after cardiac surgery are around 20% and 0.5% respectively. Prevention follows NICE guidelines (*NICE CG92 – venous thrombo-embolism. March 2010*):

Offer VTE prophylaxis to patients undergoing cardiac surgery who are not having other anticoagulation therapy and are assessed to be at increased risk of VTE.

Start mechanical VTE prophylaxis at admission. Choose any one of:

1. *anti-embolism stockings (thigh or knee length)*
2. *foot impulse devices*
3. *Intermittent pneumatic compression devices (thigh or knee length).*

Continue mechanical VTE prophylaxis until the patient no longer has significantly reduced mobility.

Add pharmacological VTE prophylaxis for patients who have a low risk of major bleeding, taking into account individual patient factors and according to clinical judgement. Choose one of:

1. *LMWH*
2. *Unfractionated Heparin (for patients with renal failure).*

Continue pharmacological VTE prophylaxis until the patient no longer has significantly reduced mobility (generally 5–7 days).

CABG

Aspirin 300mg PR is prescribed after bleeding has settled on the evening of surgery and daily (300mg OD) thereafter. Some surgeons use clopidogrel after 'Off-pump' surgery – or in the presence of difficult anastomoses.

Valve Surgery

Oral anticoagulation is usually started on the first post-operative day. Since most patients have a degree of coagulopathy after surgery, there is usually no need to use heparin until the therapeutic range is reached, aside from routine thromboprophylaxis.

The first post-operative month is the highest risk for valve thrombosis – so falls below the target INR should be avoided. ESC guidance on anti-coagulation for prosthetic valves is listed below:

Each surgeon at the BHI has their own protocol.

Common valve anticoagulation protocols are:

- Bio AVR – Aspirin 75mg OD from Day 1
- Mech AVR – Warfarin from Day 1
- MVR / MV Repair – Warfarin from day 1

Mitral valve repairs require warfarin in the immediate post-operative phase only. This may be discontinued at 6 weeks to 3 months.

Warfarin

Patients who have had cardiac surgery will almost always have a degree of inherent coagulopathy. They should therefore **NOT** be prescribed warfarin in the usual '10, 10, 5' loading manner as this will result in overshoot of the INR. A starting dose of 5mg per day for the first two days is adequate for those with a normal INR. Patients who were previously on warfarin prior to admission can simply be restarted on their pre-admission dose without loading.

Oral anticoagulation is recommended in the following situations:

- Lifelong for all patients with mechanical valves
- Lifelong for patients with bioprostheses who have other indications for anticoagulation, e.g. atrial fibrillation, or with a lesser degree of evidence; heart failure and impaired LV function (EF <30%).
- For the first 3 months after insertion in all patients with bioprostheses (target INR 2.5). However, there is widespread use of aspirin (low dose: 75–100 mg) as an alternative to anticoagulation for the first 3 months, but no randomized studies to support the safety of this strategy.

Medication Concentrations for the Cardiac Intensive Care Unit (Bristol Heart Institute)

Medication	Diluent	Composition (Total Volume)	Concentration	Notes	Normal Dose
Dopamine	5% Glucose	200mg in 50ml	4mg/ml	Note 1	2.5 – 15 mcg/kg/min
Dobutamine	5% Glucose	250mg in 50ml	5mg/ml		5-15 mcg/kg/min
Adrenaline	5% Glucose	5mg in 50ml	100micrograms/ml	Note 1	0.05-0.2 mcg/kg/min
Noradrenaline	5% Glucose	4mg in 50ml	80micrograms/ml	Note 1	0.05-0.2 mcg/kg/min
Enoximone	Sodium chloride 0.9%	100mg in 40ml	2.5mg/ml		1-5 mcg/kg/min
Vasopressin	5% Glucose	40units in 40ml	1 unit/ml		0.01-0.04 units/min
Levosimendan	5% Glucose	12.5mg in 250ml	50micrograms/ml	Note 2	0.05-0.2 mcg/kg/min
Isoprenaline	5% Glucose	5mg in 50ml	100micrograms/ml		0.01-0.15 mcg/kg/min
GTN	Neat	50mg in 50ml	1mg/ml		0.5-5mg/hr
SNP	5% Glucose	50mg in 50ml	1mg/ml		0.5-4.0 mcg/kg/min
Milrinone	5% Glucose	20mg in 50ml	0.4mg/ml		0.375-0.75 mcg/kg/min
Esmolol	Neat	2500mg in 250ml	10mg/ml		50-300 mcg/kg/min
Labetalol	Neat	250mg in 50ml	5mg/ml		20-50mg/hr
Morphine	Sodium chloride 0.9%	50mg in 50ml	1mg/ml		0.5-5mg/hr
Alfentanil	Neat	25mg in 50ml	0.5mg/ml		1-4mg/hr
Remifentanil	Sodium chloride 0.9%	5mg in 50ml	100micrograms/ml		0.1-0.3 mcg/kg/min
Midazolam	Neat	50mg in 50ml	1mg/ml		1-6mg/hr
Propofol	Neat	500mg in 50ml	10mg/ml		50-150mg/hr

Amiodarone (300mg loading)	5% Glucose	300mg in 50ml	6mg/ml		Over 1 hour
Amiodarone (900mg loading)	5% Glucose	900mg in 50ml	18mg/ml		Over 24hour
Amiodarone (600mg maintenance)	5% Glucose	600mg in 50ml	12mg/ml		Over 24 hour
Furosemide	Neat	500mg in 50ml	10mg/ml		1-20mg/hr
Aminophylline	Sodium chloride 0.9%	250mg in 50ml	5mg/ml		0.5mg/kg/hr
Actrapid Insulin	Sodium chloride 0.9%	50units in 50ml	1unit/ml		As per protocol
Heparin	Sodium chloride 0.9%	20000units in 40ml	500units/ml	Note 3	As per protocol
Clonidine	Sodium chloride 0.9%	750micrograms in 50ml	15micrograms/ml		0.5-2.0 mcg/kg/hr
Epoprostenol	Sodium chloride 0.9%	100micrograms in 50ml	2micrograms/ml		As per protocol

Notes:

1. If high doses x2 or x4 strength can be used, but must be clearly labelled
2. Refer to the CICU Levosimendan protocol for dose/administration etc.
3. Refer to the BRI Heparin protocol.

Levosimendan

Levosimendan is one of a class of inotropes that works through the novel action of calcium sensitization. Unlike other inotropes, it increases myocardial contractility without increasing myocardial oxygen demand. It has also been shown to exhibit anti-ischaemic effects mediated via the opening of sarcolemma & mitochondrial ATP-sensitive potassium channels.

Two recent meta-analyses have demonstrated that in cardiac surgical patients levosimendan improves mortality, hospital length of stay, post-op cardiac troponin release, and incidence of post-op atrial fibrillation.

Indications:

1. Acute post-operative ventricular failure refractory to other inotropes, or where arrhythmic/chronotropic effects of other inotropes are not tolerated – where there is reasonable expectation that the ventricular performance will improve.
2. High risk cardiac surgery patients (LVEF <30%, and euroscore >6), undergoing on-pump surgery.

NB The decision to start Levosimendan must be made by a consultant cardiac anaesthetist/intensivist. The cost of levosimendan is £910/12.5mg ampoule.

Preparation and administration:

- Available as 12.5 mg in 5 mL vials (concentration of 2.5 mg per mL).
- Levosimendan should be diluted with dextrose 5% to a final concentration of 0.05 mg /mL (= 50 mcg /mL) i.e. One 5 mL vial should be added to 250 mL dextrose 5%
- Preparation is stable for 24 hours once diluted and may be administered via Central or Peripheral routes.

Dosage:

Loading Dose: 12-24 mcg/kg over at least 10mins may be given, but may cause haemodynamic instability.

Maintenance: 0.05-0.2 mcg/kg/min for a maximum of 24 hours
Commence at 0.1mcg/kg/min & titrate up/down as necessary

Additional Monitoring:

All patients must have arterial & central venous pressure monitoring.

Prior to starting Levosimendan:

1. Echocardiogram to document left & right ventricular performance
2. Insert PA catheter
3. Document PAWP, cardiac index, stroke volume index, SVR

During Levosimendan infusion:

1. Document PAWP, cardiac index, stroke volume index, SVR every 4 hours

After discontinuation of infusion:

1. Repeat echocardiogram if practical

Storage:

Levosimendan is stored in a refrigerator at 2-8°C

Cautions:

It should be noted that approximately 5% of a levosimendan dose leads to the formation of an active metabolite (ORG 1896), with a plasma half-life of around 80 hours. ORG 1896 may accumulate in renal failure, the clinical implications of which are unknown at present.

Frequent adverse events:

(Incidence of between 1 in 100 and 1 in 10)

- Decrease in haemoglobin
- Hypokalaemia
- Dizziness and headache
- Hypotension
- Myocardial ischaemia (2%)
- Extrasystoles (1.3%), AF (1.4%), atrial tachycardia (2.4%) and ventricular tachycardia (1%)
- Nausea and vomiting

Temporary Epicardial Pacing

- Epicardial pacing wires are sited by some surgeons for all their patients. Other surgeons will only insert them when specifically indicated e.g. valve surgery where there is often transient dysfunction of the AV node or conducting system, due to direct injury.
- Generally atrial wires are brought out on the right side of the chest and ventricular wires are brought out on the left.
- Pacing wires are usually removed on Day 4 post-op.
- Patients who still require pacing 5 days post-op should be considered for a permanent pacemaker by the Cardiology team.
- Heparin should be stopped 4 hours prior to removal and the INR should be <2.5 for patients on warfarin.
- Tamponade is a real risk, even in patients with a normal coagulation profile and therefore patients should be closely observed for 4 hours following wire removal.