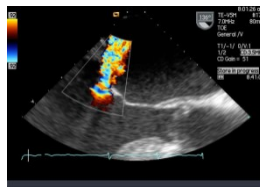




# Wessex Cardiac Intensive Care Unit

*University Hospital Southampton NHS Foundation Trust*

## Anaesthetic registrar and clinical fellow handbook



**2<sup>nd</sup> Edition 2016 - Revised by Dr Kirstin Wilkinson**

Original Version by Dr Paul Diprose & Dr Andrew Richardson



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# ***Introduction***

## **Welcome**

Welcome to the cardiac intensive care unit I hope that you enjoy your time with us. The purpose of this handbook is to provide a guide to how our unit runs and to give you an overview of how to manage various conditions that you will encounter while with us. All that is contained within this handbook represents guidelines only; they should not be looked upon as 'absolute protocols' rather as guides as to how various situations should be approached.

The cardiac intensive care unit in Southampton is one of the busiest dedicated CICUs in the United Kingdom. During the last financial year we had over 1300 admissions, the vast majority directly from cardiac theatres with a small proportion of cardiology and other admissions contributing to this total.

Feel free to use this handbook as a ready reference guide but remember to use all resources around you including the extensive clinical experience of our senior nursing team and the consultant on duty on the unit. Never be afraid to ask for help or advice. If there are topics or pieces of information that you feel are missing from this handbook then do please let us know so that it can be improved upon in the future.

The first edition of this handbook was published in 2012 following extensive help from Dr Andrew Richardson. Andrew was a very valued consultant colleague of ours who sadly died in a motorbike accident in 2013. We remain hugely grateful for all of his hard work that he undertook in trainee and fellow education, and for the effort that he put in to ensure the original version of this handbook became a reality.

With best wishes,

Dr Paul Diprose and Dr Kirstin Wilkinson

*Consultant Cardiac Anaesthetists*

February 2016

# General Housekeeping

Authors: Dr Paul Diprose & Kirstin Wilkinson

Revised: February 2016

## Staff List

A large number of staff work on the intensive care unit; all consultant cardiac anaesthetists with the exception of Linda Nel (who has additional responsibilities for thoracics and ENT), Dr Weidman (additional responsibilities in neuroanaesthesia) and Dr Smith (congenital cardiac anaesthesia only) provide cover for the cardiac intensive care unit. Contact details for all consultants can be found in the flip folders on each side of the unit.

|                                   |   |
|-----------------------------------|---|
| <i>Consultant Medical Staff</i>   |   |
| Dr Mike Herbertson                | Cardiac Intensive Care Lead and Consultant Cardiac Anaesthetist |
| Dr Gareth Charlton                | Consultant Cardiac Anaesthetist                                 |
| Dr Richard Cope                   | Consultant Cardiac Anaesthetist                                 |
| Dr Andy Curry                     | Consultant Cardiac Anaesthetist                                 |
| Professor Charles Deakin          | Consultant Cardiac Anaesthetist                                 |
| Dr Paul Diprose                   | Consultant Cardiac Anaesthetist                                 |
| Dr Ravi Gill                      | Consultant Cardiac Anaesthetist                                 |
| Dr David Hett                     | Consultant Cardiac Anaesthetist                                 |
| Dr Linda Nel                      | Consultant Cardiac Anaesthetist (also covers non-cardiac work)  |
| Dr Tom Pierce                     | Consultant Cardiac Anaesthetist                                 |
| Dr David Smith                    | Consultant Paediatric Cardiac Anaesthetist                      |
| Dr Crispin Weidmann               | Consultant Cardiac Anaesthetist (also covers non-cardiac work)  |
| Dr Kirstin Wilkinson              | Consultant Cardiac Anaesthetist                                 |
| <i>Other Medical Staff</i>        |   |
| Dr Omar Al-Azzawi                 | Specialty doctor  |
| Dr Stina Erricson                 | Specialty doctor  |
| Dr Maria Filippaki                | Specialty doctor  |
| Dr Kausalya Raman                 | Specialty doctor  |
| <i>Senior Nursing Team</i>        |   |
| Rachel Spreadborough              | Matron  |
| <i>Other Key Staff</i>            |   |
| Julie Robinshaw & Natalie Simmons | Ward Clerk  |
| David Young                       | Pharmacist  |
| Mark Tomlin                       | Consultant Pharmacist   |
| Bethin Jenkins                    | Dietician   |
| Louisa Nunez                      | Senior Physiotherapist  |
| Dr Tatshing Yam                   | Consultant Microbiologist                                       |
| Rev Sue Pitkin                    | Trust Chaplain and Pastoral Support                             |

## Maps and Layout

The cardiac intensive care unit is split into two distinct areas (a blue and a pink side). In general, the unit is run by 2 senior nurses-in-charge with one responsible for clinical duties and one for bed management. The map in appendix 1 shows the approximate layout of these areas. You should familiarise yourself both with the local geography but also with the places you are likely to be called to urgently, this includes the cardiac high

dependency unit, the coronary care unit (both on D-level), and cardiothoracic theatres, cardiac pre and post-op wards and cardiac catheter laboratories (all on E level).

### *Familiarisation with Equipment*

There is a large array of equipment used on the cardiac intensive care unit. Some of this will be familiar to you and some may be less so. There is a list of the equipment that you may encounter in appendix 2. Please look at this list and ensure that you have at least a working knowledge on how to use the equipment.

### *Supervision and Tutorial Programme*

All specialist trainees coming onto the unit should have already been allocated an educational supervisor. Any specialist trainee who requires specific support or educational assistance while doing their cardiac block should approach Dr Andrew Curry or the clinical lead for CICU. The responsibility for fellows on the CICU rests with Dr Wilkinson. All fellows will be allocated an educational supervisor during their first week on the unit. Fellows should approach either their educational supervisor or Dr Wilkinson should they require educational assistance or guidance while with us.

Teaching opportunities will be both formal and informal. Informal teaching opportunities will occur on ward rounds, in theatres and in the cardiology labs. There is a regular Friday morning educational meeting held in the anaesthetic department seminar room on E-level, this starts at 07:30am and runs for approximately 40 minutes (allowing then for those on lists to get into theatre). All trainees should try to attend these. In addition, there are training sessions on specific topics on a rotational basis designed for trainees in cardiac anaesthesia and intensive care and transoesophageal echocardiography. Preparation for these will be required by the participants. Dr Wilkinson publishes the rota for these sessions.

### *Resource Room*

There is a trainee resource room on the 'blue side' of the cardiac intensive care unit. It can be accessed via a keypad – the code is C12457. Within this room there are two comfortable chairs designed for rest when on-call overnight, computer terminals and a variety of relevant books, CD-ROMs and DVDs. It is most important that this room is used sensibly and appropriately otherwise we risk losing it. Please observe the following practices:

- Keep the room clean and tidy and locked at all times when not in use
- Do not leave bedding lying around
- Do not remove any resource material from the room
- While you may access personal email accounts and clinically relevant websites absolutely no inappropriate internet access will be tolerated

Any problems with this room or requests for additional resources should be addressed to Drs Curry or Wilkinson.

### *Study Leave*

All CICU fellows are entitled to apply for up to £600 pro rata per year to help pay towards costs incurred attending approved study leave (rotating registrar trainees have their own arrangements). This must be approved prospectively and agreed as appropriate by the individual's educational supervisor or Dr Wilkinson. The relevant form can be obtained from the anaesthetic department secretaries. No study leave will be reimbursed before the fellow has been in post for at least 6 months. Should any fellow leave before one year of employment any paid study leave (for their remaining time) may have to be returned to the Trust. Any queries about this system should be addressed to the CICU lead consultant or your educational supervisor.

### *Conduct on the Cardiac Intensive Care Unit*

While on CICU it is of course expected that you will act in a professional manner at all times under the principles of the GMC guidance for good medical practice. <http://www.gmc->

[uk.org/guidance/good\\_medical\\_practice.asp](http://uk.org/guidance/good_medical_practice.asp) There are some specific areas where your cooperation is expected:

- You are expected to arrive changed and ready to start work at the allotted time for your shift
- Do not eat or drink in any clinical area or around the doctors' or nurses' stations
- You are expected to wear theatre scrubs at all times but if you do walk onto the unit wearing your own clothes you should be 'nil below the elbows' i.e. shirt sleeves rolled up, watch off and no jewellery whenever you enter CICU
- Careful attention to hand hygiene should be taken, this includes hand washing and alcohol rubbing the hands between every patient contact and when moving between clinical areas

### *Out of Hours Contact*

The consultant cardiac intensivist on-call will usually expect to be called in the following circumstances:

- Significant clinical deterioration in any patient
- If any patient needs to go to theatre
- If a request for an admission to CICU is made
- If there are significant disagreements on clinical care between the CICU medical staff and surgical/cardiology colleagues or with the senior nurse on duty on CICU
- In the event of an unexpected cardiac arrest on CICU

The consultant intensivist on-call should be contacted initially via their preferred route (either mobile or home number) as given in the flip folders on both sides of the unit.

### *Note Keeping and Documentation*

We have recently introduced a fully computerised clinical information system (Metavision Clinical Information System) in both theatres and cardiac intensive care. Training will be given on this when you first arrive. Please pay careful attention to making clear and legible clinical records at all times. Always login with your username to ensure all entries are assigned to you.

Pay particular attention to documenting changes in microbiology therapy (with the rationale) in the microbiology ward round section. Also, ensure that if any investigations (such as chest x-rays or CT scans) have been ordered that the outcome of that investigation is clearly documented in the clinical notes.

The other area where clear documentation is especially important is when patients are transferred to another clinical area. If patients are repatriated to cardiac HDU, the ward, GICU or another ICU in the region then they should be accompanied with a clear and comprehensive discharge summary.

### *Time Management on CICU*

There are three 'shift patterns' worked on CICU:

|                 |                    |                          |
|-----------------|--------------------|--------------------------|
| Day shift       | 08:00hr to 21:00hr | (Registrars and fellows) |
| Short Day Shift | 08:00hr to 17:00hr | (Fellows only)           |
| Night Shift     | 20:00hr to 09:00   | (Registrars and fellows) |

At all times of the day there are two junior doctors rostered to cover CICU and during weekdays between 08:00hr and 17:00hr there will be an additional 'short-day' fellow. At the start of each shift you should take a 'handover' from the doctors that are finishing their shift. Accurate and up to date handover sheets are an important way to ensure that this occurs as efficiently and effectively as possible. These are now managed through the clinical information system.

Once handover has taken place you should allocate daily reviews and jobs by mutual agreement. It is often most effective for the 'short day' person to take on the responsibility of ensuring that the patients to be



discharged have been reviewed and a plan written in the notes. This leaves the doctors on duty for the whole day to concentrate on the patients that will remain in CICU. All patients should be examined, all systems reviewed and accurate notes made in the clinical record (see the chapter on admission and review note keeping). You should construct a brief management plan for the patient and communicate this to the bedside nurse.

You should liaise with the consultant in charge of CICU by 9am in the morning to discuss with them any current problems or issues, to decide which patients are to be discharged and to establish when a ward round will occur. Usually consultant ward rounds will occur in the late morning or early afternoon. You will be expected to present the relevant findings from your daily review on this round and to present a plan for how you feel the patient should be managed.

The afternoon is usually devoted to practical procedures including tracheostomy and line insertion, scans and executing the plan agreed on the ward round. Usually around 3pm the microbiology team will arrive for a ward round. You should accompany them on the unit and take them to any patients on antibiotics or who have microbiological issues. It is most important that you document in the clinical notes positive cultures identified or decisions made from this ward round (there is a specific place to put these decisions in the clinical information system).

The night shift doctors will identify those patients that need urgent review and management and deal with those first. Some long stay patients may not require a full night examination and review and indeed this may disturb their sleep patterns. These patients will usually have been identified on the daily ward round; a brief discussion with the bedside nurse is sufficient for these patients. Otherwise, patients should be reviewed, their care discussed with the nurse managing them and a plan for care documented.

# General Patient Management

## Admission and Review Notes

Author: Dr Kirstin Wilkinson

Revised: January 2015

### Admission to CICU

This should clearly state the reason for CICU admission and any relevant past medical history, and the aims of ICU stay.

Age, gender of patient

Presenting complaint:

Operation performed  
(look at surgical op notes for exact details)

History of presenting complaint:

Brief reasons for why operation was needed  
Pre-op echo and angiogram results  
(LV function, valves, coronary arteries affected)  
Pre-op renal function and pulmonary function tests  
On anticoagulants pre-op? If so, stopped and when?

Operation details:

Any surgical complication  
Bypass (CPB) time, Cross clamp (XC) time  
Grade of intubation (relevant for extubation)  
Inotropes pre and post bypass?  
Balloon pump pre and post bypass  
Any blood products given  
TEG and near patient haematology results

Relevant past medical history, treatment and allergies

Full clinical examination including look at drainage from chest if post-op patient

### Clinical Plan

This is normally handed over by the anaesthetist bringing the patient down from theatre

If you are unsure then discuss with senior staff on CICU

Physiological aims – BP, MAP, CVP, Hb, urine output

Warm, wake and extubate timing as appropriate

Bloods, consider if they need a TEG

Consider whether CXR is needed (not routinely performed but will be required if pulmonary artery catheter or IABP in situ)

### Daily Review

Highlight current and any new problems. It should be thorough but concise. Below is a suggested scheme.

- Age of patient.
- Number of days on CTITU

- Main Issue : likely to be post op – what operation and how long ago
- Main problems: simple list e.g. pneumonia/ renal failure/ liver failure/ low CO
- Relevant past medical history inc allergies

### *Clinical examination*

|              |   |  |
|--------------|---|--|
| <b>A:</b>    | Intubated – if so, what size ETT or tracheostomy  |  |
| <b>B:</b>    | Respiratory system<br>Gross or focal chest signs<br>Gas exchange (ABG)<br>Sputum                              | Ventilation – SV/ IPPV<br>Mode of ventilation<br>TV, RR, FiO <sub>2</sub> ,<br>Peak/ Mean airway pressure                          |
| <b>C:</b>    | Pulse – rate/ rhythm (underlying)<br>Pacing mode (inc thresholds)<br>BP/ MAP<br>CVP, Heart Sounds<br>Oedema   | Inotropes – doses<br>IABP – settings<br>CO studies if PAC/ LiDCO<br><br>Drainage from chest drains                                 |
| <b>D:</b>    | Sedation doses if running<br>GCS/ Gross neurology/ focal neurology  |  |
| <b>Abdo:</b> | Routine examination findings<br>Feeding method? absorbing<br>Ulcer prophylaxis<br>LFT abnormalities<br>Bowels | <b>Renal:</b> Urine output<br>Current fluid balance<br>Last 24hr balance<br>Overall balance<br>Frusemide use<br>Urea/ creat/ Na/ K |
| <b>Haem:</b> | Hb, clotting<br>Anticoagulation   | <b>Micro:</b> Temperature<br>Known organisms<br>Antibiotics<br>Age of invasive lines   |

### *Recent radiology*

*Ensure micro sheet (at rear of blood result sheet) is fully up to date*

### *Impression of current and new problems*

#### *Daily plan*

To cover current and new problems

#### *Ward Round*

Write daily ward round plan when consultant ward round occurs and check with the consultant.

Always document the outcome of the micro ward round in the clinical notes.

Please time and date (with legible signatures) all entries into the medical notes. These occur routinely with the introduction of the Computer Integrated System if you are logged in appropriately.

# Infection Issues

Authors: Dr A Richardson & Dr K Wilkinson

Revised: May 2015

## Standard Antibiotic Prophylaxis after Cardiac Surgery

Routine antimicrobial prophylaxis after surgery in most patients consists of a single dose of cefuroxime 1.5g 8 hours after the post-bypass dose given in theatre. Patients with serious allergy (i.e. anaphylaxis) to penicillins or allergy to cephalosporins should receive vancomycin and gentamicin intra-operatively, as should patients who have recently received courses of antibiotics altering the patterns of resistance of endogenous flora. These antibiotics are usually given as single doses only, and any continuation should be discussed with the consultant surgeon or anaesthetist performing the case.

## Infection Prevention

### Screening for MRSA & Reducing the Risk of Infection

Southampton University Hospitals have comprehensive policies and other measures in place to reduce the risk to patients and staff of infection with methicillin-resistant *Staphylococcus aureus* (MRSA) or other microorganisms. The latest versions of all these policies are available on the SUHTranet, but some key points are listed below:

### Standard Precautions

ALL staff are expected to comply with standard precautions for infection prevention and control. These include:

- Wearing nothing below the elbow while in any clinical area (regardless of the reason for being there) in order to facilitate hand hygiene
- Cleaning hands with either alcohol hand rub or soap and water before and after any contact with a patient
- Disposing of any needles, introducers or other sharps immediately after use, at the point of use. (See needlestick injury procedures in the appendix 3 of this manual)

### MRSA Screening and Risk Reduction Measures

Almost all adult admissions to SUHT are screened for MRSA and receive daily whole-body washes and hair shampoo with 4% chlorhexidine gluconate (Hibiscrub<sup>®</sup>) for the first five days of admission. Additional measures apply in some units. In very high-risk units caring for adult patients, including cardiac intensive care and CHDU, the following routine measures apply:

- All patients will be screened for MRSA on admission to the unit. You should verify that this has been done and be aware of the result, when available.
- For the first five days of admission all patients will receive treatment with topical agents to reduce bio burden, regardless of MRSA status. This treatment consists of daily shower/bath/blanket bath with chlorhexidine gluconate, to include the hair on two of the five days, and three-times-daily application of 2% mupirocin ointment (Bactroban<sup>®</sup>). If Bactroban is not available, Naseptin is used for 10 days.
- The treatment must be prescribed on the drug chart. You may use the relevant stickers for the prescription, if available.

If the patient is transferred to another area within the five-day period, the course of treatment must be completed in the receiving area. If the patient is transferred from one very high-risk area to another after completing the course of treatment, the receiving area should NOT initiate a second course.

All patients not already known to be MRSA positive should be screened for carriage on a weekly basis. You should verify that this has been done and be aware of the result, when available.

Patients found to be MRSA positive should be prescribed decolonisation therapy consisting of a five-day course of chlorhexidine gluconate and mupirocin ointment as for bio burden reduction as above. Further sets of screening specimens should be taken to establish if this has been successful. The first set must be taken no less than 48 hours after completion of therapy, and there should be at least 48 hours between sets. A

minimum of three sets of clear specimens is required to verify clearance; the Infection Prevention Team or Consultant Medical Microbiologist may request more.

Biopatch® chlorhexidine-impregnated dressings should be considered for use on all CVC insertion sites in patients who are known to be MRSA positive.

### Ventilator Care Bundle

Ventilator-associated pneumonia (VAP) is defined as nosocomial pneumonia in a patient receiving mechanical ventilatory support for more than 48 hours. It is the most common nosocomial ICU infection, occurring in 9 – 28% of intubated patients, with a peak incidence around day 5 of ventilation. Whilst it remains unclear whether VAP independently increases mortality, the duration of ventilation and lengths of ICU and hospital stays are increased as a consequence.

Most cases of VAP occur after the aspiration of oropharyngeal secretions that pool above the cuff of the tracheal tube. These secretions are contaminated with the aerobic gram-negative bacilli that rapidly colonise the oropharynx of most critically ill patients. A biofilm develops on the tracheal tube, which becomes colonised with bacteria, which are then propelled into the distal airways through the action of the ventilator. The stomach may also act a reservoir for bacteria, especially if gastric acidity is reduced, although the clinical significance of the gastro-pulmonary route of infection remains unclear. Pathogens responsible for VAP include, *Pseudomonas aeruginosa* (24%), *Staphylococcus aureus* (20%) of which >50% is MRSA, MSSA, *Klebsiella* species (14%), fungi (4%), *Haemophilus* (10%), *Streptococcus* (12%) and *Acinetobacter* (8%).

The accurate diagnosis of VAP remains challenging, with no universally accepted diagnostic criteria in place. Prompt treatment with empirical antibiotics is required, with choice of therapy influenced by knowledge of the likely organisms, local microbiological epidemiology, and the results of surveillance cultures from the patient.

Prevention of any nosocomial infection in ICU requires a multi-disciplinary approach; however, a number of specific interventions have been shown to reduce the incidence of VAP. These fall into three groups: reducing bacterial colonisation of upper aero-digestive tract, reducing aspiration of secretions, and minimising the duration of mechanical ventilation.

### Reducing Bacterial Colonisation

- Topical chlorhexidine dental gel 1% 6 hourly 1 hour after tooth-brushing (NOT if chlorhexidine allergic) for all intubated patients until extubated. This is also used in all patients with a tracheostomy until they are decannulated.
- Prevent condensate in breathing circuits from entering patient's airway and replace according to manufacturer's guidance
- Reposition tracheal tube daily to prevent pressure ulcers

### Reducing Aspiration of Secretions

- Regular suctioning of respiratory and oropharyngeal secretions
- Elevate end of bed 30-45 degrees **except** where immediately post op/following IABP removal/open chest

### Minimising the Duration of Mechanical Ventilation

- Daily sedation holidays - accumulation of infusions delays weaning, increases complications: thus sedation is stopped until it is deemed necessary to recommence for ventilation & safety issues
- Humidification of gases in patients ventilated for more than 24 hours to prevent inspissation of secretions

### ***Infective Endocarditis***

Patients undergoing valve surgery for infective endocarditis are on long-term antimicrobial regimes determined by the organism sensitivities, and any changes to antibiotic therapy must always be discussed with the microbiologists.

### ***Catheter-related Blood Stream Infections (CRBSIS)***

CRBSIs are a key performance indicator of any intensive care unit. Scrupulous attention to asepsis must be observed when inserting or using any central venous catheter (CVC), as described in the section entitled 'Line Insertion and Documentation.' All CVCs are silver or silver-sulfadiazine-chlorhexidine coated, although with an increasing frequency of serious allergy to chlorhexidine, our practice is currently under review in this regard. We do not routinely change CVCs as part of our CRBSI-prevention strategy, as there is currently no good evidence that this reduces CRBSIs, and this practice exposes patients to an increased risk of complications from central line insertion.

### ***Initial Management of Sepsis***

Differentiation of sepsis and the Systemic Inflammatory Response Syndrome (SIRS) is not easy in the post-cardiac surgical patient.

**SIRS:** two or more of:

- Temperature > 38°C or < 36°C
- Heart rate > 90 beats per minute
- Respiratory rate > 20 breaths per minute, or PaCO<sub>2</sub> < 32 mmHg (4.27 kPa)
- WBC > 12 x 10<sup>6</sup> cells/mL or < 2 x 10<sup>6</sup> cells/mL

Sepsis:

- SIRS associated with proven or clinically suspected infection

Severe Sepsis:

- Sepsis associated with organ dysfunction, hypoperfusion or hypotension

Assessment of potential sources of infection, the likelihood of an infective aetiology, and the consequences of delayed treatment of sepsis in the particular patient all contribute to the decision to start anti-microbial treatment. Empirical antibiotic therapy should not be started without prior discussion with the consultant covering the CICU. Every effort should be made to obtain relevant specimens for microbiological investigations prior to starting anti-microbial therapy. Although narrow-spectrum agents should be prescribed in preference to broad-spectrum agents this is frequently not possible in the critically ill intensive care patient, where the wide range of potential sources of sepsis and the disastrous consequences of late treatment of sepsis mean that there is often no choice but to use broad spectrum agents to ensure adequate and effective treatment. Such empirical anti-microbial prescriptions should be reviewed no later than 48 hours after commencement, and de-escalated to pathogen-directed narrow spectrum agents promptly where this is appropriate. Prolonged use of broad-spectrum antibiotics is highly undesirable, as it increases selection pressure for multi-resistant micro-organisms and limits options for salvage treatment in those patients who later relapse. Advice should be sought from the CICU microbiologist (Dr. Tat Yam) for all patients who are on broad-spectrum anti-microbials, and especially those with complex infections, positive culture and sensitivity results or with failed empirical treatment.

Dosing of antibiotics must be appropriate for the patient's size, and renal and hepatic function. This is especially important for aminoglycosides (gentamicin) and glycopeptides (vancomycin is our preferred agent on CICU). On CICU, vancomycin is administered by continuous infusion using the unit protocol, whereas in CHDU it is given by intermittent dosing. Advice should be sought from the unit pharmacists where there is any uncertainty about the appropriate dose of these antibiotics for a patient.

Rifampicin and sodium fusidate must NOT be prescribed as monotherapy due to the high risk of resistant organisms emerging following therapy.

### ***Documentation of Decision-making***

It is absolutely vital that the decision-making process for initiating, altering and stopping antibiotics is clearly documented in the medico-legal notes. This not only allows for the defence of retrospective claims made against the Trust, but also (and arguably, more importantly) allows good continuity of care on the unit, despite changes in personnel.

The minimum data set for anti-microbial treatment documentation includes

Date of starting antibiotics

Indications and choice of agents

Cultures taken prior to starting antibiotics

Intended duration of treatment, and plans for de-escalation to narrower spectrum cover when positive cultures obtained

Any positive cultures obtained, with any sensitivities

Date of changes to treatment regimes, and reasons for change, with intended duration of new regime

Date antibiotics actually stopped

**Please document all microbiology ward round decisions**

### ***Blood Culture Sampling***

False positive blood cultures may occur from contamination of samples sent for culture with micro-organisms from a site outside the bloodstream. This is a common problem, and may account for up to 50% of positive blood cultures. False positive cultures may result in the administration of inappropriate antibiotic therapy. It is therefore important that proper sampling technique is used, to reduce the risk of contamination.

- Careful hand hygiene must precede the taking of blood samples for culture
- Personal protective equipment must be worn
- Clean (rather than sterile) gloves may be worn **if** the venepuncture site is not palpated after it has been cleaned
- The venepuncture site should be cleaned with 2% chlorhexidine in 70% isopropyl alcohol, which then must be allowed to dry
- **Do not re-palpate** the vein after this
- For sites such as the femoral artery/vein, where palpation of the femoral arterial pulse is a necessary part of the procedure, sterile gloves should be worn
- The culture bottles must be prepared for inoculation; the caps are flipped off, and each bottle top is disinfected with 2% chlorhexidine in 70% isopropyl alcohol, which is allowed to dry
- Blood is taken using a 'no-touch' technique; the same needle is then used to inoculate the culture bottles; 20 ml of blood per set of cultures is recommended for adult patients; do not re-sheath the needle
- Hand decontamination is then performed, before labelling the bottles and sending to the laboratory; **DO NOT COVER THE BARCODES ON THE BOTTLES WHEN LABELLING!**
- Blood cultures should not normally be taken through pre-existing vascular access devices **unless** these are suspected sources of sepsis; where this is the case, the sampling hub must be carefully decontaminated using 2% chlorhexidine in 70% isopropyl alcohol and then allowed to dry before 20 ml of blood is collected; do **not** discard the first 5 –10 mls of blood aspirated; apply a new sterile needle to the syringe before inoculating the culture bottles in the usual manner
- A set of percutaneous blood cultures **must** be drawn simultaneously when sepsis with a vascular access device is suspected

# Bleeding & Coagulopathy

Authors: Dr Tom Pierce & Dr Kirstin Wilkinson

Revised: June 2015

Every patient after CPB develops a coagulopathy, which for the most part, is sub-clinical. You may find yourself spending a great deal of time managing abnormal coagulation and its impact on post-operative haemorrhage.

When handing over a patient on CICU or CHDU you must ensure that if any blood products are being held for that patient in the transfusion lab that this fact is communicated clearly to the medical and nursing staff on the unit. A plan must be made to either use or release these products before they expire.

## Aetiology

### Patient

- Pre-existing coagulation abnormalities INR and APTR
- Thrombocytopenia
- Anti-platelet therapy
- Aspirin, clopidogrel
- LMWH
- Thrombolytic therapy (aortic dissection mistaken for MI)

### Anaesthetic

- Excess heparin
- Inadequate protamine
- Slow and inadequate response to surgical haemorrhage
- Resultant dilutional coagulopathy

### Bypass

- Hypothermia
- Dilution of coagulation factors (especially paediatric surgery)
- Reversible platelet abnormality
- Thrombocytopenia (sequestration on the CPB circuit)
- Inappropriate activation of platelets
- Partial degranulation of platelets
- Inflammatory response of CPB activating coagulation and consuming factors
- DIC and thrombolysis (v rare)

### Surgical

- Ongoing bleeding from surgical sites may aggravate a coagulopathy and indeed a coagulopathy may disguise surgical haemorrhage. Common sites include
  - Mammary artery bed
  - Aortic cannulation site
  - Venous cannulation site
  - Side branches of the mammary artery or aorto-coronary vein grafts
  - Distal anastomotic sites.

Despite the multiple aetiologies, chest drain loss of more than 600 ml over the first postoperative night is not unusual. The trends in hourly losses are useful. If the patient is not bleeding then no matter what the post-operative coagulation status is, correction is unnecessary.



### *Investigation of unexpected haemorrhage*

- Chest drains - the rate of loss will often give a clue, >4-6 ml/kg/hr a surgical cause is likely.
- Bloods
  - INR, APTR, platelets, fibrinogen
  - Hb
  - Thromboelastogram: plain and heparinase
- Cross-match 4 units, if large blood loss, consider 6 units

### *Supportive management*

- Circulatory stability
- Temperature control and support
- Blood warmers & external forced air re-warming
- Maintenance of adequate haemoglobin
- Correction of coagulation disturbance

### *Return to theatre*

This occurs in 5-8% of patients. In a number no surgical cause is found. Most patients still remain intubated. Preparation, supportive drugs, monitors, assistance and positioning are the same as for first time sternotomy. "Stick-on" defibrillation pads are not needed. Anaesthetic requirements are less than for first time sternotomy, 100-500µg fentanyl, isoflurane or propofol and vecuronium are appropriate. Beware a sudden increase in cardiac output as the chest is opened in the setting of cardiac tamponade; risk of awareness. Remember to give 1g vancomycin over 1 hour to cover chest re-opening.

# Epicardial Pacing

Authors: Dr Tom Pierce & Dr Kirstin Wilkinson

Revised: May 2015

This is required for the intra- and post-operative management of patients undergoing cardiac surgery. Although the majority of patients do not require pacing to facilitate separation from cardio-pulmonary bypass (CPB) it is difficult to select those who subsequently require pacing during the early post-operative period.

## *Epicardial Pacing Wires*

In most cases, two wires are placed on the right atrium (RA) and two on the right ventricle (RV). The ability to pace the atria is advantageous in many patients, especially those with reduced ventricular compliance (as occurs with ischaemia). This group have a substantially reduced cardiac output in the absence of atrial contraction to assist in ventricular preloading. Atrial or A-V sequential pacing thus offers the advantage of increasing cardiac output by up to 25%.

One end of the epicardial wire is lightly sutured to the myocardium with the other penetrating the chest wall to the surface. Wires attached to the RA emerge through the skin on the right of the sternum, those from the RV to the left of the sternum.

## *External Pacing Box*

At Southampton, we are currently in a transition period for pacing boxes. The current supply of APC Micro-Pace 4570 dual chamber external pacemaker is being replaced with the Cardio Logic Pace T20 pacing boxes. We also have a number of Osypka and St. Jude boxes in circulation. The latter 2 boxes are identical in function, just differing in colour. It can be confusing and potentially dangerous to have more than 1 type of pacing box in circulation, so you should familiarise yourself with all the boxes until the stock is of one type. Please ask a senior cardiac anaesthetist to explain how the boxes function before changing any parameters.

The two atrial wires are connected to the atrial ports of the pacing box. By convention in Southampton, the **blue** wires are connected to the **ventricles**, whilst the **white** wires are connected to the **atria**. **YOU MUST CHECK YOU HAVE INSERTED THE WIRES CORRECTLY INTO THE PACING BOX.** The atrial and ventricular wires should be labelled clearly. Which atrial wire is connected to the positive or negative port is of no consequence; this is the same for the ventricular leads.

On the front of the 2 new boxes, is the 'OFF-ON' button. Programming can only be undertaken in the ON position but after this THE PACEMAKER SHOULD ALWAYS BE LOCKED by pressing yellow "key" button.

When the external pacing box is turned on it will be by default in DDD mode with a rate of 60, with atrial and ventricular sensing and pacing output voltages set to appropriate levels for a standard patient. These voltages are not normally adjusted in theatre.

Most patients leave theatre DDD paced with a rate of 80-90 in order to optimise cardiac output. In some cases DDD may not be appropriate e.g. in chronic atrial fibrillation VVI or DVI may be chosen instead.



Micro-Pace™  
4570  
Dual Chamber  
External  
Pacemaker



Osypka pacing  
box



St. Jude Medical  
Pacing Box



Cardio Logic Pace  
T20 Box

### General Care of a Patient with Epicardial Wires

#### Micro shock

Epicardial pacing wires are low resistance connections to the heart and thus there is a potential for micro-shock induced arrhythmia, particularly VF.

Patients must be nursed in a cardiac-protected environment - adequately isolated electrical equipment and measure to prevent build-up of static electricity. Wires should only be handled with non-conductive gloves and a large metal object e.g. the bed should be touched first to discharge static potential prior to touching the wires. The wires should be protected in a non-conductive container when not in use.

#### Tips on Monitoring

Modern digital ECG monitors apply a high frequency filter to the incoming signal to minimise unwanted electrical interference – this often filters out the brief pacemaker spike making it difficult to tell whether a pacing stimulus is being delivered. Therefore, select the ‘pacemaker’ mode which will record each spike, often highlighted with a marker.

Electrical pacemaker output does not necessarily equate to mechanical capture of the myocardium and therefore it is helpful to have a monitor demonstrating the timing of cardiac contraction e.g. an arterial pressure tracing or pulse oximeter waveform. If attempting to find pacing settings that produce optimal cardiac output it is beneficial to monitor cardiac output via echocardiography, a pulmonary artery catheter, mixed venous oxygen or pulse contour analysis.

Due to the risks of pacemaker system failure or pacemaker generated arrhythmia patients should have as a minimum, continuous ECG monitoring and access to a cardiac defibrillator with the capacity for transcutaneous pacing.

#### Routine Checks

These should occur every day and ideally with each change of nursing/medical shift.

- Battery indicator
- Appropriate pacing mode and rate
- Underlying rhythm
- Sensitivity
- Capture threshold

### **Underlying rhythm**

Need for ongoing pacing should be regularly reassessed. The output may be temporarily stopped in order to assess the underlying rhythm, as follows:

Press the PAUSE key until pacing is inhibited. Assess the rhythm. To restore pacing, simply release the PAUSE key. This will result in return to the normal status display.

The risk of this is that there may be no underlying rhythm and it is occasionally impossible to re-establish capture once it has been lost.

### **Sensitivity**

This is the minimum current that the pacemaker is able to sense – a lower number thus corresponds to a greater sensitivity.

The pacemaker rate should be set below the endogenous rate (if present) & placed in VVI, AAI or DDD modes. The sensitivity number is increased (making the pacemaker *less* sensitive) until the sense indicator stops flashing. Pacing should then occur asynchronously in the chamber being tested – do not allow this to persist for too long because of the risk of precipitating atrial or ventricular fibrillation if the pacing spike is delivered late in the repolarisation phase (an artificial R-on-T). The sensitivity number is then turned down (making the pacemaker *more* sensitive) until the sense indicator flashes with each endogenous depolarisation (in time with the P or R wave on the surface ECG) – the number at which this first occurs is the pacing threshold. It is recommended to leave the pacing generator set at half the pacing threshold, to allow for detection of abnormally small signals & to reduce possibility of peri-lead fibrosis that will reduce the current transmitted to the pacemaker.

If there is no endogenous rhythm this cannot be done. In this situation the sensitivity is typically set to 2 mV.

If the sensitivity is too low (the pacemaker is too sensitive), there will be inappropriate sensing of R or T waves which may inappropriately inhibit pacing.

### **Capture threshold**

This is the minimum pacemaker output required to stimulate an action potential in the myocardium. It should not be checked if there is no underlying rhythm – in this situation careful attention should be paid to the development of occasional missed beats which may indicate a rise in the capture threshold.

If safe to check:

Set the mode to VVI and set the rate to 10 bpm above the patient's rate. Set the ventricular output to 2.5V. Slowly decrease output until capture is lost. Slowly increase output until capture is regained. THIS IS THE CAPTURE (OR STIMULATION) THRESHOLD. Increase the output to provide an ample margin of safety for capture: 5.0 VOLTS OR 3x the threshold (whichever is the greater). It is recommended that the output be kept at 5.0 V or above unless special circumstances with the patient necessitate otherwise.

An inflammatory reaction can develop around the wire/myocardial interface. This is accelerated when higher energy is applied – one reason to limit pacemaker energy output. Increases in stimulation threshold commonly occur after 4 days in both atrial and ventricular pacing wires with failure to pace observed in greater than 60% right & 80% left atrial wires after 5 days.

### **Rate**

Every patient will have an optimal heart rate for cardiac output after which as heart rate increases, stroke volume falls.

However, in practice optimal heart rate is rarely titrated to cardiac output – it is usually left at 80-90 beats per minute (after the above adjustments are made).

Some advocate a period of 'back-up' pacing (with the pacemaker rate set at 40 beats per minute) which allows the patient to remain in an endogenous rhythm until the point of significant haemodynamic compromise. The advantage of this is that the sensing threshold of the pacemaker can be continuously monitored. If full pacing

is again required, it can be commenced with the confidence that the pacing threshold will not have become too excessive.

### Pacing Settings

Only the first 3 of the 5 positions of the North American society of Pacing and Electrophysiology (now the Heart Rhythm Society)/British Pacing and Electrophysiology Group Generic Code (the NBG code) are relevant to temporary epicardial pacemakers:

| I                | II               | III                 |
|------------------|------------------|---------------------|
| Chamber paced    | Chamber sensed   | Response to sensing |
| O = none         | O = none         | O = none            |
| A = atrium       | A = atrium       | T = triggered       |
| V = ventricle    | V = ventricle    | I = inhibited       |
| D = dual (A + V) | D = dual (A + V) | D = dual (T + I)    |

The following are pacing modes applicable to temporary epicardial pacing:

#### DDD

The most commonly used mode in patients with both atrial and ventricular pacing wires. The pacemaker waits for an endogenous atrial depolarisation. If none is sensed, an atrial spike is delivered. The pacemaker then waits for an endogenous ventricular depolarisation, in response to either the atrial pacing spike or endogenous atrial depolarization, should this have occurred. If there is no endogenous ventricular depolarization, a ventricular pacing spike is delivered.

The maximal rate in DDD is not the set lower rate limit; instead the ventricular pacing spikes can be delivered at a higher rate so as to 'track' atrial activity. DDI is thus better than DDD in the context of rapid atrial arrhythmias, as in DDD the ventricle will potentially be paced too rapidly.

Indications: All indications for pacing, with the exception of atrial tachyarrhythmia.

#### AAI

Used in patients with an intact and reliable atrio-ventricular conducting system. The pulse generator has a sensing 'timing cycle' determined by the rate set on the pacemaker. If no endogenous depolarization is sensed by the end of the cycle, a pacing spike is delivered to the atrium. If an endogenous depolarization is sensed, no spike is delivered and the timing cycle begins again.

Ventricular ectopics can be problematic as no ventricular depolarization is sensed - an atrial stimulus can potentially be conducted to the ventricle whilst it is in the repolarisation phase of a ventricular ectopic precipitating R-on-T VF. Fortunately this is usually prevented by the AV node that has entered its refractory period following the ventricular ectopic and so blocks transmission of the atrial impulse.

Indications:

Relative bradycardia with an endogenous atrial rhythm sufficiently quick to compete with the pacemaker rate.

Limitations:

Contra-indicated in atrial tachycardia, AF/flutter (due to inability to capture the atrium) and AV node block.

#### VVI

Used when atrial pacing is futile e.g. atrial fibrillation. This is the same as AAI except the sensing and pacing is in the ventricle.

Indications:

Relative bradycardia with AF & Atrial flutter, sick sinus syndrome

Limitations:

No atrial contribution to ventricular preload.

If these synchronous modes are used in the presence of diathermy there is the potential for interference to cause inhibition of pacemaker output in the absence of an endogenous rhythm. Asynchronous modes AOO, VOO & DOO are indicated in theatre when diathermy is being used. In addition DOO is indicated for the emergency management of pacemaker-mediated tachycardia.

#### AOO (Atrial asynchronous)

Pacing spikes are delivered to the atrium at a set rate regardless of electrical activity in either chamber of the heart. Ventricular contraction in this mode relies on intact conduction through the AV node. There is a risk that a pacing spike might be delivered in the repolarisation phase of an endogenous atrial beat, which may precipitate AF.

Indications:

Bradycardia with intact AV node conduction, in situations where synchronous modes are contra-indicated i.e. use of diathermy that can interfere with pacing.

Limitations:

Contra-indicated in atrial tachycardia, AF/flutter (due to inability to capture the atrium) and AV node block.

#### VOO (Ventricular asynchronous)

Pacing spikes are delivered to the ventricle regardless of electrical activity in either chamber of the heart. During a paced beat, there is no co-ordinated atrial contraction which can significantly reduce CO. There is a risk that a ventricular pacing spike may be delivered when the ventricle is in the repolarisation phase of an endogenous beat. This is the classic 'R-on-T' phenomenon, known to precipitate VF.

Indications:

Bradycardia without reliable AV node conduction, in situations where synchronous modes are contra-indicated e.g. use of diathermy.

In an emergency, to preserve CO in the case of malfunction of pacing in one of the more sophisticated pacemaker modes.

Limitations:

Competition with intrinsic rhythm; possibility of R-on-T VF, no atrial contribution to ventricular preload.

#### DOO (AV sequential asynchronous)

First the atrium and then the ventricle receive a pacing spike with the spikes separated by a programmed AV delay. The same risk of R-on-T VF as in the AOO & VOO modes is present. Although mechanical efficiency is better than VOO, it is not as efficient as that of an endogenous impulse through an intact conducting system and therefore AOO is preferred if the conducting system is intact.

Indications:

As for VOO but in particular in patients who derive substantial haemodynamic benefit from the contribution of atrial contraction to ventricular preload, in situations where synchronous modes are contra-indicated i.e. diathermy.

## Removal of Epicardial Wires

The epicardial wires are usually removed from the patient after instruction by the surgeon. They are usually removed by the CICI nurses. They should be removed with constant gentle traction. Occasionally the wires

may be caught by a tight suture and in this situation they are pulled as far as felt safe then cut as close to the skin as possible – the cut ends will retract. There is no evidence that wires left like this have any adverse effect.

Observe the patient for a few hours for:

- Tamponade – small but definite risk at this point
- Ventricular arrhythmia
- Damage to coronary anastomoses

Postoperative cardiac patients on IV heparin infusion will have their epicardial wires removed on Day 4 or after. Heparin should be stopped and the wires removed after 2 hours. The APTR does not need to be checked. The Redivac drain can then be removed an hour after this if no bleeding has been seen from the pericardial space. The heparin can then be restarted immediately. Sending bloods for an APTR will delay pacing wire removal which would be high risk in patients with mechanical heart valves.

### *Transition to a Permanent Pacemaker*

Risk factors for requiring this include age, pre-operative bundle branch block, prolonged cardiopulmonary bypass & suboptimal intra-operative myocardial protection.

Common indications for permanent pacing after cardiac surgery include CHB, sinus node dysfunction, slow ventricular response to AF and second degree Mobitz type 2 heart block with an inadequate ventricular rate.

The optimal timing of the decision for this depends on the clinical course but at 4-5 days it is reasonable to consider this option as by this time the epicardial wires will have begun to fail. At the time of pacemaker box implantation the patient should not be anticoagulated.

### *Intra-Aortic Balloon Pump (IABP) and pacing*

If this is timed according to a cardiac monitor the high frequency filter should be on, or the spikes may be misinterpreted by the IABP as QRS complexes. Alternatively, the IABP should be timed according to the arterial waveform.

### *Complications of Permanent Pacemaker Insertion*

- Infection
- Myocardial damage
- Perforation
- Tamponade
- Disruption of coronary anastomoses

# Sedation and difficult to sedate patients

Authors: Victoria Tricker (Pharmacist) & Dr Kirstin Wilkinson

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Sedatives and analgesia are used in CICU patients to reduce patient distress and enable appropriate treatment (e.g. ventilation, therapeutic procedures) to be carried out. The choice of agent used will depend on the indication for use and likely duration, presence of renal/ hepatic failure, underlying pathology and pain/distress. Care should be taken to avoid over sedation and accumulation of these agents.

A comprehensive guide to all the common parenteral drugs used on cardiac ICU is available at each end of the unit. This includes information on drug doses and dilutions, compatibility, drug monitoring and options in fluid restricted patients.

## *Sedative options on CICU*

Opioids are used for their analgesic as well as sedative properties. They are potent respiratory depressants, and also reduce gastric motility (consider need for pro-kinetics).

Propofol, a short acting iv anaesthetic, has no analgesic properties. It is a negative inotrope.

Benzodiazepines are useful as they have anxiolytic and amnesia properties.

CAUTION - note the difference in midazolam strengths held in the trust- theatres keep 1mg/ml concentration whereas CICU keep 5mg/ml concentration.

The majority of post-op patients admitted to CICU who are likely to be extubated quickly are maintained on propofol infusions. An opioid e.g. morphine is given in small incremental doses pre- and post-extubation (along with paracetamol) for patient comfort.

Patients who may require sedation for up to three days are usually maintained on a combination of propofol and fentanyl. Patients who are likely to require longer-term sedation owing to post-op complications (e.g. ARDS, chest sepsis) may be converted to a combination of morphine and midazolam - remember these may accumulate with prolonged use, particularly in renal/ hepatic dysfunction. Fentanyl is the preferred agent in renal failure. Where fentanyl infusions have been running beyond a week, signs of accumulation may occur. Sedation breaks should be performed if this is appropriate to do so.

Remifentanyl is an ultra-short acting opioid analgesic. As it is an expensive drug, it is restricted to consultant use only in line with the guideline (available for reference on the unit). Uses include patients with severe renal or hepatic dysfunction, where frequent neurological reassessment is required, in the morbidly obese and when alternative sedative agents have been tried and failed. It should not be used for more than 48 hours unless there is a compelling reason.

## Clonidine

Clonidine is partial agonist acting on predominantly  $\alpha_2$  receptors in the CNS and peripheries. It has sedative and analgesic properties (including opioid sparing effects), but can also cause some other well-recognised effects such as hypotension and bradycardia. Tolerance may develop after ~1 week. Doses should be titrated down when stopping to prevent rebound hypertension.

## *Difficult to sedate patients*

Some patients can be a challenge to keep calm and sedate - you should be mindful of possibility of ICU delirium. Delirium is an independent predictor of mortality at six months; it is associated with an increased length of hospital stay, and can predispose patients to prolonged neurophysiological disturbances after they leave intensive care. It is thought to have an incidence of 15-80% in the critically ill.<sup>2</sup>



ICU delirium is associated with a fluctuation or change in mental status, and inattention. Inattention is characterised by either disorganised thinking or an altered level of consciousness; patients often have a deranged sleep-awake cycles. The three delirium subtypes are:

Hyperactive (better recognised) – patients are agitated, paranoid and disorientated

Hypoactive – patients are withdrawn, quiet, and paranoid. Often misdiagnosed as depression (remember disorientation is common in delirium but NOT a feature of depression)

Mixed

Delirium is thought to occur when there is either dopaminergic (DA) excess and acetylcholine (ACH) suppression (or both).

Benzodiazepines are principally GABA agonists, but also inhibit both dopaminergic and acetylcholinergic pathways. Following the withdrawal of a continuous infusion of midazolam, a relative DA and ACH excess can occur, and may worsen delirium. Fever, hypoglycaemia, liver, renal and cardiac failure can also worsen delirium owing to the effects on the DA/ ACH pathways.

Predisposing and precipitating factors are multifactorial,

Predisposing factors: alcohol abuse, polypharmacy, old age, chronic renal failure, malnutrition, inactivity, hearing and visual impairment

Precipitating factors: sedatives, opioids, alcohol / drug withdrawal, polypharmacy, shock, infection, pain, metabolic disturbances.

Non-pharmacological management of delirium

- Support and orientation
- Unambiguous environment
- Maintain competence- sensory care, participation/ activity where possible, sleep
- Remove potential organic drivers e.g. pain, hypoxia, haemodynamic instability

Pharmacological management of delirium

Treatments commonly tackle the dopaminergic overdrive e.g. haloperidol, but little is done to counter the ACH suppression. Drug choices for managing delirium,

Avoid drugs with dopaminergic (e.g. dopamine, levodopa) or anticholinergic side effects (e.g. codeine, digoxin, frusemide, prednisolone, ranitidine). If possible, avoid antidepressants (e.g. paroxetine, amitriptyline), hypnotic agents and anticonvulsants (e.g. phenytoin- but often not practical).

Haloperidol: start with 2.5-5mg po/ng tds regularly (patients identified earlier often need lower doses).

Agitated patients may require higher doses e.g. 10mg iv. In severe agitation wait 20-30 mins for effect, and if none, double the dose and give again. There is no maximum dose established for this indication, but remember that haloperidol can affect the QT interval.

Benzodiazepines may be required when haloperidol is failing and the patient is a danger to themselves or staff.

Olanzapine has been used for delirium when haloperidol is not tolerated (olanzapine 5mg od is equivalent to haloperidol 2.5-5mg tds). Antipsychotics are associated with an increased risk of stroke in dementia patients.

### *Withdrawal delirium*

Benzodiazepines: withdrawal usually occurs after prolonged periods of sedation, but can occur after just several days. Ensure regular sedation breaks if practical. Lorazepam ng can be used to aid withdrawal.

Opiates: titrate analgesia and reassess daily. Use paracetamol to reduce opiate need and consider type of pain (e.g. neuropathic) and the need for adjunct agents.

Antidepressants: withdrawal symptoms include anxiety, agitation, tremors and insomnia for SSRIs, and sleep disturbance with tricyclics. Restart regular medication promptly and remember there is a balance between withdrawal symptoms and the risk of ICU delirium.

Nicotine: evidence for Nicotine Replacement Therapy (NRT) in the critically ill is limited. Clonidine has been used with some success; if NRT is used, remember nicotine is thrombogenic.

Alcohol: use short acting sedatives e.g. chlordiazepoxide (not if on midazolam infusion). Remember vitamin replacement.

### *Difficulties with the patient sleeping?*

Consider haloperidol 2-5mg IV nocte.

Benzodiazepines can worsen ICU delirium (delirium can present as disturbed sleep).

Chloral hydrate is metabolised to alcohol and has been used with success in some difficult to manage patients. Consider 500mg tds (or 1-1.5g nocte) po/ng. This has the potential to worsen delirium so monitor patient carefully - if used for longer than a week gradually reduce the dose over several days to stop.

Melatonin has been used to reset a patient's sleep-awake cycle if the patient is sleeping during the day and not at night.

### *References*

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# Nutrition on Cardiac ICU

Author: Bethan Jenkins (Critical Care Dietitian)

Date: March 2015

Nutrition is a basic human need and research has shown that patients who are malnourished have higher morbidity and mortality levels, higher incidence of infection and complications, increased number of ventilator days and greater length of hospital stay overall (**Stratton et al 2005**). Thus nutrition is an important part of a patient's treatment.

Here on CICU, we have procedures in place to facilitate optimum nutrition for our patients.

## Identification of At Risk Patients

All patients undergo nutritional assessment within the first 24 hours on CICU using **MUST** (Malnutrition Universal Assessment Tool). This is UHS Policy. It is usually completed by the nursing staff, but is the responsibility of all the Team. The MUST score will identify malnourished patients or patients at high risk of becoming malnourished. However all patients who have a prolonged ICU stay will be at high risk of malnutrition and consideration should be made as to how to best meet nutritional needs and minimise losses.

## Route of feeding

Our patients may be unable to eat & drink, because they are intubated and ventilated, neurologically unsafe or swallow impaired. Enteral feeding should be used where the gut is viable. Enteral nutrition maintains the integrity of the gut, ensuring an adequate blood supply to the tissue and minimising the risk of sepsis. If the gut cannot be used, e.g. due to intestinal ischaemia, ileus or complete mechanical obstruction, then parenteral nutrition should be considered via discussion with the consultant, dietitian and pharmacist.

## Enteral Feeding

This should be initiated ideally within 24-48hrs post operatively, but this may be delayed pending extubation. Early enteral feeding is thought to reduce complications and improve outcomes. The aim of feeding is to minimise nutritional losses, not to replenish nutritional reserves. While early enteral feeding is desirable, it is important not to overfeed critically ill patients, and the dietitian will take into account other sources of energy, such as propofol and dextrose infusions, when prescribing the feeding regimen.

Initially, a wide bore NG tube is passed (14fr Ryles<sup>®</sup> or 10-12fr 'Corflo<sup>®</sup>' tube). These are easier to aspirate, which is necessary to assess feed tolerance in sedated patients. If a Ryles<sup>®</sup> tube is used (14fr), it has to be replaced after 5 days, with an 8-12fr Corflo<sup>®</sup> tube.

Before starting to feed, correct placement of the NG tube should be confirmed. This is usually done by checking that the pH of the aspirate is <5 (**UHS Adult Enteral Feeding Guidelines**), but as our patients will be on gastric prophylaxis, the pH may be higher than this (Metheny et al 1998), in which case, a chest X-ray will be necessary to confirm position of the NG tube. Please make sure the radiographer knows that it is to confirm NG placement, and they will use a different penetration of X-ray to facilitate visualisation of the tube. Confirmation of correct positioning of the NG tube should also be done when restarting feeding, after re-insertion or repositioning of a NG tube, if there is suspicion of tube displacement, or daily where there is continuous 24hour feeding. Following an x-ray the results need to be clearly documented in the medical notes before the tube should be used.

We have an **Enteral Feeding Flowchart** on CICU, which should ensure that all patients are fed using the same best practice. Copies of the protocol are available on the unit.

All patients are usually started on Nutrison Standard at 25ml/hour.

The NG tube is aspirated every 4 hours, and providing gastric aspirate is less than 250ml, the rate is increased 25ml/hr 4 hourly until target rate is reached. If gastric aspirates are greater than 250ml, the abdomen is distended and/or there is vomiting, the rate of feeding is reduced by 25ml/hr and the procedure repeated until feed tolerance is established or feed rate reduced to a minimum rate of 10 ml/hr.

Large gastric residual volumes are sometimes seen in critically ill, sedated patients, because of gastroparesis. **Prokinetic** agents are started if this persists. Metoclopramide is given initially, with Erythromycin added if necessary, to avoid promotion of bacterial resistance.

If feed intolerance persists despite prokinetics a semi-elemental / MCT feed can be trialled, please discuss with dietitian. If NG feeding is not successful after the introduction of prokinetic agents and review of feed type, jejunal feeding should be considered. If the previous measures have not assisted, then PN should be considered. If a patient is experiencing feed tolerance problems please discuss with the dietitian for advice on alternative feed types, nutritional adequacy of current intake and alternative feeding routes.

On CICU, we feed continuously over 24 hours to promote better glycaemic control (NICE-SUGAR study 2009). However many patients will experience interruptions to their enteral feeding for procedures such as extubation, tracheostomy insertion, theatre etc. Repeated interruptions to feeding can lead to large cumulative nutritional deficits so it is important to consider the timing and rationale for stopping feeds and not to stop feeds unnecessarily. If feeding has been stopped for a procedure the dietitian may review the feed rate to try and provide some "catch up" feeding if possible.

### **Parenteral Nutrition (TPN)**

If the gut cannot be used, e.g. due to intestinal ischaemia, ileus or complete mechanical obstruction, then parenteral nutrition should be considered. PN should also be considered for patients with poor enteral feed tolerance that are unable to meet nutritional requirements enterally.

Initiation of parenteral nutrition should be discussed with the consultant, dietitian and pharmacist. Parenteral nutrition is ordered by the ICU pharmacist in discussion with the dietitian.

Parenteral nutrition is comprised of glucose, lipids and amino acids with added electrolytes, vitamins, minerals and trace elements. Feed regimens will be adjusted depending on the patient's clinical condition, nutritional requirements, other nutritional intake (EN / propofol etc), blood glucose levels and biochemistry.

### **The Dietitian**

The dietitian is normally available Mon-Fri, 8.00am to 4.00pm.

The starter regimen and enteral feeding flowcharts are designed to allow the safe initiation of feeding when the dietitian is not available and to allow prompt initiation of feeding.

Enterally fed patients should be referred to the dietitian by Day 2 of enteral feeding. This may be later if extubation is pending or earlier if the patient is on significant amounts of propofol, CVVHF or at risk of re-feeding Syndrome.

The dietitian will:

Assess the patient's nutritional requirements

Prescribe a feeding regimen appropriate to their clinical condition

Take into account clinical conditions, e.g. DM, Renal failure, obesity

Monitor the feed

Oversee cessation of feeding and the move to oral diet.

Please telephone or bleep the dietitian if you have a query regarding the feeding regimen or you think that a change of feed is needed due to a decision relating to the clinical management of the patient, e.g. fluid or electrolyte restriction. She is also happy to attend the ward round if that is helpful, but this is not routine.

### **Diarrhoea**

This is a common problem for enterally fed patients. If your patient has frequent, type 6-7 stools, consider:

If laxatives have been prescribed appropriately

If they are on antibiotics

Send off a stool sample to eliminate C.Difficile

Is there any underlying gut pathology

Is it appropriate to use anti-diarrhoeal agents?

Discuss with the dietitian whether the feed needs to be altered

### **Albumin**

While albumin is a reliable marker of morbidity & mortality, it is not a good nutritional marker.

Under the body's cytokine response, vascular permeability increases, accelerating the leakage of albumin from the circulation, causing serum levels to drop. During sepsis, hepatic protein synthesis is prioritised towards +ve acute phase proteins, such as CRP, and away from -ve acute phase proteins, e.g. albumin. Thus, if the CRP is  $\uparrow$ , albumin will be  $\downarrow$ .

Nutritional support will not influence albumin levels. The underlying cause must be treated.

### **Re-feeding Syndrome**

Please refer to the UHS Re-feeding Guidelines for further details.

Patients who may be at risk of re-feeding syndrome are those who have:

- A BMI of  $< 16\text{kg}$ , or  $< 18\text{kg}/\text{m}^2$  plus another risk factor
- Unintentional weight loss  $> 15\%$ , or  $> 10\%$  plus another risk factor
- Minimal nutritional intake of at least 5 days
- Low levels of  $\text{K}^+$ ,  $\text{Mg}^{2+}$ ,  $\text{PO}_4^-$  prior to feeding
- History of alcohol or drug abuse

If a patient is at risk, the feed must be started more slowly, e.g.  $10\text{kcal}/\text{kg}$  for initial 24 hours, with gradual increase in feed rate. Electrolytes should be checked and corrected prior to feeding and before the rate is increased further.

Restore and monitor circulatory volume, fluid balance and electrolytes.

High risk patients must have their ECG monitored for the first 48hrs of feeding.

Patients must also be supplemented with the following:-

Thiamine  $100\text{mg}$  bd 10 days

Vitamin B co strong 1 bd 10 days

Pabrinex I + II od can be used in place of Thiamine / Vit B for the first 3 days

Forceval soluble od

Refer the patient to the dietitian as soon as possible, ideally before feeding is commenced.

### **Progressing to Oral diet**

Once enteral feeding is stopped, or patients start to eat and drink following their procedure, food record charts are kept in order to monitor their intake.

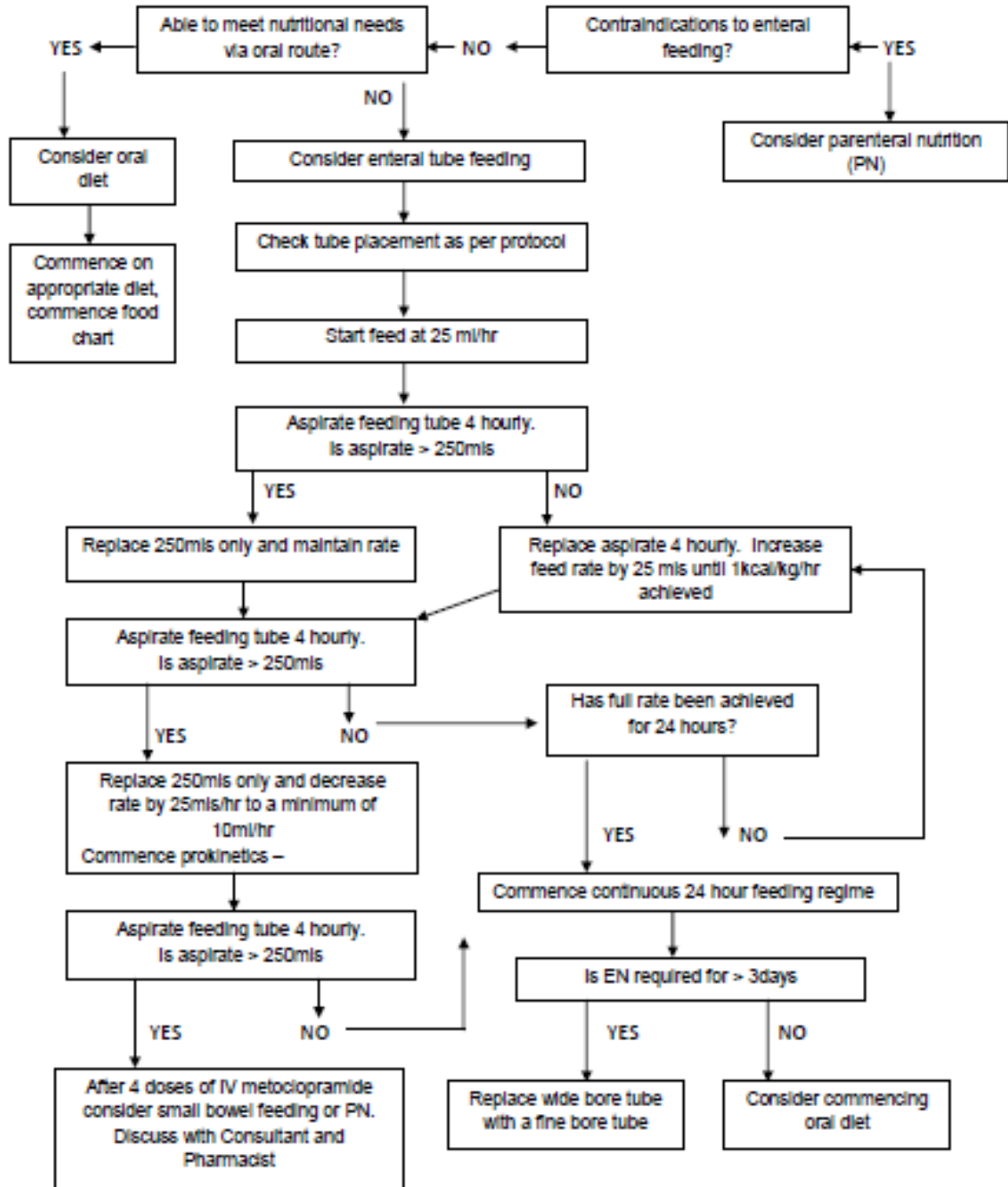
If their intake is not improving after 3 days, refer to the dietitian for oral nutritional support.

All patients should be ordered high energy/high protein meals to maximise intake.

Patients should be provided with Fortisip Compact bd for the first 72 hours post operatively as oral intake is unlikely to meet nutritional requirements during this time (as long as there is no contraindication for this indicated by the medical team).

**Southampton General and Cardiac Intensive Care Units Enteral Nutrition (EN)  
Feeding Flowchart**

**Nutritional assessment within 24 hours of admission to unit - MUST score, current weight obtained and approximate energy requirements (1kcal per kg/hour, actual body weight) calculated.  
Dietetic referral to be made following SUHT referral guide**



# Gastrointestinal Disorders on CICU

Authors: Dr A Richardson & Dr K Wilkinson

Revised: January 2015

GI complications following cardiac surgery are not common (0.5 – 1% of patients), but may be associated with prolonged ICU stay and high mortality rates. The complications range from simple, temporary paralytic ileus to more serious conditions such as gastrointestinal haemorrhage and mesenteric ischaemia. The signs of intra-abdominal pathology may be masked by sedation and mechanical ventilation so a high index of suspicion combined with aggressive investigation is required.

## Constipation

Reduced enteral intake and the effects of opioid analgesics frequently combine to produce constipation. However, if left untreated, faecal impaction and bowel obstruction may occur. Signs of faecal impaction include abdominal discomfort and distension, loaded rectum on digital rectal examination (DRE), or overflow diarrhoea.

If no contra-indications, osmotic laxatives (e.g. lactulose) and pro-motility agents (e.g. senna) should be used in patients who have not had a bowel motion for more than 72 hours. If unsuccessful or there is the suspicion of faecal impaction, an abdominal radiograph should be obtained to exclude ileus or bowel obstruction. Further interventions are documented in the CICU local guidelines.

## Diarrhoea

Diarrhoea is common in the longer stay CICU patients, and has a wide differential diagnosis. Even diarrhoea with no serious underlying pathology will delay patient discharge and make bed management more difficult. Causes of diarrhoea include:

- Continued excessive administration of laxatives
- Overflow from faecal impaction
- Medication
- Antimicrobial-induced bacterial overgrowth (e.g. *Clostridium difficile*)

Risk factors include prolonged ICU stay and use of broad-spectrum antibiotics, particularly clindamycin, cephalosporins and fluoroquinolones. Mortality rises significantly in the over 60 years patient group. Presentation varies from diarrhoea, to toxic megacolon, systemic sepsis and colonic perforation. Three samples for *C. Difficile* toxin must be sent and microbiology should be informed. Consider changing the existing antibiotic regime and commencing enteral metronidazole or vancomycin.

- Ischaemic colitis: may present with bloody diarrhoea
- Viral or bacterial gastroenteritis

DRE must be performed and treatment commenced as below:

- Calorie-dense enteral nutrition
- Adding non-absorbable fibre or changing to a more dilute feed may help
- Anti-diarrheal drugs (e.g. loperamide or codeine) should be used only where pathological causes have been excluded.

## Bowel obstruction and ileus

Distinguishing ileus, mechanical obstruction and pseudo-obstruction in sedated, ventilated patients may be extremely difficult. All are characterised by abdominal distension and intolerance of enteral feeding, and the presence/character of bowel sounds is non-diagnostic. Initial treatment of all three conditions is conservative, with nasogastric drainage, cessation of enteral feeding, the avoidance of drugs that impair gastro-intestinal motility, and the replacement of fluid sequestered in the bowel lumen and wall or lost via the NG tube.

Ileus:

This reversible reduction in motility may involve the stomach, small bowel or large bowel, causing abdominal distension: pain is a less common feature. Abdominal radiographs show non-specific distension of large and

small bowel, with colonic gas present to the recto-sigmoid junction. Management is conservative, and resolution usually occurs within a few days. Ensure any electrolyte disturbances are corrected.

#### Mechanical Obstruction:

Volvulus, strangulated hernia or faecal impaction may give rise to this rare but serious condition. Colicky pain is usually a feature in the awake patient. Abdominal radiographs may show gaseous distension and air-fluid levels that are limited to a specific region of bowel, and there may be an absence of gas at the recto-sigmoid junction. Ischaemia and perforation may result if mechanical bowel obstruction is left untreated, and therefore these patients must be urgently referred for laparotomy, although sometimes volvulus of the sigmoid colon may be decompressed via colonoscopy.

#### Pseudo-obstruction:

Pseudo-obstruction results from colonic atony and dilatation, usually in the critically ill patient, and is difficult to distinguish from mechanical obstruction. Plain abdominal radiographs may show marked colonic distension, with relatively normal small bowel and gas extending to the recto-sigmoid junction. Although frequently self-limiting, the progressive distension may result in mesenteric ischaemia and perforation. Caecal dilatation of 12cm or greater is an indication for urgent colonoscopic or surgical decompression. If mechanical obstruction has been excluded, treatment with intravenous neostigmine (administered with appropriate doses of atropine) may be considered. However, this should only be performed with agreement of the CICU consultant anaesthetist and the advising consultant general surgeon.

### *Gastro-intestinal Haemorrhage*

This is usually caused by gastritis or acute stress ulceration of the stomach or duodenum. Other sources include severe oesophagitis or the large bowel (e.g. ischaemic colitis, angiodysplasia, bleeding diverticulum). Presentation is usually with melaena or dark red blood per rectum, or altered blood (“coffee grounds”) on nasogastric aspiration. Fresh blood from the NG tube suggests rapid upper GI bleeding, whilst minimally altered blood passed per rectum suggests either a lower GI source, or torrential upper GI haemorrhage.

Risk factors for stress ulceration include:

- Increased age
- History of peptic ulceration
- Prolonged period on cardio-pulmonary bypass, complex procedures, emergency surgery
- High inotrope/vasopressor requirements, intra-aortic balloon pump use
- Renal replacement therapy
- Coagulopathy/anticoagulation
- Preop fasting
- Prolonged mechanical ventilation
- Atrial fibrillation

Stress ulcer prophylaxis reduces the risk of GI ulceration and bleeding, but may increase the risk of gastric bacterial colonisation and ventilator-associated pneumonia (VAP). Therapy is therefore a balance of risks and benefits, but is probably justified in higher risk patients. Pre-operative ulcer prophylaxis should be recommended immediately after surgery. Although sucralfate has been associated with a lower incidence of VAP than ranitidine, it is less effective than ranitidine at preventing stress ulcers. The most effective agents are the proton pump inhibitors, and we use pantoprazole 40mg IV od, switching to lansoprazole 30mg nasogastric od when enteral feeding is established.

Patients with minor GI bleeding causing a fall in haemoglobin concentration over time require non-urgent upper GI endoscopy, whilst those with evidence of GI bleeding associated with haemodynamic instability or requirement for blood transfusion require urgent investigation. During working hours, the endoscopy suite (#3447) will have a rota for the gastroenterology registrar covering urgent endoscopies. Out of hours, the bleeding rota is covered by a gastroenterology consultant who is contacted through switchboard.

Consideration must be given to airway protection before and during the procedure, which may require tracheal intubation. Adequate cardiovascular resuscitation and correction of coagulopathy must also occur. Patients with confirmed gastric or duodenal lesions must be started on pantoprazole 80mg iv bolus followed by an infusion at 8mg per hour for 72 hours (Zargar *et al.* J Gastroenterol Hepatol. 2006;21(4):716-721). Indications for laparotomy include failed endoscopic attempts at haemostasis, recurrent bleeding, massive blood transfusion (> 6 units of red cells) and bowel perforation.



If upper GI endoscopy is normal in the face of clear GI bleeding, then colonoscopy must be performed. This is frequently unsuccessful due to the lack of bowel preparation, and then mesenteric angiography must be performed. Angiography and embolisation may also be considered as an alternative to laparotomy in patients who are considered poor candidates for surgery.

### *Mesenteric ischaemia and infarction*

Mesenteric infarction is a rare occurrence after cardiac surgery (0.5%) but has a high mortality (75%). It usually occurs between two days and three weeks after surgery. The spectrum of severity ranges from subclinical ischaemia to complete segmental or total gut infarction. It may affect any part of the small or large bowel. It is associated with a variety of mechanisms:

- Splanchnic hypoperfusion: This occurs in the setting of a low cardiac output state, and administration of exogenous vasopressors, especially in the context of hypovolaemia. Mesenteric atheroma, high venous pressures, as occurring with right ventricular failure or cardiac tamponade increase the risk.
- Arterial embolism: Intra-cardiac thrombus, material from ruptured aortic atheromatous plaques, or calcific debris from the aortic valve may give rise to distal embolisation.
- Arterial or venous thrombosis
- Gross bowel distension from any cause
- Aortic dissection

#### Warning Signs

Gastrointestinal dysfunction (e.g. no longer absorbing enteral feed), rising lactate and small volume bloody diarrhoea should raise suspicion of mesenteric ischaemia. This requires immediate discussion with the consultant on call, usually followed by referral to the most senior general surgeon in the hospital for urgent review. Urea, electrolytes, liver function tests and serum amylase tests must be performed. Continuous haemofiltration may mask the onset and extent of any lactataemia.

### *Acute cholecystitis*

This includes both calculous and acalculous cholecystitis. It is attributed to systemic hypoperfusion and the systemic inflammatory response which is associated with cardiac bypass through coagulation disorders, fluid removal towards the interstitial space, raised white cell count and complement activation. Treatment should be guided by surgical opinion and may be conservative or surgical depending on severity of the condition.

### *Acute pancreatitis*

This is rare but may be severe with a similar pathophysiological background to acalculous cholecystitis. Again, treatment should be guided by general surgeons.

### *Liver failure*

Liver dysfunction may affect up to 25% of cardiac surgical patients. Isolated raised liver enzymes may occur in mild forms but severe forms may consist of hypoalbuminaemia, malabsorption of hepatically metabolised drugs and abnormal coagulation. The causes include hypoperfusion, anaesthetic and inotropic drugs and mechanical pressure from low-placed vena cava inferior cannula. Conservative treatment includes control of fluids and electrolytes with replenishment of nutritive and coagulation factors. An urgent liver team opinion is important.

# Acute renal failure following cardiac surgery

Authors: Dr David Smith & Dr Kirstin Wilkinson

Revised: January 2016

The incidence of acute renal failure (ARF) after cardiac surgery has not changed since the 1960s. There is still no standardized definition of ARF, but it is usually quantified as an increase in serum creatinine, a reduction in creatinine clearance, or a decrease in urine output. On this basis the incidence of ARF after cardiac surgery is approximately 10% with a mortality of 10-20%, compared to 1% mortality for similar patients without ARF. However, sub-clinical renal injury, insufficient to influence routine biochemical parameters of renal function, probably occurs in all cardiac patients. The incidence of ARF requiring dialysis is approximately 2%, and this is associated with a mortality of 50-60%. Audit data from our own unit confirm these figures, with a median survival of only 20 months. ARF following cardiac surgery increases the risk of post-operative sepsis, gastrointestinal bleeding, neurological disturbance and myocardial infarction. ARF therefore results in a 2-3 fold increase in total hospital stay, increases the length of time spent in a high dependency facility, and triples the likelihood of discharge to an extended care facility.

## Risk factors

The presence of known preoperative risk factors accounts for only 30% of the incidence of ARF after cardiac surgery.

### Preoperative factors

Pre-existing renal dysfunction, and 20% of patients presenting for cardiac surgery fall into this category. Although these patients do not have a greater predisposition to perioperative renal injury, the small additional deterioration in renal function may precipitate a need for dialysis and result in progression to end-stage renal failure. Older adult patients have a greater risk of developing ARF. Other demographic risk factors include excess body mass, vascular disease, and diabetes. Previous cardiac surgery, active bacterial endocarditis, chronic obstructive pulmonary disease, and greater urgency for surgery also increase the risk, as do a low serum ferritin and low haematocrit.

### Perioperative factors

Increasing duration of CPB has consistently been associated with an increasing risk of ARF, with 180 minutes identified as the point at which the risk begins to increase substantially. Coronary artery bypass graft (CABG) operations have a relatively low incidence of postoperative ARF, whereas valve operations, combined valve replacement and CABG operations, and other operations associated with a long aortic cross clamp time or a period of circulatory arrest, have a greater risk. There is accumulating evidence that impaired renal oxygen delivery, rather than renal perfusion pressure, is the culprit in most cases.

### Postoperative factors

Indicators of reduced cardiac output in the peri- or post-operative period, such as intra-aortic balloon pump support, post-operative hypotension, significant inotropic support, and haemorrhage requiring re-operation are associated with an increased risk of ARF. These factors may indicate reduced renal perfusion, less successful surgery, or general deterioration of the patient. Early post-operative complications such as atrial fibrillation and sepsis are also associated with increased risk of renal dysfunction.

## Pathogenesis of ARF

The kidney is uniquely vulnerable to injury as a result of its anatomy and physiology, and cardiac surgery provides a plethora of renal insults. The clinical picture is too complex to be a simple story of either hypoperfusion or ischaemia-reperfusion injury. Contributory mechanisms are shown in the table below:

|                     |                              |                                |
|---------------------|------------------------------|--------------------------------|
| Medullary ischaemia | Neprottoxins                 | Renal vasoconstriction         |
| Interstitial oedema | Haemolysis                   | Renal arterial embolisation    |
| Tubular obstruction | Ischaemia-reperfusion injury | Systemic inflammatory response |

### Assessment of renal function

- **Urine output** increases linearly with arterial blood pressure, so is related to many factors including intravascular volume status and cardiac output, and is therefore not a sensitive index of renal function.
- **Creatinine** Around 50% of the excretory function of the kidney can be lost without an increase in plasma creatinine, and GFR can fall below  $30 \text{ ml}\cdot\text{min}^{-1}$  without urea retention. However, after a discrete renal injury the serum creatinine increases gradually to a new steady-state value despite non-progression of the renal injury, giving the impression that renal function is worsening progressively. However, changes in serum creatinine or creatinine clearance are still the most commonly used markers of renal dysfunction, and have been validated against long-term outcome after cardiac surgery in several large studies.
- **Sodium excretion** As renal perfusion decreases the kidney conserves sodium, so urinary sodium excretion tends to decrease in conditions leading to pre-renal failure. With intrinsic renal disease sodium resorption fails and the urinary sodium concentration increases. Fractional sodium excretion, the fraction of filtered sodium excreted in the urine, is the sodium clearance divided by the creatinine clearance. Although the estimate is awkward to perform, an oliguric patient with a fractional sodium excretion less than 1% has a pre-renal problem, while a fractional sodium excretion above 2% indicates intrinsic renal disease.

### Management of ARF

The management of ARF is supportive rather than curative. Adequate hydration and perfusion pressure, and maintenance of cardiac output in the post-operative period are all beneficial, although there is always a balance to be struck between the desire to remove water and the risk of hypovolaemia (bad for the kidney). Patients with a low serum albumin will always have peripheral oedema that will not resolve until their albumin recovers.

Many drugs have been studied for the prevention of ARF but none has been conclusively proven to be effective. The three most extensively studied agents have been dopamine, mannitol and furosemide.

**Dopamine** ‘Low dose’ dopamine is the most highly debated strategy for prevention of ARF. Dopamine produces a dose dependent increase in renal blood flow over the dose range  $0.5\text{-}3.0 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ . However, the  $\alpha$ - and  $\beta$ -adrenergic effects of dopamine are still present at this dose rate so it is likely that there is no true “renal dose”. Although dopamine does increase the urine output, recent studies have suggested that dopamine affords no renal protection to patients undergoing cardiac surgery, although a clinically relevant effect may occur during the pre-CPB phase of surgery. Dopamine has potentially harmful side effects and there is some evidence that it may exacerbate renal tubular injury in the early post-operative period.

**Mannitol** was the earliest pharmacological prophylactic agent for ARF after cardiac surgery because it produces an osmotic diuresis. Unlike loop diuretics mannitol retains its effect as the GFR decreases, and is independent of the action of anti-diuretic hormone. Although mannitol produces a transient increase in urine output, even in oliguric patients, its ability to prevent ARF is less clear. A diuretic response to mannitol may be a predictor of outcome rather than an effect of therapy. Mannitol is often put into the cardiopulmonary bypass prime so most patients here will have received some.

**Furosemide** Despite the theoretical attractiveness of renal ischaemia secondary to hypoperfusion as the primary pathophysiological cause of ARF, loop diuretics such as furosemide, used to increase urine flow and reduce renal medullary oxygen consumption, are ineffective at preventing ARF. This may be because less than 10% of an administered dose reaches the renal tubules in ARF. Continuous infusion may be better than bolus dosing, since the diuretic effect is a function of exposure time to the drug, but the optimum dose is unknown.

### *Indications for haemofiltration*

When pharmacological methods of augmenting renal function have failed and the patient is anuric or urine output is inadequate to sustain metabolic balance, consideration should be given to renal replacement therapy. In our unit we use continuous veno-venous haemofiltration or haemodiafiltration via a dedicated large bore catheter. The ideal cannulation site is the right internal jugular vein, followed by the left subclavian, as these approaches provide the straightest lie of the catheter. We prefer to avoid the femoral route if possible.

Renal replacement therapy may be indicated for fluid volume control, or for biochemical derangements such as persistent hyperkalaemia, acidosis, or a serum creatinine above  $400 \mu\text{mol.l}^{-1}$ . The decision to begin renal replacement therapy should always be made in discussion with the consultant intensivist, and should include a plan for fluid volume management. Normothermic patients in the ICU will have a fluid deficit around 500ml per day in 'insensible' loss from the airway, gut, and as sweat. This loss increases by 100 ml for every  $0.5^{\circ}\text{C}$  above  $37^{\circ}\text{C}$ , and also increases by 300-1000 ml per 24 hours if there is visible sweating. Fluid also accumulates in the GI tract if there is an ileus, and is lost from it if the patient has diarrhoea.

Specific anticoagulation plans for patients needing filtration should be discussed with the consultant intensivist. If filtration is required in the first 12 hours or so after cardiac surgery the anticoagulation will usually be omitted.

### *Conclusion*

Acute renal failure continues to affect a significant number of patients following cardiac surgery. Despite the theoretical attractiveness of several strategies for the prevention of renal dysfunction, there is no current strategy known to reliably reduce its incidence.

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# End of Life Care

Authors: Dr Paul Diprose & Pascale Withey (Transplant coordinator)

Revised: April 2015

Whilst overall mortality within critical care units remains at 20%, less than 5% of patients admitted to our CICU will die while on the unit. It is unusual for patients to suddenly arrest and die on the unit. This means that most deaths that occur are relatively expected and can be actively managed to ensure that patient dignity and comfort can be maintained, and relatives can be taken through the grieving process in a compassionate way.

## *Withdrawal or Limitation of Therapy*

The decision to withdraw or limit any therapies of CICU patients will be taken only by the consultant in charge of CICU in consultation with the wider team and family. Decisions made will be on the basis of the *best interests* of the individual patient. No decision to withdraw or limit therapy should be made on CICU without first discussing with the consultant on duty for CICU. It is very important that once a decision has been made to either withdraw or limit therapy that this is clearly documented in the patient's clinical notes.

## *Palliative Care*

For some patients, it may be appropriate to involve and get advice from the hospital palliative care team. This should be discussed with the consultant in charge first.

## *Organ Donation*

Most patients that die on CICU do so from multiple organ failure, frequently in the presence of sepsis. This situation may change with the increasing numbers of patients admitted to CICU after sustaining an out of hospital arrest. Although the opportunity for organ donation is rare on CICU it should be considered as part of end of life care. (NICE 2011, GMC 2010).

All patients should be discussed with the Specialist Nurse Organ Donation (SNOD):

- Where there is an intention to perform brain stem tests
- Where there is an intention to withdraw life sustaining treatment in patients with life threatening or a life limiting condition which will, or is expected to result in circulatory death.

The SNOD team can be contacted on pager 07659 183499.

Tissue donation following death may also be a possibility and the SNOD team will also be able to provide advice of a patient's suitability.

Always discuss with the duty CICU consultant before embarking on discussions around this area with the SNOD. Please see appendix 5 where a summary of the information given to relatives regarding post-mortem tissue donation is presented.

## *Certification of Death*

After death you may be asked to certify the death of the patient. This should be clearly recorded in the clinical notes with the date and time that it was completed. It is the responsibility of the duty CICU consultant to discuss the case with the IMEG team and (where appropriate) the coroner, to complete a death certificate and the cremation form. Fellows or registrars should only do any of these tasks if specifically asked to do so by the duty CICU consultant, not by any other member of the team.

## *Relative liaison*

We have specific patient information leaflets regarding end of life care and death on CICU with relevant contact details. These will usually be issued by the nurse in charge but you should ensure that relevant information has been delivered to permit handover to bereavement services.

### *Further follow-up after a death*

On the next working day after a death has occurred on the unit there should be liaison with the consultant surgeon or his team to inform them of the death (although generally this would have occurred at the time of death). The G.P. surgery should also be informed of the death (this will usually be performed by the bereavement care team).

All deaths that occur on CICU will need to be discussed with the IMEG team on the next working day after death. This is a meeting chaired by the associate medical director for patient safety where the circumstances of the death are briefly discussed to ensure that any learning points are rapidly picked up. The meetings occur between 08:30hr and 09:30hr every morning and (by appointment with bereavement care) in the afternoons. A decision as to whether to discuss with the coroner's officer (the usual situation in post-op patients) is made at the IMEG meeting.

If the coroner is content for issue of a death certificate then this should be completed together with a cremation form by someone who has seen and examined the body after death.

### *Checklist after death*

- Appropriate documentation including tissue donation explained and leaflet given to relatives
- Death discussed at the next IMEG meeting
- Outcome from IMEG meeting written in the clinical notes
- Discussed (where appropriate) with Coroner's officer
- Surgical team aware
- G.P. surgery informed
- Death certificate written (if not a Coroner's case)
- Cremation form written (if not a Coroner's case)
- All the above documented as completed in the patient's clinical notes

# Emergency Patient Management

## Hypotension after Cardiac Surgery

Authors: Dr Tom Pierce & Dr Kirstin Wilkinson

Revised: June 2015

Acceptable ranges of normal pressures after cardiac surgery are:

Systolic pressure 100-140 mmHg

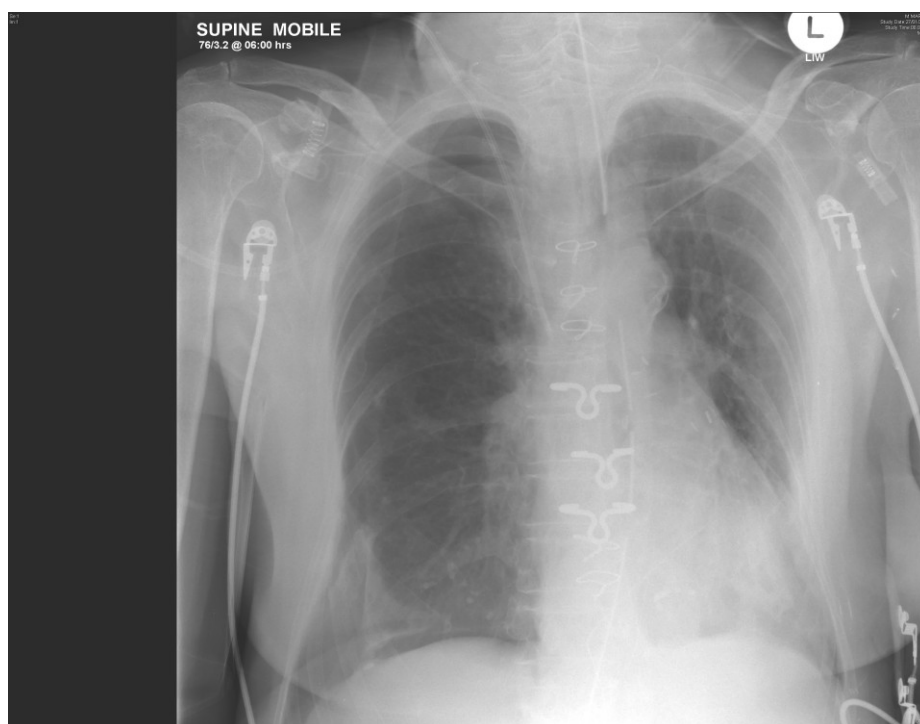
Mean arterial pressure 65-85 mmHg

The anaesthetist from theatre will more than likely have set the haemodynamic parameters based on the operation and the patient's pre-morbid state. The patient and these parameters should be reviewed regularly to ensure the haemodynamic aims remain appropriate.

Hypotension results from either a fall in stroke volume (SV), and/or a fall in systemic vascular resistance (SVR).

Deciding which of these is the cause relies on a careful history, examination, investigations and, where indicated, invasive monitoring.

- **The perfusion chart** may give a clue, if, for example, the perfusionist has had to give 10mg of phenylephrine during bypass to maintain the systemic perfusion pressures, and then the SVR will have been low and may still be.
- **The anaesthetic chart** may reveal doses of vasodilators that may still be active postoperatively.
- **The nurses' observation chart**, an excessive loss from the chest drains may indicate hypovolaemia.
- **Your examination of the chest** may reveal reduced breath sounds on one side indicating a tension pneumothorax.
- **A chest radiograph** may confirm your suspicion of a tension pneumothorax,



Note from this image, the displaced trachea to the left and shift of the heart to the left so that the spine is visible to the right of the heart. The pneumothorax is not easy to see on the right (especially on the screens in the CICU); however in the setting of hypotension and elevated central venous pressure (CVP), the diagnosis was made. The image below followed chest drain insertion. Note the resolution of mediastinal shift and the re-expansion of the right lung.



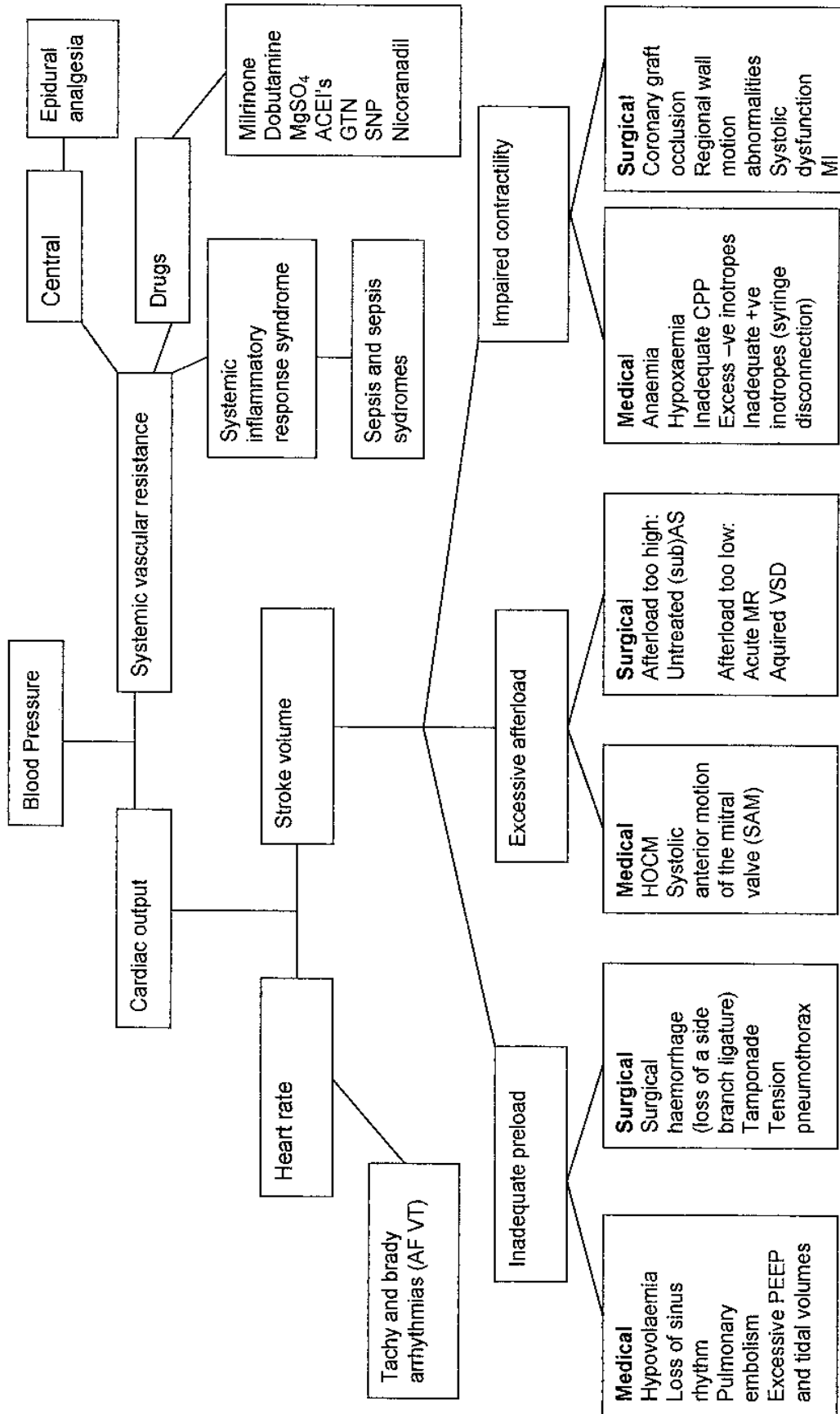
*If the nurses call you to see a postoperative hypotensive patient.....*

- Review the patient's history, charts and drugs
- Examine the patient
- Note the BP and CVP, pulse rate and rhythm
- Examine the heart
- Look at the chest drains and the rate at which they are filling
- Look at the ventilator and the inflation pressures
- Examine the chest and lung fields
- Consider additional investigations where indicated
  - ECG (rhythm and ischaemia)
  - Chest radiograph
  - Transthoracic or transoesophageal echocardiogram
- Call for consultant advice
- If time and the clinical condition permits, a pulmonary artery catheter may be useful to measure pulmonary capillary wedge pressure (PCWP) or to derive SVR.

The guide overleaf, summarises the causes of hypotension.



**A GUIDE TO THE MANAGEMENT OF POSTOPERATIVE HYPOTENSION**



# Cardiac Arrest after Cardiac Surgery

Authors: Prof Charles Deakin and Dr Paul Diprose

Revise: April 2015

The incidence of cardiac arrest after cardiac surgery is around 0.7 to 2.9%; in contrast to other causes of in-hospital arrest, the survival to hospital discharge is 17-79%. This is because there are a high proportion of reversible causes of arrest after cardiac surgery.

There are important differences from standard resuscitation in the management of patients who arrest in the first ten days after cardiac surgery. This is not least because rapid re-sternotomy should be considered in all patients who remain arrested after basic procedures such as institution of pacing or cardioversion. Figure 1 summarises the basic steps to be undertaken once an arrest has been diagnosed. Figure 2 shows the key personnel that will need to be present to undertake emergency chest re-opening. There is a detailed paper on this subject on file in the Fellow's resource room.

## *Diagnosing and Calling Cardiac Arrest*

- If the ECG shows VF or asystole (with no arterial pressure waveform) then immediately call the cardiac arrest
- If the ECG is compatible with a cardiac output feel for a pulse and check the arterial pressure waveform. If there is no pulse and no pulsatile arterial pressure waveform then call the cardiac arrest
- Ensure that someone rings ext 2222 and states 'Cardiac arrest in Cardiac Intensive Care', this will ensure that the cardiac surgical registrar will be fast-bleeped to CICU

## *Airway*

- Immediately turn the oxygen up to 100%
- Check for breath sounds to attempt to exclude a haemothorax or pneumothorax
- Turn off PEEP on the ventilator

## *Syringe Drivers*

- In an established cardiac arrest turn off infusions and syringe drivers
- You should however continue to administer sedative drugs

## *Resuscitation Drugs*

You should have adrenaline available BUT be cautious with its use and consider titrating in doses of no more than 100mcg at a time to avoid risk of provoking additional myocardial ischaemia or of severe hypertension leading to graft disruption

## *Intra-aortic Balloon Pumps*

- Should be generally set to pressure trigger during the arrest
- If there is a significant time without cardiac massage triggering should be changed to internal fixed delivery of 100 bpm

### *VF and Pulseless VT*

- 3 sequential shocks (150J Biphasic) should be delivered as soon as possible and without intervening CPR
- Emergency re-sternotomy should be performed after 3 failed attempts at defibrillation
- 300mg of Amiodarone should be given via the central line if 3 failed attempts at defibrillation have occurred

### *Asystole and Bradycardia*

- Connect the epicardial pacing wires and set to DDD at rate of 90 bpm and the maximum atrial and ventricular output voltages
- Atropine should be given at a dose of 3mg via the central line

### *Pulseless Electrical Activity (PEA)*

- If the rhythm is PEA and a pacemaker is connected then briefly disconnect this to exclude underlying VF
- In non-shockable cardiac arrest emergency chest re-opening should be performed

### *Emergency Re-sternotomy*

- See 'Chest re-opening' chapter in this manual
- Internal cardiac massage is superior to external cardiac massage
- A re-opening set is kept on CICU (ensure that you familiarise yourself with the location and types of equipment available)
- It is usual to perform an anti-septic 'washout' after sternotomy and to give additional antibiotics (usually vancomycin)

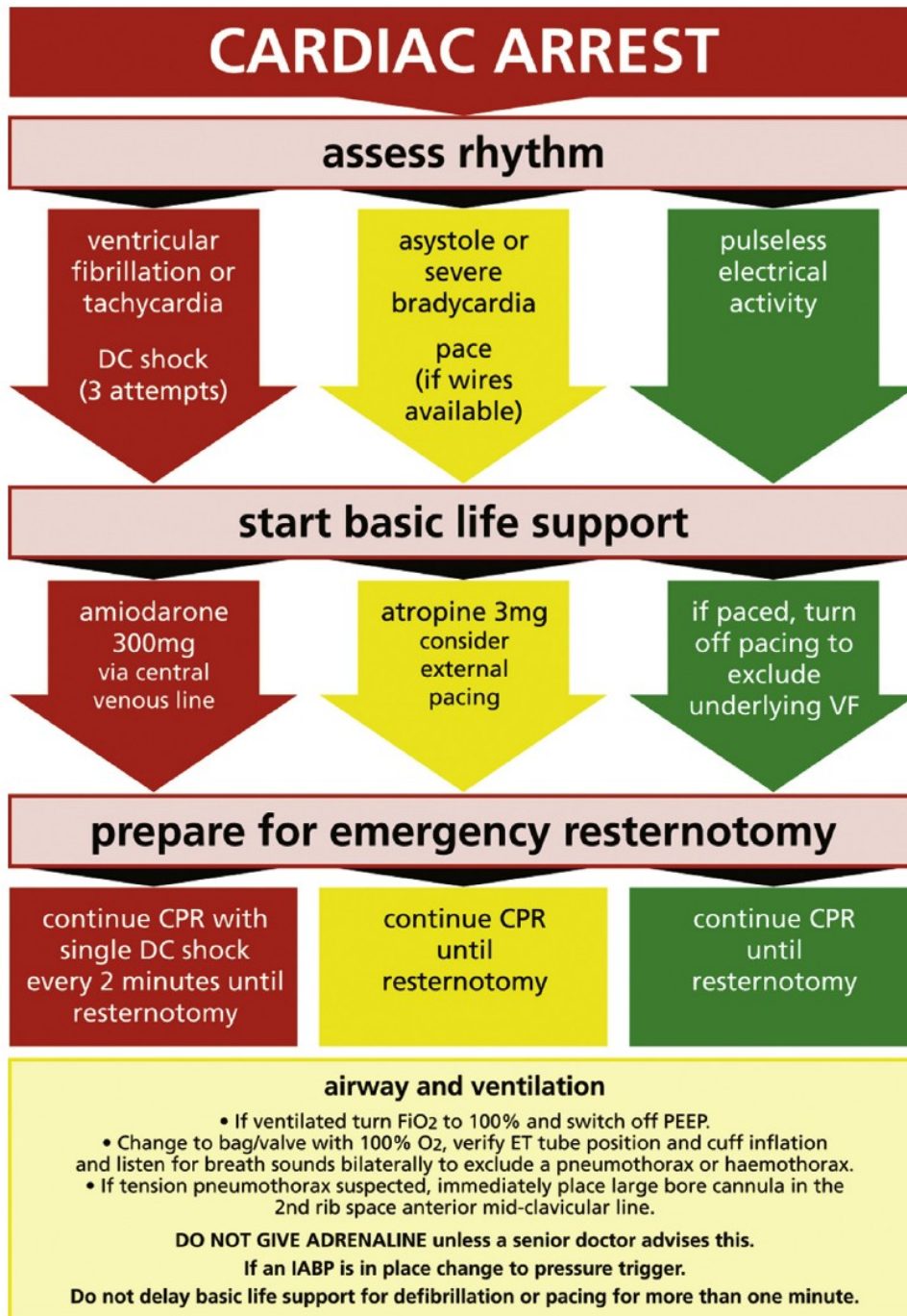


Figure 1: A guideline for the management of a Cardiac Arrest after Cardiac Surgery

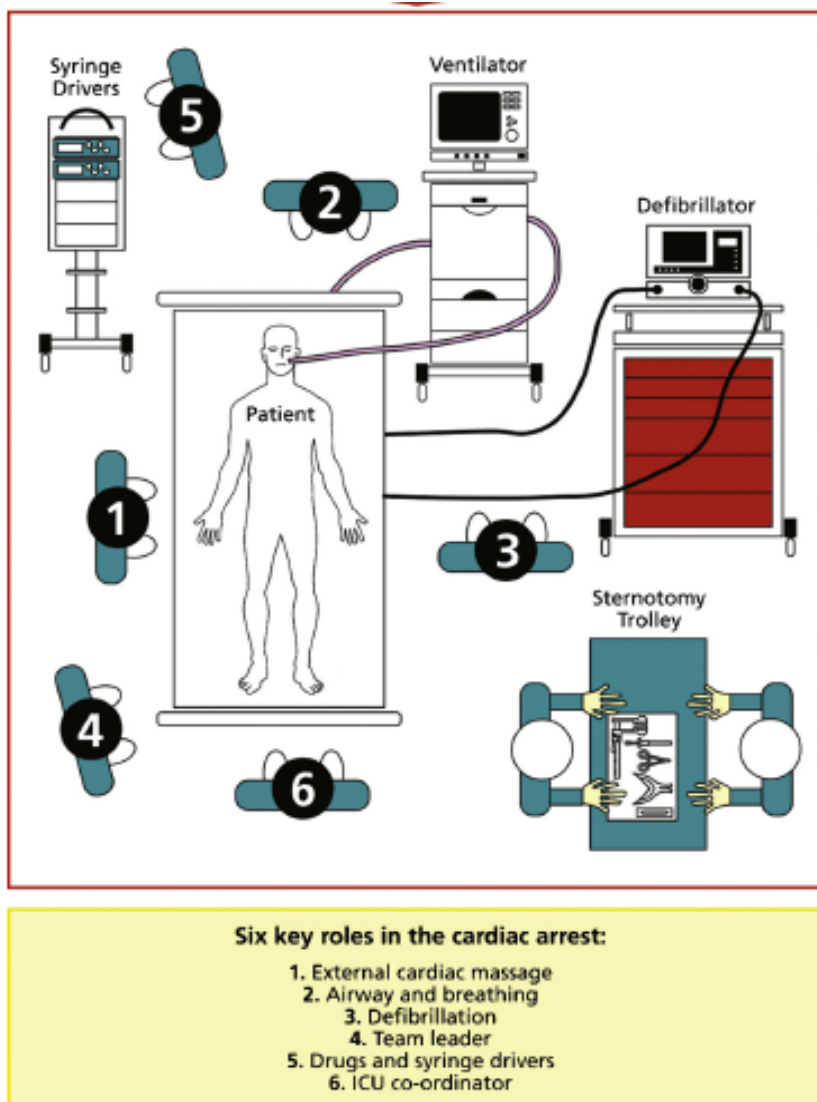


Figure 2: The six key roles for personnel during a cardiac arrest on CICU

# Emergency Chest Re-opening on CICU

Authors: Dr Tom Pierce & Dr Kirstin Wilkinson

Revised: December 2015

From time to time it is necessary to reopen the chest following cardiac surgery.

The most common indications are:

- Cardiac tamponade
- Massive haemorrhage
- As part of resuscitation in the postoperative patient when the diagnosis is uncertain

Although stressful, it should be regarded as no more than a rather urgent operation combined with resuscitation and possibly bypass. The outcome depends on the underlying problem and the patient's response to the management. The following is a scheme or aid memoir for the heat of the moment. Direct from the top end moving the bed away from the wall if necessary. Co-ordinate external or internal cardiac massage. Follow current guidelines for resuscitation (see previous chapter).

## Personnel

- Call 2222 and ask for the chest opening team. This should bring the theatre nurses and ODP.
- It will usually be one of the cardiac surgical registrars who opens the chest
- Identify a nurse or nurses whose responsibility is to act as your assistant(s). Try to limit the numbers in the room to only those who are necessary. The senior CTITU nurse should be present.
- Call the consultant cardiac anaesthetist
- Think of calling a perfusionist if cardiac bypass likely to be needed. Bear in mind, the time it will take for them to arrive and set up a pump.

## Anaesthetic and other drugs

- The patient needs to be anaesthetised
- Sedatives, propofol or midazolam in appropriate dose for the patient and the circulation.
- Relaxants, as indicated.
- Analgesia, fentanyl is indicated.
- Antibiotics, usually 1g Vancomycin by infusion over 2 hours (unless contra-indicated)
- Have adrenaline pre-filled syringes available BUT only use in increments of 50-100mcg (0.5 to 1ml) because of the risk of pressure 'over shoot' and damage to surgical suture lines especially if opening the chest releases a cardiac tamponade
- You may need heparin for the rare case where CPB is required (20-30,000 units)

## Fluids and Equipment

There is a chest re-opening trolley on CTITU. It is worth familiarising yourself with its contents. Ensure you have the following:

- 2 drip stands to take among other things the blood brain barrier
- The drip on your side of the erected blood brain barrier
- Blood warmer
- Blood and gelofusine
- Pressure bags
- The pacemaker if attached



### **Airway**

- Most patients needing emergency reopening will still be intubated.
- Ventilate the lungs with 100% oxygen via an ETT
- Have a sucker to hand
- Beware of disconnections

### **Investigations**

When the initial dust has settled think of the following:

- Blood gases
- Potassium
- Haemoglobin
- Cross match
- Coagulation profile incl. INR APTR platelets fibrinogen
- Thrombelastogram
- Blood products and protamine as indicated

# Procedures and Investigations

## TEG and Near Patient Coagulation Testing

Authors: Dr A Richardson & Dr K Wilkinson

Revised: April 2015

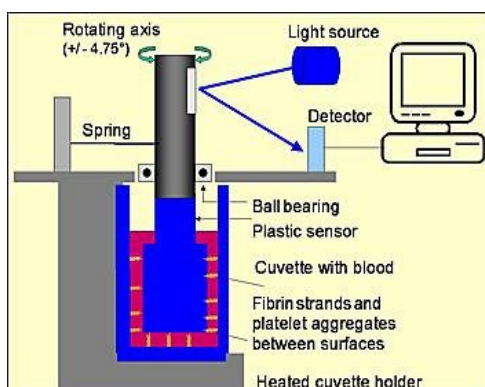
### Point-of-care haematology and coagulation testing facilities on Cardiac ICU

In the cardiac laboratory outside CICU, we have machines to perform TEGs, as well as point-of-care testing for full blood counts and conventional coagulation screens (INR, APTR, and fibrinogen). Platelet function analysis is also possible with the Multiplate machine located in E level theatre laboratory. Teaching regarding the use of these machines is provided through the perfusion department.

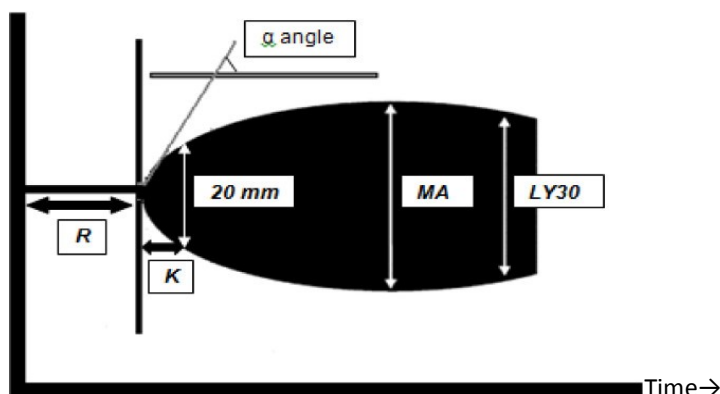
### Principles of TEG

TEG measures the whole process of blood coagulation by using the viscoelastic changes of blood that are associated with fibrin polymerisation.

A cuvette filled with whole blood and a pin, suspended by a torsion wire, is lowered into the sample. Clot formation is induced by putting whole blood in a low shear environment (which mimics sluggish venous flow). The patterns of shear-elasticity alteration reveal the kinetics of clot formation & growth, and the strength and stability of the formed clot. The kinetics of clot formation reflect the adequacy of quantitative factors available, whilst the clot strength/stability reveal the ability of the clot to perform the work of haemostasis. As a blood sample placed between the cylinders clots, it forms a mechanical bond between the inner and outer cylinders of the thrombelastograph such that the oscillatory motion of the outer cylinder is imparted to the inner cylinder. A plot of the movement of the inner cylinder is known as a thrombelastogram.



Variation over time of the clot strength results in different thrombelastogram profiles that are useful for diagnosis. The electrical signal from the coupled cylinders is amplified to create a TEG trace, with the result displayed graphically. The deflection of the trace increases as clot strength increases & decreases as clot strength decreases.

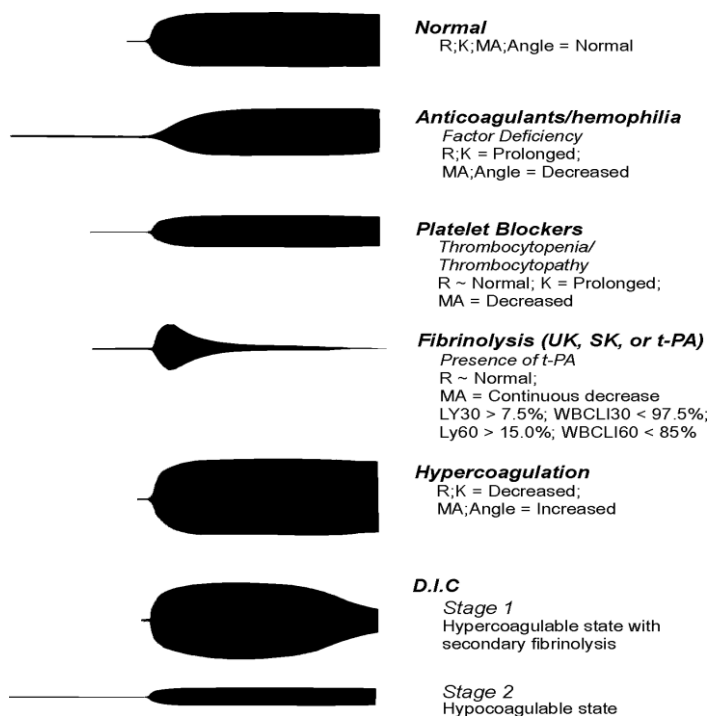


Schematic TEG waveform



What information does TEG give us?

| Parameter              | What is measures  | Increased by  | Decreased by  |
|------------------------|---|---|---|
| R time                 | Time from start of measurement to start clot formation. Reflects start of clotting, thrombin formation and fibrin polymerisation                              | Factor deficiency<br>Anticoagulation<br>Severe hypofibrinogenaemia<br>Severe thrombocytopenia | Hypercoagulability syndromes                                |
| K time                 | Time from start of clot formation to amplitude of 20mm. represents fibrin polymerisation and clot stabilisation with platelets and Factor XIII                | Factor deficiency<br>Thrombocytopenia<br>Thrombocytopathy<br>Hypofibrinogenaemia              | Hypercoagulability state                                    |
| Angle ( $\alpha$ )     | Measures the rapidity of fibrin build-up and cross-linking (clot strengthening)   | Hypercoagulable state   | Hypofibrinogenaemia or thrombocytopenia                     |
| Maximum amplitude (MA) | A direct function of fibrin and platelet bonding via GPIIb/IIIa, reflecting ultimate clot strength. Correlates with platelet numbers & function, & fibrinogen | Hypercoagulable state   | Thrombocytopenia<br>Thrombocytopathy<br>Hypofibrinogenaemia |
| LY30 or LY60           | Measures % decrease in amplitude 30 or 60 minutes post-MA, thereby giving a measure of the degree of fibrinolysis. Normal range < 7.5%                        |   |   |



#### Advantages of TEG

- Uses whole blood, which may be native or citrated
- Provides results within 10-20 min: short turnaround time
- High negative predictive value (90-98%) on surgical bleeding versus coagulopathy
- Has been shown to reduce transfusion requirements
- Can differentiate between factor deficiency and inadequate heparin reversal with the use of heparinase cups

#### Limitations of TEG

- Cannot detect deficiency of vWF
- Poor at detecting platelet dysfunction
- Cannot detect mild/moderate haemophilia
- Low/no sensitivity to visualize the potential haemostatic effect of rFVIIa, Haemate, vWF, tranexamic acid
- Takes no account of the anti-coagulant effects of *in vivo* acidosis or hypothermia in a sick patient

## Multiplate™ Platelet Function Analysis

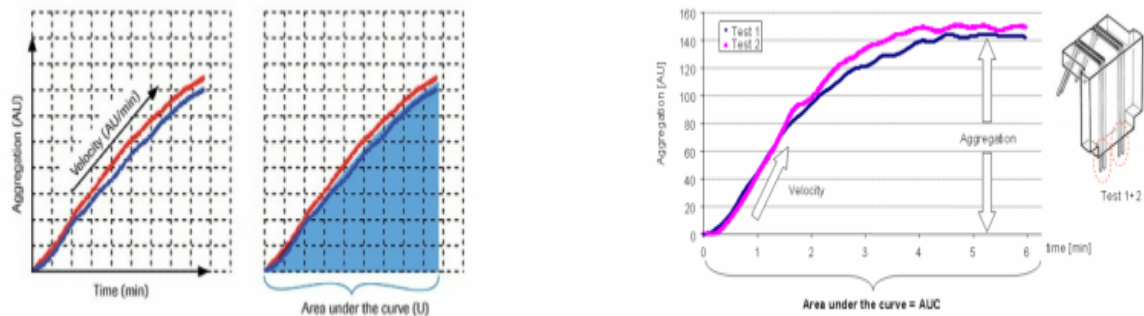


This is a new method of assessing platelet function that is based on impedance aggregometry. There is a machine available in the theatre lab but is only to be used by the clinical perfusionists. Platelet rich plasma (PRP) is stirred within a cuvette located between a light source and a detector. After addition of a various panel of agonists, e.g. arachidonic acid, ADP, the platelets aggregate and light transmission increases.

Various states of platelet activation are visible, such as spreading of platelets on the surface as well as platelets aggregated with each other. Several test reagents are available to allow triggering of different receptors / signal transduction pathways of the platelet in order to detect its function or drug effects.

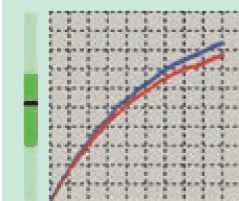
| Test            | Measures  | Drug effect detected  |
|-----------------|---|---|
| <b>ASPttest</b> | Activation by arachidonic acid, the substrate of the cyclooxygenase (COX). COX forms Thromboxane A2 (TXA <sub>2</sub> ) which is a potent platelet agonist          | Aspirin   |
| <b>TRAPtest</b> | TRAP-6 stimulates the thrombin receptor on the platelet surface. Thrombin is a very potent platelet activator. Its action is not blocked by Aspirin® or clopidogrel | Glycoprotein 2b/3a receptor antagonists e.g. abciximab, tirofiban |
| <b>ADPttest</b> | ADP stimulates platelet activation by the ADP receptors. The most important ADP receptor (P2Y <sub>12</sub> ) is blocked  | Clopidogrel, prasugrel and ticlopidine                            |

Multiplate continuously records platelet aggregation. The increase of impedance by the attachment of platelets onto the Multiplate sensors is transformed to arbitrary aggregation units (AU) and plotted against time. The most important parameter calculated is the **area under the aggregation curve (AUC)**.



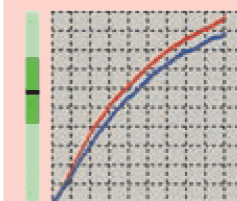
Healthy individual with no platelet inhibition

### ADPttest



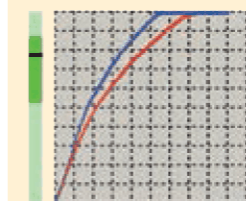
94 U

### ASPItest



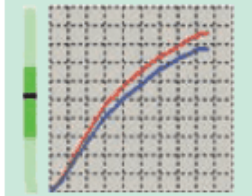
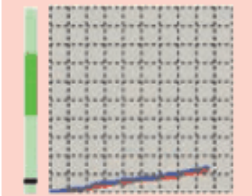
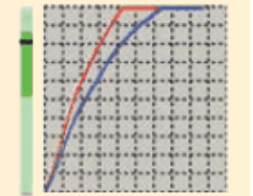
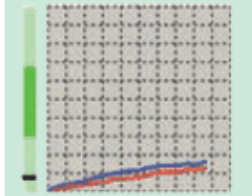
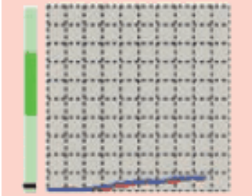
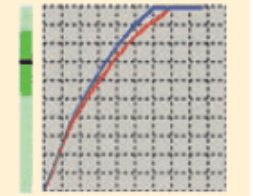
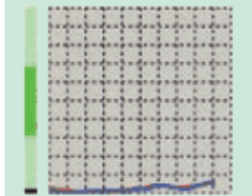
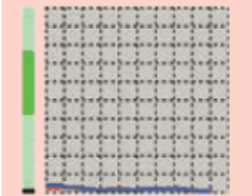
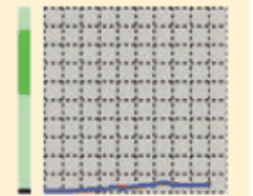
105 U

### TRAPtest



140 U

**Medication**                      **Normal responder**

|   | ADPtest  | ASPItest  | TRAPtest  |
|---|--|---|---|
| <b>Aspirin<sup>®</sup></b><br>100 mg aspirin                                    |  <p><b>92 U</b></p> |  <p><b>10 U</b></p> |  <p><b>146 U</b></p> |
| <b>Aspirin<sup>®</sup> + clopidogrel</b><br>75 mg clopidogrel<br>100 mg aspirin |  <p><b>14 U</b></p> |  <p><b>5 U</b></p>  |  <p><b>130 U</b></p> |
| <b>GPIIb/IIIa antagonist</b><br>32 mL/h tirofiban                               |  <p><b>3 U</b></p> |  <p><b>2 U</b></p> |  <p><b>4 U</b></p>  |

*Multiplate AUC: 1U = 10 AU\*min*

# Cardiac Output Monitoring

Authors: Dr Andrew Richardson & Dr Kirstin Wilkinson

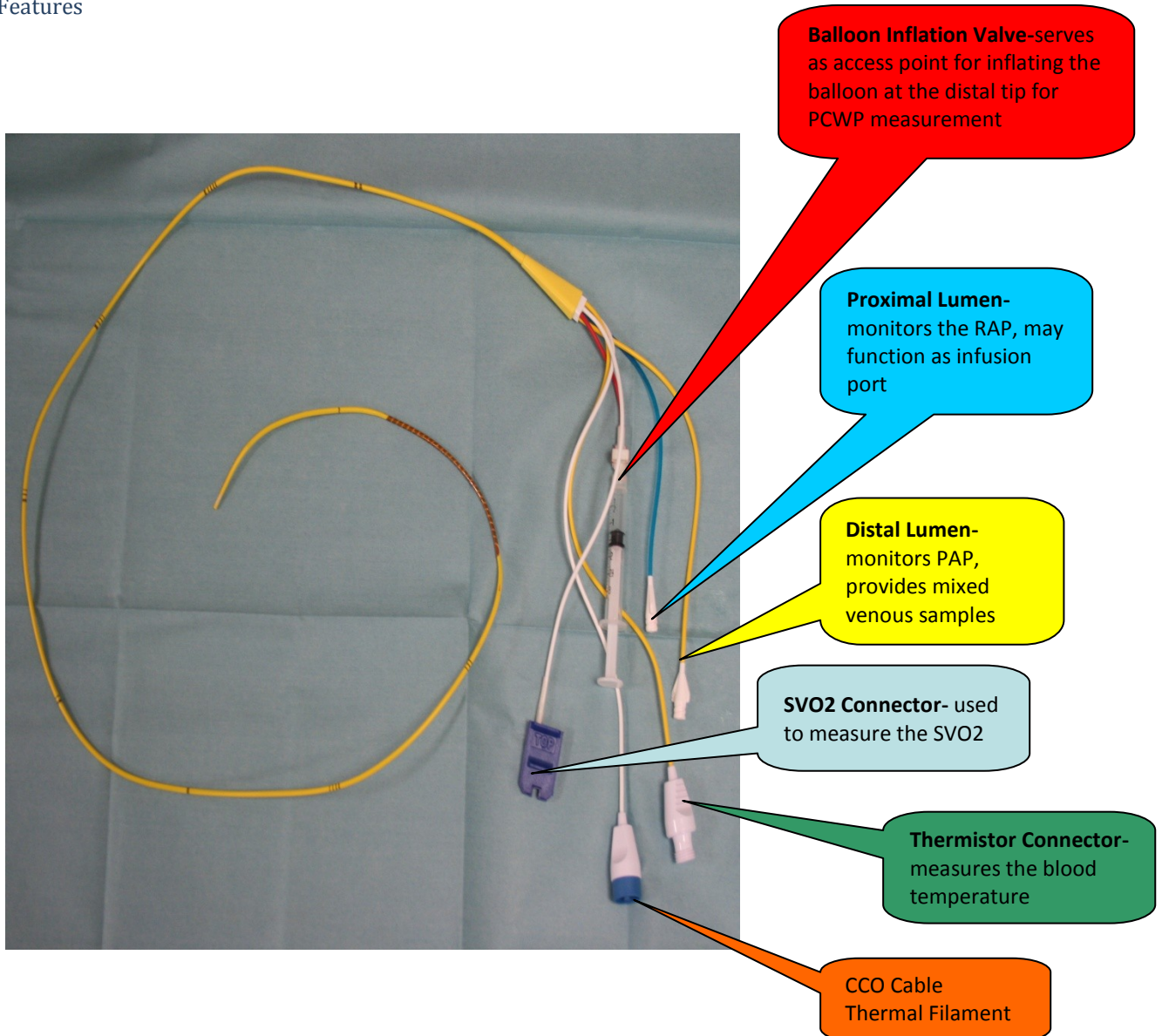
Revised: April 2015

## Pulmonary Artery Catheter

The pulmonary artery catheter (PAC), also called a Swan-Ganz Catheter, is a flexible, balloon-tipped, flow directed catheter that is guided through the right side of the heart and into the pulmonary artery.

It has been in clinical use since 1970, and its use has largely been superseded in non-cardiac critical care practice by less invasive methods of cardiac output monitoring. However, most pulse contour-derived methods of cardiac output (CO) measurement assume relatively small changes in systemic vascular resistance over time – an erroneous assumption in patients that have cooled on bypass, re-warmed, and who then may develop a variable degree of systemic inflammatory response or low cardiac output state. The PAC thermodilution method of CO measurement provides a more reliable of CO estimation, and also allows measurement (& therefore targeted treatment) of elevated pulmonary artery pressures.

## Features



## Indications for PAC Insertion

- Hypotension unresponsive to fluid and empirical inotropic support, where additional haemodynamic information is needed
- Severe pulmonary hypertension requiring treatment
- Pathology that means right heart pressures may not accurately reflect left sided filling (e.g. MS, pulmonary vascular occlusive disease, etc), and more information is needed

## Placement

Placement of PACs is potentially dangerous: it should not be attempted without discussion with the consultant on call. In any case, if the patient is sick enough to require a PAC, then they require senior review.

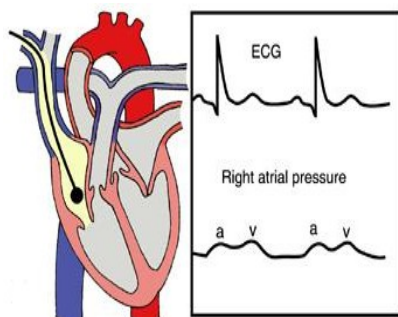
Furthermore, although the nursing staff may not be able to assist you with PAC insertion, most of the senior nurses have seen enough to know how it is done, and most importantly - when it is being done wrong! If the nurse is worried – listen.

A PA introducer is inserted into a central vein with the same technique as for any central venous cannulation.

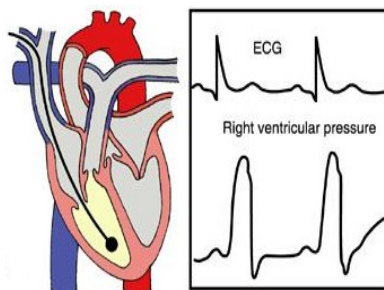


A third transducer is set up and zeroed. The PAC is removed from its packaging in a sterile fashion. Place (but do not unfold) the sterile catheter sheath over the PAC at this stage, before it is forgotten. Having to remove a PAC to apply the sheath after inserting a PAC perfectly on the first attempt is tedious! Three way taps are attached to the distal and proximal lumens, and a sterile manometer line is passed from the operator to the nurse assisting, and both lumens are flushed.

## Waveforms seen during insertion

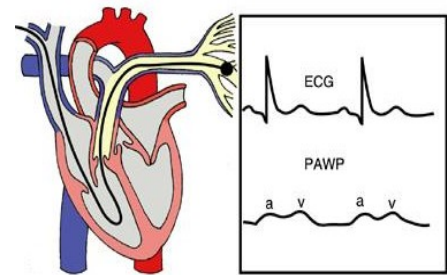
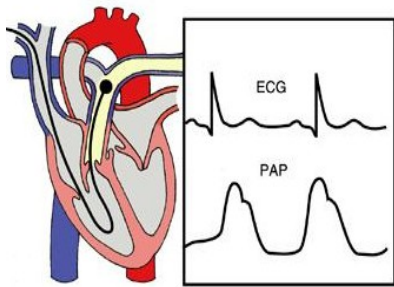


**Right Atrium**  
Classical CVP waveform  
Usually at 20 – 25 cm



**Right Ventricle**  
Sudden increase in systolic pressure, wide pulse pressure & low diastolic pressure that approximates CVP  
Usually at 30 – 35 cm





**Pulmonary Artery Usually at 40 – 45 cm**

Passage through the RVOT and PV is often accompanied by dysrhythmias. A dicrotic notch & a sudden step-up in diastolic pressure are seen. However, pressure values are not always easily distinguishable

The key is the different waveform – diastolic filling of the RV produces a steadily increasing diastolic pressure, whereas a steady decrease is seen in the PA diastolic pressure as blood flows towards the LA.

**Pulmonary Capillary Wedge Pressure**

Usually at 45 – 55 cm

Resembles a CVP trace

Should be measured as average of several cardiac cycles, at end-expiration

On the monitor, select the PA pressure tab, and go to 'PA Wedge/Insert'. Insert the PAC until a CVP trace is obtained. Inflate the balloon (do not use more than 1.5ml of air), and with bold movements (~ 3 cm at a time) insert the catheter, observing for the characteristic pressure trace changes.

Watch the insertion distance all the time, and do not significantly over-insert for any of the expected waveforms, to reduce the risk of catheter looping and knotting. If the desired position has not been found and the catheter is inserted more than 10 cm than the expected distance, deflate the balloon and withdraw, before reinserting. **Do not ever withdraw the catheter with the balloon inflated.**

Once wedged, let down the balloon and check that the trace is un-wedged, that a good PA pressure trace is seen, and that the catheter is not intermittently 'self-wedging' – if these things are not present, the PAC should be withdrawn a few centimetres. Ensure that PA catheter syringe is always deflated and left in the "locked" position. Under no circumstances should the balloon be left wedged for a prolonged period – this may lead to pulmonary infarction or PA rupture. When documenting the procedure, record the PA catheter length at its final position.

If the PAC is being used during cardiac surgery it should be withdrawn 5 – 10 cm prior to the onset of bypass, to reduce the risk of catheter migration & pulmonary infarction & PA rupture.

The PAC is then attached to the Vigilance cardiac output monitoring system, and the SvO<sub>2</sub> monitoring is calibrated with an *in vivo* mixed venous blood gas.



**Measured and Derived Values**

Derived values are usually subject to considerable error, and are only useful for confirming your clinical opinion. No significant treatment decisions should be made purely on the basis of a number!

Cardiac output or index (cardiac output adjusted for body surface area)

- Normal CI 2.7 – 4 l/min/m<sup>2</sup>
- CI > 2.0 l/min/m<sup>2</sup> is usually enough to sustain life

### Pulmonary Capillary Wedge Pressure

- Normal PCWP 5 – 15 mmHg

Absolute readings are of little use, unless wildly elevated – the response to small (100 ml) fluid boluses are more valuable

If the PCWP appears to be greater than the PA diastolic pressure, and the pressure trace varies markedly with respiration, it is likely that the catheter tip is mal-placed in an area of lung where alveolar pressure exceeds pulmonary arterial pressure (West zone I) or pulmonary venous (West zone II): under these circumstances the PCWP is artefactually elevated

### PA pressures

- Normal 15 – 30/5 – 15 mmHg

### Oximeter-measured SvO<sub>2</sub>

- Normal > 60%

### Systemic Vascular Resistance

- Normal 770 – 1500 dyne s/cm-5

### Pulmonary Vascular Resistance

- Normal 100 – 250 dyne s/cm-5

### Potential Contra-Indications to PAC Placement

#### Tricuspid or Pulmonary Valve Stenosis

- Catheter may be difficult/impossible to pass and may lead to a significant reduction in venous return

#### Presence of a prosthetic tricuspid or pulmonary valve

- Catheter may be entangled in the valve mechanism

#### Right atrium or right ventricle mass

- Tumour or thrombus may be dislodged by the catheter leading to pulmonary embolism

#### Cyanotic heart disease

- Pulmonary blood flow is reduced and therefore a flow directed catheter is more likely to follow the bulk flow to the systemic side of the circulation

### Complications of PAC Monitoring

- Dysrhythmias (transient – 50%, sustained – 3%)
- Ventricular ectopics, ventricular tachycardia
- Transient RBBB – occurs in 5%
- Complete heart block (rare – pre-existing LBBB increases the risk)
- Thrombus formation
- Platelet aggregation begins within hours
- Infection
- Incidence rises significantly after 3 days
- Pulmonary infarction
- Associated with prolonged wedging of balloon or catheter tip
- PA rupture
- Incidence 0.02 – 0.2%, but mortality 50%
- Other
- Myocardial perforation, air embolism, catheter coiling or knotting, balloon rupture or embolism, data misinterpretation

## *Lithium Dilution Cardiac Output Measurement*

It has long been recognised that the change in pulse pressure is a function of the magnitude of stroke volume. However, translating this relationship into a way of measuring stroke volume is complicated by a number of factors:

The compliance of the aorta is not a linear relationship between pressure and volume.

The pulse pressure measured from an arterial trace is actually the sum of two waves – the incident pressure wave ejected from the heart, and the reflected wave from the periphery. In order to calculate the stroke volume these two waves must be recognised and separated.

This is further complicated by the fact that reflected waves change in size dependant on the proximity of the arterial cannula to the heart, the patient's age and degree of vascular disease.

Damping within arterial pressure measurement systems leads to imperfect waveforms and measurements – yet accurate measurements are vital since derivation of stroke volume will come from these.

The LiDCO system is a technique of pulse power analysis, which is non-morphology based (i.e. not a pulse contour method) and gets around a number of the problems discussed above. It is based on the assumption that the net power change in a heartbeat is the balance between the input of a mass of blood (the stroke volume) minus the blood mass lost to the periphery during the beat, and that after correction for compliance there is a linear relationship between net power and net flow. Using a process called 'autocorrelation', it defines the beat period and the net power change across the whole beat. Looking at the whole beat, rather than just a portion of it, renders it independent of the position of the reflected wave. Also, since autocorrelation is a time-based method that avoids the frequency approach to measuring power (such as the Fourier transforms), the effects of arterial damping (which change frequency response) are limited.

The LiDCO system uses an indicator dilution technique to measure the cardiac output and the volume of the arterial tree in the particular patient being monitored. This is used to calibrate a complex mathematical algorithm that estimates stroke volume from the arterial waveform. The arterial blood pressure trace undergoes a three-step transformation.

### ***Step 1 - arterial pressure transformation into a volume-time waveform***

An accurate way of determining the change in blood volume in the arterial tree from maximum to minimum dilatation would allow an estimate of the volume of blood flowing out of the arterial tree during a period slightly longer than diastole. Since the whole period of the cardiac cycle usually bears a fixed relation to diastole, simple scaling would give the stroke volume. The relationship between the capacity of the arterial side of the circulation and the intravascular pressure can be expressed as the compliance (i.e. pressure change per unit volume change). This relationship would be straightforward if the compliance were constant. However, arterial compliance changes as arterial pressure changes. A stiffening of the vasculature occurs as pressure and volume increase such that, at higher pressures, a given increase in pressure expands the arterial tree by a smaller volume. Nevertheless, the form of this curvilinear relationship, though differing in its scaling, appears to be very similar in different subjects.

The transformation of the arterial pressure to the 'standardised' volume-time waveform is made by the application of the equation

$$\text{Volume} = \text{CF} * 250 * (1 - \exp^{-k*P})$$

where CF is the calibration factor, 250 is the nominal volume in mls of the aorta/arterial system (nominal Vmax), P is the pressure in mmHg, and k is an exponential function that relates pressure to volume (i.e. compliance).

### ***Step 2 - deriving nominal stroke volume and the heartbeat duration***

In order to obtain cardiac output as volume per unit time, the algorithm needs to calculate the duration of the cardiac cycle and the stroke volume, or a value proportional to it (the nominal stroke volume). The mathematical technique of autocorrelation can be used to give both these values.



Nominal stroke volume: initially the software subtracts the mean value of the derived arterial blood volume record, giving a description of how much the arterial blood volume changes around it. This is periodic like a sine wave but with differently shaped areas above and below zero. Figure 1 shows how the method works by first using a pure sine wave and then subjecting it to the algorithm. Initially an estimate of the mean deviation from zero is obtained by multiplying all values of the waveform by themselves. This gives positive waves for both the positive and negative parts of the original sine wave, creating a double waveform. The mean of the values of this new waveform is the mean square and the square root of this value is a constant proportion of the amplitude of the original waveform - known as the root mean square value). This value is approximately 0.7 of the waveform amplitude and is linearly related to the stroke volume. Figure 1 shows the original sine wave and the squared (double) waveform is shown for three cycles.

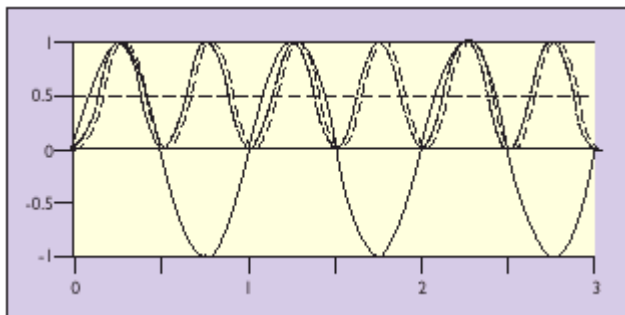


Figure 1

Estimation of the heart beat duration: having determined the nominal stroke volume, the precise period of the cycle can be obtained by moving one version of the volume waveform relative to another. Again, for autocorrelation, cross multiplication and addition of the values deliver values that are both positive and negative. The sum for a given displacement, or the tau shift, becomes less with maximum opposition of the two derived versions of the waveform and increases as the waveforms reinforce each other. Continuation of the step-by-step movement of one version of the waveform relative to the other generates an autocorrelogram with a series of maxima and minima at tau shifts, which represent the duration of the cardiac cycle.

### **Step 3 - nominal stroke volume and calibration**

The algorithm derived stroke volume and therefore cardiac output are initially uncalibrated. They are converted to actual values by multiplying the nominal stroke volume by a calibration factor. This is a patient-specific correction factor generated by the PulseCO algorithm when the nominal data are corrected to actual data by a LiDCO calibration. The  $V_{max}$  has been found to vary by up to 400% between patients, according to age, sex, size and underlying pathology. The lithium indicator calibration allows the nominal value of 250ml to be scaled up and down according to the patient's actual  $V_{max}$ .

In summary, raw haemodynamic data from the patient bedside monitor are converted to volume using the pressure-volume transform and autocorrelation. The lithium dilution cardiac output measurement is performed and the result is entered into the calibration screen to derive actual cardiac output from PulseCO.

### **Calibration by Indicator Dilution**

Lithium chloride 0.3 mmol (2ml) is used as the indicator. This is injected into a vein and its concentration in arterial blood is measured over time using a sensor attached to a peripheral arterial cannula (figure 2). The sensor is a disposable polycarbonate flow-through cell containing a lithium-selective electrode, with an eccentric inlet that causes the blood to swirl past the tip of the electrode. The hollow polyurethane electrode is filled with a reference material to maintain a constant ionic environment, and coated internally and externally with silver-silver chloride. A polyvinyl chloride membrane containing a lithium ionophore that renders it selectively permeable to lithium covers the electrode. However, despite this, the membrane still has a relatively low selectivity for lithium over sodium, and so a correction factor must be applied. The voltage across the membrane is logarithmically related to plasma lithium concentration by the Nernst equation, and is amplified and digitised before being inputted to the system computer.

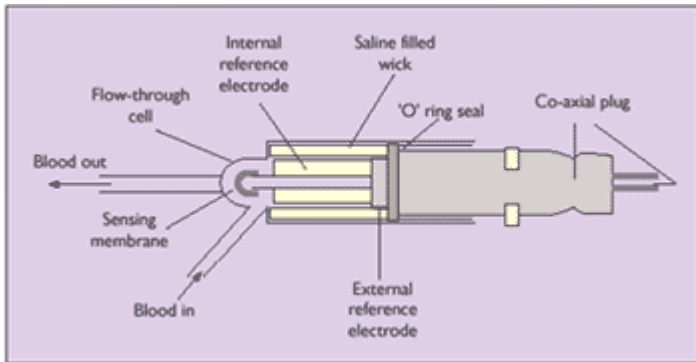


Figure 2

Lithium is an appropriate indicator substance because its plasma concentration is normally negligible, and as it does not bind to plasma or tissue proteins, there is minimal loss of indicator as it passes through the heart and lungs. Furthermore, at the doses used to make measurements, there is no significant risk of toxicity. The system must be calibrated every 8 hours, or more frequently if there appears to be significant 'drift' in the displayed values.

**The ICU technicians perform the actual set-up and calibration of the LiDCO system, and are contactable via the nurse in charge of CICU.** The sterile, disposable electrode is primed with NaCl 0.9% and is attached to the arterial manometer line via a three-way tap (Figure 3). When set up and the tap opened, blood flows into the sensor assembly at a rate that is controlled by a peristaltic, battery-powered pump to remain at 4 ml/minute.

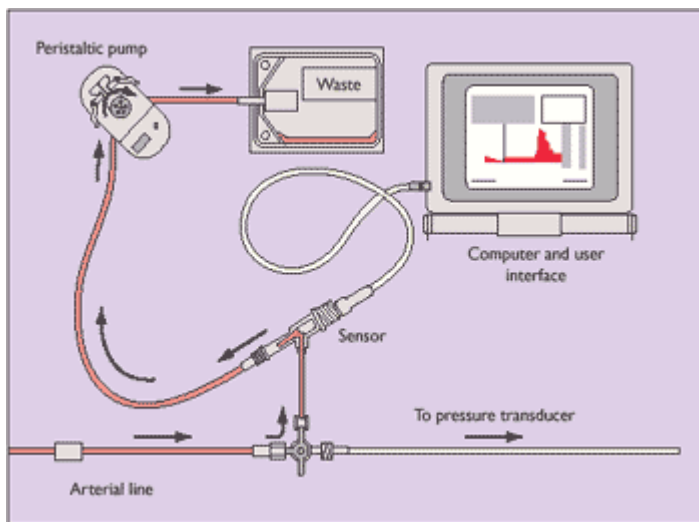


Figure 3

Isotonic lithium chloride is injected as a bolus usually via the central venous route (the peripheral route may also be used, and has been validated) and a concentration-time curve generated from the arterial sampling system. The cardiac output is calculated from the lithium dose and the area under the concentration-time curve prior to recirculation using the equation

$$\text{Cardiac output} = \text{Lithium dose (in mmol)} * 60 / \text{Area} * (1 - \text{PCV}) \text{ (in mmol per sec)}$$

where the area is the integral of the primary curve, and PCV is packed cell volume (Hb (g/ dl)/ 34). (A correction for PCV is necessary because lithium is distributed in the plasma.)

#### Limitations of LiDCO

Because the concentration change of lithium is used to calculate the cardiac output, this technique cannot be used in patients receiving lithium therapy, since the increased background lithium concentration causes an overestimation of cardiac output. The electrode may also drift in the presence of certain muscle relaxant infusions, since these are large polar molecules that interfere with the electrode. If muscle paralysis is used,

bolus techniques of administration have to be adopted, and calibration performed before the bolus of muscle relaxant is administered.

Intra-cardiac shunts, aortic valve regurgitation, intra-aortic balloon pumps, severe peripheral arterial vasoconstriction and highly damped peripheral arterial lines will all increase the likelihood of inaccurate results using the LiDCO system, as will any rhythm disturbance that produces significant beat-to-beat changes in stroke volume that are not a consequence of simple heart-lung interactions.

The key consideration of any cardiac output monitoring system is that the data produced should be used to confirm the clinical assessment made of the patient.

# CICU Investigations

Authors: Dr A Richardson & Dr K Wilkinson

Revised: May 2015

The key principles underpinning the ordering of any investigation are:

- It must seek to answer a specific clinical question
- Once performed, it must be reviewed in a timely fashion and where appropriate, acted on
- The result of any investigation must be documented in the clinical notes

## *Chest Radiography*

Chest radiographs are ordered by calling the X-ray department and completing the request on the computerised eQuest system. The patient details and history and specific questions to be answered must be completed in the appropriate areas

Radiographs are reviewed using the GE PACS system on the computers, and several large screen monitors are available specifically for this purpose.

If you have difficulty interpreting a particular chest film, then the CICU duty consultant or one of the cardiothoracic radiologists (found on E level) should be asked for their opinion. If you discuss cases with the cardiothoracic radiologists, be prepared to give them a concise but detailed history of the patient, and you should know what previous imaging the patient has had and which outside hospital they have come from.

Indications for ordering chest radiographs are usually obvious, but would include checking the position of nasogastric tubes/ lines/drains/balloon pumps, etc, looking for pleural or mediastinal collections in patients who may be bleeding, and looking for causes of respiratory embarrassment. Whilst we do not perform 'routine' chest radiographs, it is standard practice to obtain a chest film after central line insertion performed (or attempted but unsuccessful) on the unit, and after the removal of chest drains (to exclude pneumothoraces). Central lines that are placed in theatre under consultant supervision, transduced with an appropriate waveform, & used throughout the case do not require an X-ray. Please document the results of the chest xray in the notes.

## *Pleural Ultrasound*

If the chest radiograph appears to show a pleural collection, then the advice of the cardiothoracic radiologists should be sought as to whether pleural ultrasound is required. The cardiothoracic radiologists are Dr. Ivan Brown, Dr. Charles Peebles, Dr James Shambrook and Dr. Stephen Harden, and they may usually be found on E level in the Cardiothoracic Radiology department. Once they have reviewed the chest film and agreed that an ultrasound is necessary, they will usually arrange for the investigation to take place. You will have to submit a request form. The radiologists may insert a pig-tailed catheter whilst performing the ultrasound or mark an appropriate drainage point. You will then have to request that the cardiothoracic surgical registrar (bleep 9211) on call inserts the drain.

## *CT Scans*

The decision to take a patient to the CT scanner is a balance of risks and benefits, since transporting critically ill patients around the hospital is potentially hazardous. CT scans are therefore only arranged after discussion with the duty CICU consultant.

Head CT scans will usually be ordered for patients who fail to show a return to a suitable level of consciousness despite an appropriate time off sedation, for those who display abnormal neurology, or for those who exhibit acute agitation preventing the weaning of sedation despite having been given time for CNS recovery to occur.

Thoracic CT scans may be ordered to look for evidence of deep-seated sub-sternal infection in patients with unresolving sepsis, or to investigate patients with severe lung disease preventing weaning from mechanical ventilation.

Abdominal CT scans are usually reserved for patients who develop signs of intra-abdominal catastrophe (perforated viscus or mesenteric ischaemia).

Logistically, all but the simplest and fittest of patients leaving the unit for a scan should be accompanied by an experienced doctor with anaesthetic skills, an experienced ICU nurse and an ICU technician. Where a patient has an intra-aortic balloon pump in situ, a perfusionist must also attend. Patients who do not require much equipment for the transfer may be transferred with the transfer bag and equipment attached to the bed. For those requiring more support we have a transfer frame that fits onto the end of the bed and provides points of attachment for pumps, oxygen & ventilator, as well as a single power supply to all equipment.

There are a number of Oxylog ventilators available to ventilate the patient for transfer. **Capnography should be used for all ventilated patients.**



Oxylog 1000



Oxylog 2000



Oxylog 3000

### *Echocardiography*

An echo is frequently very useful in managing patients' post-cardiac surgery. Indications include:

- Haemodynamic instability post-surgery to look for evidence of...
  - cardiac tamponade or valvar dysfunction or regional ventricular dysfunction suggesting graft failure, or global LV dysfunction
- New murmurs
- Failure to wean from mechanical ventilation due to pulmonary oedema

During working hours, echocardiograms are booked through the echo department (extension 3145, 6404 or bleeps 2860 or 2997) and performed by senior echo technicians. A request should be entered on the eQuest computer system. If urgent, it should be requested in person. The results of these scans are put onto the eQuest computer program. Out of hours or very urgent scans in unstable patients are requested through the cardiology registrar on call (bleep 2390). If you are requesting an urgent or out-of-hours echo, be prepared to explain concisely exactly why you want it and what questions you want answered. You should make every effort to be present when the echo is being performed, as it is an excellent learning opportunity for you, and you will gain more understanding of your patient's cardiac status than from simply reading the report.

Transthoracic echo is useful for assessing valve lesions and LV function. However, post-cardiac surgery, the dressings, drains, air in the thorax and tissue oedema all frequently combine to produce poor quality images. Where this is the case, serious consideration should be given to performing a transoesophageal echo (TOE). Please see the section on Focused TOE in Cardiac ITU for details of TOE in CITU.

It is also worth noting that cardiac tamponade in the post-cardiac surgical setting does not usually have the same echo appearance as the textbook-described medical pericardial effusion. Inexperienced operators may need to be reminded that focal tamponade must be excluded after cardiac surgery, and TOE is often needed for this. Even TOE may fail to demonstrate tamponade, however, and the final decision as to whether the patient needs urgent mediastinal re-exploration is a clinical one.

# Line Insertion and Documentation

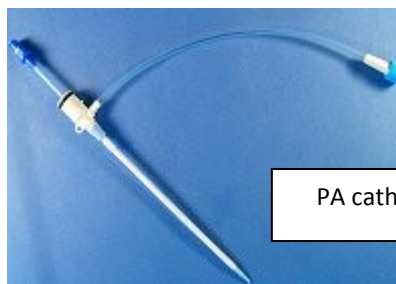
Authors: Dr Andrew Richardson & Dr Kirstin Wilkinson

Revised: May 2015

Central venous catheterisation is frequently performed on CICU. We routinely use a 5-lumen central venous catheter (CVC) for the administration of vasoactive drugs and multiple infusions in the critically ill. In addition to this, central venous cannulation may be required for PA catheter insertion or to facilitate renal replacement therapy.



CVVH catheter



PA catheter introducer

However, like all medical interventions, there are a number of complications associated with CVC placement, chiefly:

- Arterial puncture, arteriovenous fistula, pneumothorax, nerve injury
- Multiple unsuccessful attempts at catheterisation, which delay treatment

The risks and the consequences of complications vary across different patient groups depending on the patient's anatomy (e.g. morbid obesity, short neck, or local scarring from surgery or radiation treatment), the circumstances in which CVC insertion is carried out (e.g. mechanically ventilated patients & the risk of tension pneumothorax, or during emergencies such as cardiac arrest) and pre-existing co-morbidities (e.g. bullous emphysema or coagulopathy).

## *CICU Guidelines for CVC Insertion:*

1. As per NICE guidelines, all trainees should use real-time 2D ultrasound guidance for line insertion. Where there is any doubt regarding venous versus arterial cannulation (with or without the use of ultrasound), the needle or cannula should be transduced BEFORE the dilator is passed.

The subclavian route is NOT to be attempted in patients with INR > 1.3, APTR > 1.3 or platelet count < 50 without explicit prior discussion with the consultant on call. Other contra-indications would include:

- Pneumothorax or haemothorax on the contralateral side
- Inability to tolerate pneumothorax on the ipsilateral side
- Morbid obesity
- Recently discontinued subclavian catheter at the same location
- Patients receiving ventilatory support with high end expiratory pressures (temporarily reduce the pressures)

Try to avoid the subclavian route for the placement of haemodialysis catheters, as the line frequently gets pinched between the clavicle and 1<sup>st</sup> rib as the patient moves, leading to poor flow through the haemofilter. However, the left subclavian approach has a sweeping curve to the apex of the right ventricle and is one of the preferred approaches for PA catheter insertion. For general CVC insertion, if via the subclavian route, the right sided approach is generally preferred because the dome of the pleura of the right lung is usually lower than the left, and the left-sided large thoracic duct is less likely to be lacerated

Pre-measurement of catheter length against the patient's chest allows an estimation of the catheter length that will place the catheter tip about 2 to 3 cm below the manubriosternal junction (in the superior vena cava, just above the right atrium).

2. The central line packs will be used: these contain all equipment necessary except:

- skin preparation, sterile gloves, gel and sterile sheath for the ultrasound probe and flush solution

Chlorhexidine-coated lines should be used in all cases except where there is documented chlorhexidine allergy. Sterile gown and gloves are mandatory for all line insertions except for those performed under genuine emergency conditions. Where the emergent nature of the insertion precludes proper aseptic technique, the line should be changed for a 'clean' line as soon as is practical.

3. Skin may be prepared with chlorhexidine (e.g. 'Chloraprep' sticks) or povidone-iodine solutions. However, 2% chlorhexidine in isopropanol (Chloraprep) has greater rapid bactericidal activity & ongoing residual effect than povidone-iodine, and is therefore preferred unless there is chlorhexidine allergy.

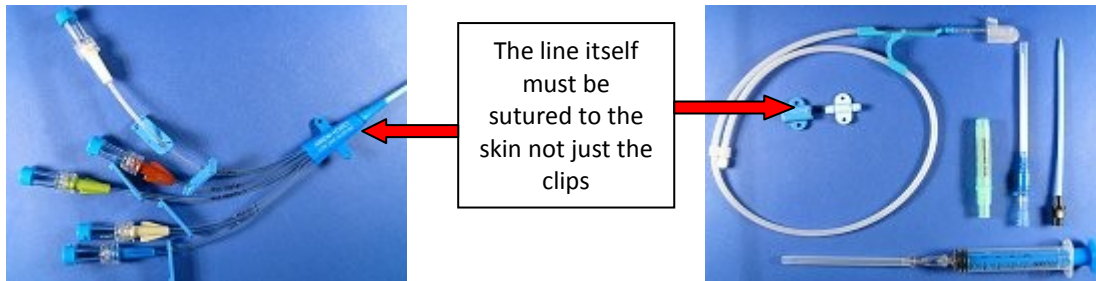
4. Awake patients must have the procedure, its indications and its potential complications explained to them, although formal written consent need not be obtained.

5. Awake or very lightly sedated patients should have local anaesthetic infiltration prior to starting the insertion procedure.

6. ECG monitoring must be used throughout the procedure. Lines should be pulled back until the P wave morphology has returned to baseline.

7. Four-point fixation must be used: it is not good enough to simply fix the attaching clip to the skin, as the line may still become displaced.

8. All lines must be transduced to confirm a venous waveform prior to any use



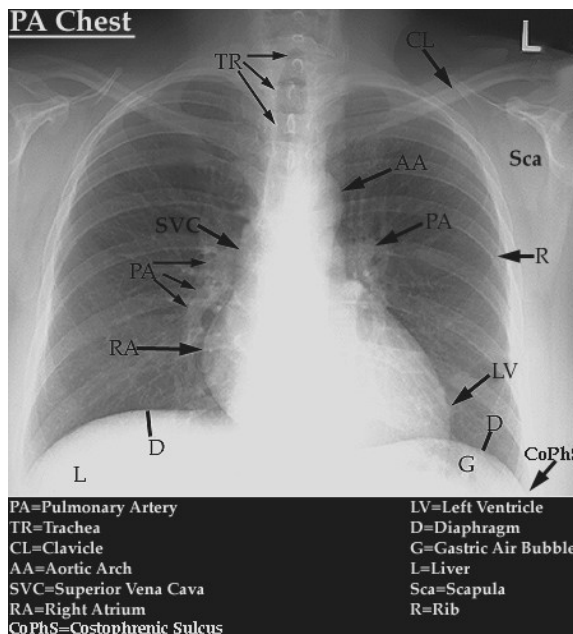
8. A sterile transparent dressing should be applied that allows visualisation of the insertion site for signs of infection.

9. A chest radiograph should be ordered for all attempted central line insertions, primarily to exclude complications such as haemo- or pneumothorax, but also to check appropriate line positioning (the tip should lie just above the carina).



In a study of 112 pulmonary CT angiograms, the mean distance ( $\pm$  standard deviation) from the carina to the cavo-atrial junction 40.3 mm  $\pm$  13.6.

Thus, placement of the central venous catheter tip at or just below the level of the carina during inspiration ensures placement in the SVC. Placement of the tip approximately 4 cm below the carina will result in placement near the cavo-atrial junction.



### Complications, Prevention and Management:

- Pneumothorax
  - Prevention: avoid subclavian approach where possible, limit depth of needle insertion, choose the right side rather than left where subclavian approach needed, avoid multiple attempts
  - Management: Check post-procedure x-ray, if pneumothorax arrange for thoracostomy depending on the size of the pneumothorax
- Haemothorax - as above
- Intra-arterial placement
  - Always exclude by transducing the line prior to any use (the administration of inotropes through a line in the carotid artery is a catastrophe)
- Bilateral Iatrogenic complications
  - Prevention: If attempted catheterization is unsuccessful, try the ipsilateral internal jugular or subclavicular approach before trying contralateral subclavian catheterization
- Catheter embolisation
  - Prevention: **Never** withdraw a catheter past a needle bevel which might shear off the catheter
  - Management: x-ray the patient and contact specialist who can remove the embolised catheter
- Infection
  - Prevention: Never choose an insertion site that goes through infected tissue; use antimicrobial-impregnated catheters; avoid the use of antibiotic ointments (increase of fungal contamination and antibiotic resistant bacteria)
- Cardiac dysrhythmia
  - Prevention: have someone watch monitor for dysrhythmia while the catheter is advanced (this comes from direct contact of the catheter tip with the myocardium of the right atrium)
  - Management: withdraw the guide-wire, reposition the catheter; treat dysrhythmia according to ALS protocols.
- Air embolism
  - Prevention: Maintain a Trendelenberg position, ask the patient to exhale while you are advancing the catheter, maintain a "closed system"
  - Management: Place the patient in a left lateral decubitus, head down position to minimize the chances of an air embolism to the brain.



### *Documentation in the Medical Record*

The following are considered a minimum data-set for documenting line insertions or attempted insertions:

- Date & time
- Name of person performing procedure
- Indications for the procedure
- The procedure including prep, anesthesia, approach, technique & use of ultrasound
- Any complications or "none"
- Who was notified about any complication (family, consultant etc.)
- The result of the post-insertion x-ray must be inserted in the notes by the operator

Please attach the pre-printed sticker that documents the above in the patient's notes. There should be a sticker in all the central venous line packs.

# Percutaneous (temporary) Tracheostomy on CICU

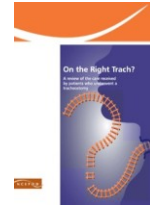
Author: Dr Nick Goddard

Revised: May 2015

## Introduction

NCEPOD 2014:

Percutaneous tracheostomy has evolved to become a routine bedside procedure within modern ICU environments. In England alone, about 15,000 percutaneous tracheostomies are managed (and probably inserted) each year in critical care [1]. However, NPSA safety reports, [2] NAP4, [3] and NCEPOD [4] have recognised significant risks associated with tracheostomy. When they occur, incidents are compounded by staff unfamiliarity, inadequate training and lack of availability of equipment. Therefore, the procedure should always be undertaken by (or supervised by) senior practitioners competent in the procedure, and staff should be well-trained in routine maintenance as well as emergency management of these devices.



## Indications

Percutaneous (elective) tracheostomy has the following advantages over trans-laryngeal intubation:

- Weaning may take place in the absence of sedative drugs, upper airway anatomical dead space is reduced (up to 50%) and earlier mobilisation

Most tracheostomies inserted in CICU are temporary in nature and will be removed after weaning from the ventilator. It is generally considered unsafe to perform tracheostomy in patients with  $FiO_2 > 0.5$  and  $PEEP > 7$ . However, beyond this timing is controversial. The potential benefits of tracheostomy, coupled with the drive to de-escalate intensity of care as soon as is feasible must be balanced against needless insertions with associated morbidity and mortality. Ultimately, this will be an individual decision for each patient.

The TracMan study [5] included 909 general intensive care patients, comparing early (day 1-4) vs late (> or equal to day 10) tracheostomy. Mortality was similar in both groups up to 2 years post-randomisation. The applicability of this to the CICU cohort of patients remains to be seen. Moreover, in NCEPOD (2014), 18% (161/910) of patients underwent decannulation in < 7 days in the critical care unit. 85/141 patients who had an early decannulation did not undergo a trial of extubation before tracheostomy insertion. 68 of these were percutaneous insertions.

## Procedure

### Preparation

The patient is usually unable to consent to the procedure due to their being mechanically ventilated and under the influence of sedative drugs. Although the patient's next of kin are not able to give legal consent, the procedure should be discussed, and a 'Form 4' completed and signed. You should specifically mention the risks of life-threatening haemorrhage and airway disruption, both of which can occur early (at the time of insertion) and later on after several weeks. Clearly the patient must not have deranged coagulation ( $INR > 1.3$ ,  $APTR > 1.3$ , platelet count < 100) and there should be adequate anaesthesia in place (e.g. combined propofol and opioid infusions) and full monitoring in situ (especially capnography).

## Equipment

The default insertion kit on CICU is the TRACOE® 'experc-Set' containing the TRACOE® *twist* tracheostomy tube type. The set consists of a Seldinger needle and wire, over which a guide (plastic sheath) and then a dilator passed. Each size of tracheostomy tube has a corresponding dilator size, and comes complete in its own insertion pack. Each tracheostomy tube comes preloaded with a stylet and an 'atraumatic inserter' (right). This smooths over the gap in diameter between the end of the tube and the inner stylet. Do NOT remove this or you will not get it back on!



*Atraumatic Insertion System:*

In addition to the equipment given within the set, you will also need:

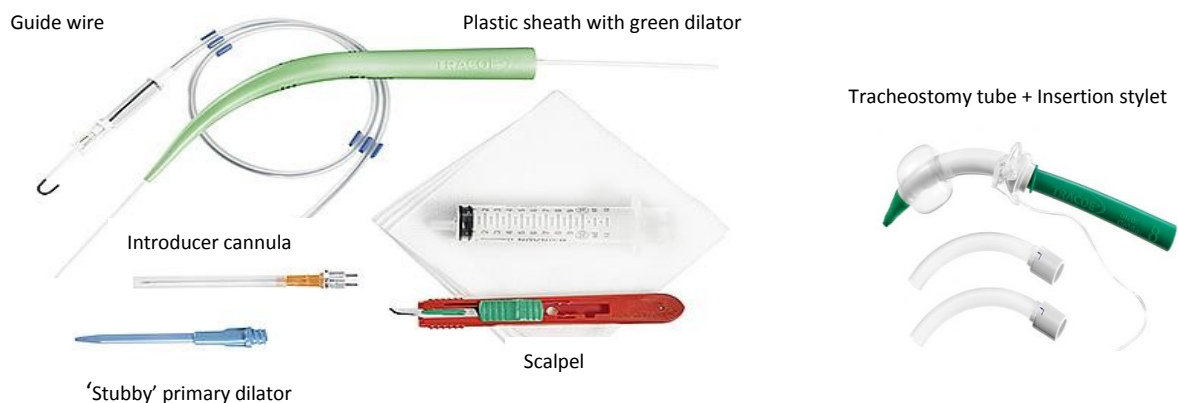
- Sterile field and 2% chlorhexidine (skin preparation)
- Lubricating jelly (for tracheostomy tube) and sterile water (for lubricating the dilator)
- Local anaesthetic with adrenaline
- Fibreoptic bronchoscope and catheter mount to accept scope

## Landmarks

The patient is positioned with the neck extended, using an intravenous fluid bag between the shoulder blades. This brings as much of the trachea as possible into the neck. The larynx and cricoid cartilage with intervening cricothyroid membrane are identified. From the cricoid, moving caudally, the tracheal rings are identified (this may also be achieved using ultrasound). Placing the airway any higher next to the cricoid can cause tracheal erosion and long-term problems. The patient is then prepped and draped, equipment is laid out and checked, then the bronchoscope is passed (usually by a technician) through a tracheal tube and the anatomy of the airway visualised. The aim of the fibreoptic scope is to ensure correct initial placement of the introducer needle, in the midline and between (not through) the second or third tracheal rings. If the tracheostomy is made in the sidewall of the trachea, life-threatening airway disruption may result, that is extremely difficult to remedy. Subsequent to this, the bronchoscope functions to monitor dilation of the trachea.

While visualising the airway, local anaesthetic with adrenaline can be infiltrated subcutaneously. Soak the one-stage dilator in a bowl of sterile water. It has a special hydrophilic coating which becomes slippery when rendered wet for any length of time so is auto-lubricated when the time comes to dilate the trachea. The tracheostomy within the pack should be tested and well lubricated.

## **TRACOE® 'experc percutan' Set (twist)**



## Insertion Technique

This is a modification of the original Ciaglia technique [6] and not strictly a *single* dilation as there is a 'stubby' primary dilator which must initially be passed and removed:

1. The tracheal tube is withdrawn (under direct/bronchoscopic vision) into the larynx
2. Keeping in the midline, the introducer cannula and syringe are advanced, at 45 degrees to the skin, until air is aspirated
3. The guide wire is passed through the cannula
4. The small 'stubby' primary dilator is passed and removed
5. A white plastic sheath is positioned over the wire (to act as a guide for the main dilator)
6. The one-stage dilator is loaded onto the guide wire AND sheath up to a safety ridge (preventing kinking of the wire and damage to the curved dilator tip)
7. The dilator is then passed into the trachea until the black line is reached (moderate force required)
8. The tracheostomy tube and insertion stylet are passed over the guide wire and guide catheter (sheath) into the trachea
9. Guide wire, sheath and stylet are removed together
10. Inner tube with 15mm connection is attached (requires a 'twist') and linked to ventilator

Full documentation of the procedure should then take place within the clinical record.

### Tips

- If the dilator does not pass easily, examine the initial incision. Often, it is the skin that impedes progress, and the incision has to be slightly widened with the scalpel. Keep in mind that the minimal incision necessary (sometimes no incision) for a tight fitting tube will better avoid infection.
- The use of the tracheal dilator is almost never necessary, and may be hazardous. However, if the introducer is inadvertently pulled out of the trachea, or some other mishap occurs, it may be useful in relocating the tract for replacement.
- The atraumatic insertion system can get a little confusing to understand (see right). Once the tracheostomy is in situ, the stylet is removed and the silicon atraumatic inserter rolls back.
- The bronchoscope can be quickly used to confirm position and to exclude bleeding prior to ventilation of the tracheostomy. Endobronchial suction may also be helpful prior to positive pressure ventilation



## General management of tracheostomies

### Introduction

At this time (2015), UHS Trust-wide policy on tracheostomy care continues to follow (adapted) guidelines produced by St.George's hospital in 2011 [7]. These guidelines are not written specifically with intensive care practice in mind, although the majority of insertions probably take place in this environment. Since the time of writing, important influences on care of the tracheostomy patients have come from NAP4 (2011) as well as 'On the Right Trach' from NCEPOD (2014). [3, 4] In the UK, in response to a need for improvements and standardisation in training and practice the national tracheostomy safety project (NTSP) was launched with a comprehensive manual produced in 2013. [8] This manual has been endorsed by ALL key national stakeholders, and the NTSP also forms part of the Global Tracheostomy Collaborative, with the aim of delivering better tracheostomy care everywhere. The practices and protocols advocated are very similar to St.George's guidelines (2011) and complementary to other nationally available guidelines (e.g. Intensive Care Society [9]). The NTSP manual is the current standard of care, and even St.George's University Hospitals NHS Trust guidelines now link to these documents. [10]

## Tracheostomy Emergencies

The NPSA have previously described 53 tracheostomy-related incidents from UK critical cares during the period 2005-7. [2] Fourteen of these were classed as major or life threatening and 8 required interventions to maintain life or may have contributed to death. NAP4 (2011) reported 14 tracheostomy related problems on ICU's, with 7 deaths and 4 hypoxic brain injuries over a 1 year period. NCEPOD (2014) reported 23.6% (461/1956) of patients in a critical care unit had complications from tracheostomy over an 11 week period, with accidental tube displacement occurring in 4.1%. The issues most frequently occurring are:

- Accidental tube displacement, obstruction and haemorrhage

In each scenario, the severity of outcome is frequently compounded by errors in judgement, lack of training, and/or access to equipment. As a result, emergency algorithms have been produced to address these issues. [11, 12] A copy of (a part of) this algorithm is included on the next page. This algorithm applies to the patient with a patent upper airway (i.e. a non-laryngectomy patient with no upper airway abnormalities).

## Tracheostomy Equipment

All patients with a tracheostomy in situ should have immediate access to individualised airway rescue equipment. In other ICU areas this may be stored in a small blue box. On CICU, this equipment is placed inside the airway trays which are present in each bed space. This equipment should also follow the patient when a transfer may be required.

Essential Tracheostomy equipment (to be kept in bedside airway trays) includes:

- Scissors + stitch cutter
- Lubricating jelly
- 10 mL syringe
- Spare tracheostomy tubes (same size + one smaller plus spare inner + suction tubes)
- Tracheostomy dressing + tapes
- Tracheal dilators
- Size 6.0 ETT (uncut +/- armoured)
- Spare oxygen 'nipple' (to provide simultaneous O2 via face and tracheostomy)
- Tracheostomy disconnection wedge (arguably not essential)



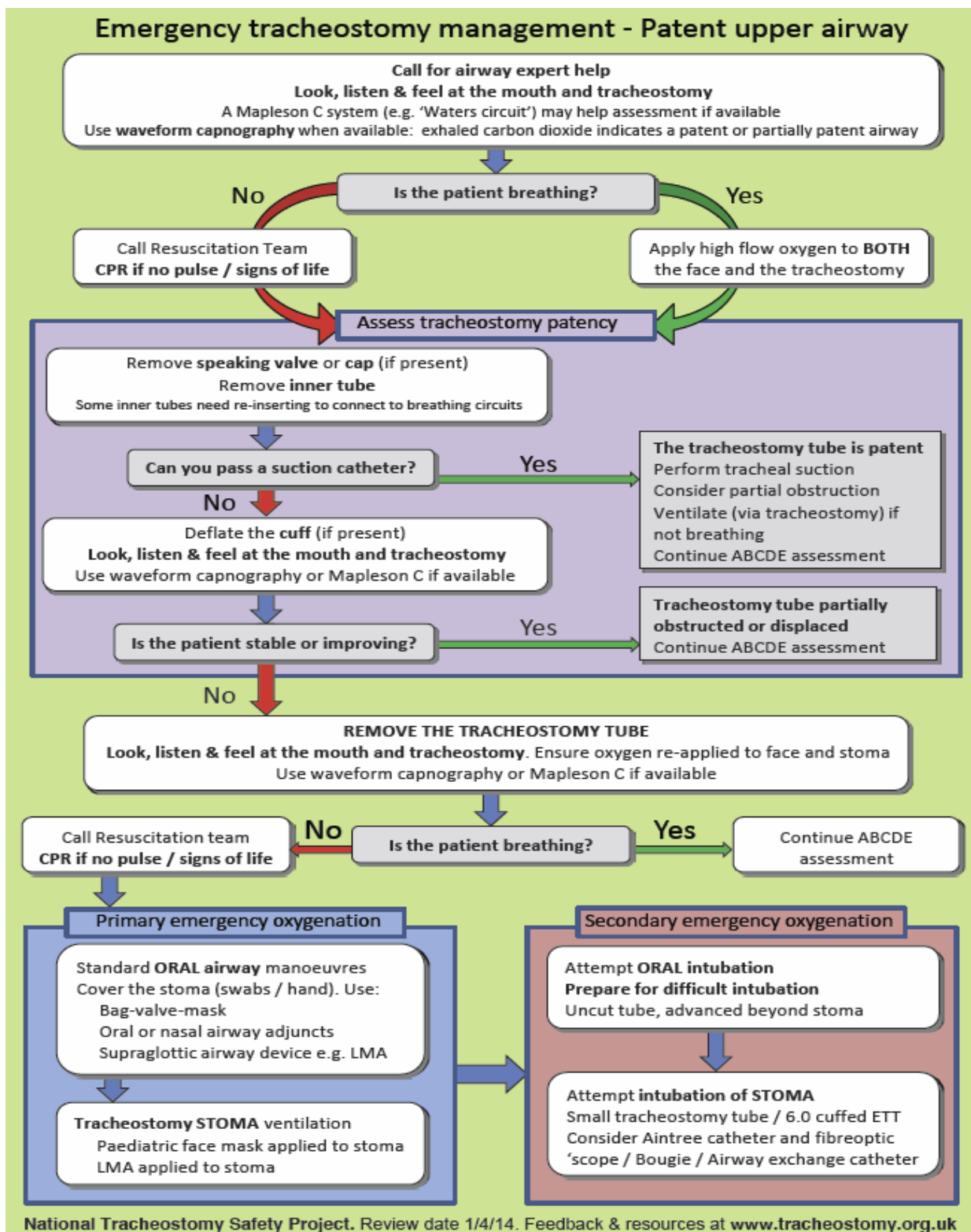
*CICU bedside airway trays:*

This equipment will be checked by nursing staff at the beginning of each shift. In addition CICU has a standardised difficult airway trolley, as well as access to fiberoptic bronchoscopy via the technicians' office (up to 0100). Out of hours, bronchoscopes can be accessed from the cupboard in the corridor between GICU A side and B side. A light source can be found either on the GICU difficult airway trolley, from the technicians, or from theatres. Bedhead signs for ready identification of information relating to each patient's tracheostomy should clearly display the tube type, size, insertion method, date of insertion and difficulties associated with the upper airway.

### Routine Tracheostomy Change:

|                                      |   |
|--------------------------------------|---|
| Single lumen perc:                   | > 10-14 days (then every 7-14 days)                                 |
| Double lumen perc (= TRACOE® twist): | Up to 30 days (= maximum by manufacturer)                           |
| Surgical Tracheostomy                | > 4 days = safe (then according to whether single vs. double lumen) |

A tracheostomy tract is likely to close quickly in the first 48 hours after surgical tracheostomy and within 7-10 days following percutaneous. A tube change during this time may be tricky or impossible. The basic equipment required to perform a tube change is essentially the same as that required for emergency management and is kept in the bedside airway trays with the additional possibility that an exchange device (bougie or Aintree catheter) may be required and fiberoptic bronchoscopy should be immediately available. Monitoring, positioning, preparation and anaesthesia are the same as for primary insertion.



## Speaking Valves

The two types of speaking valves used within this trust are Passe-Muir® and Rusch®

Prior to using speaking valves a cuff deflation trial should be applied (5 min initially). A gradual tolerance in the duration of speaking valve trial should be attempted, monitoring for distress closely i.e. RR, SpO<sub>2</sub>, accessory muscle use, increased work of breathing.

| Passe-Muir:   | Rusch Valve:   |
|---|--|
| <p><i>'Closed system' – i.e. closed at all times except on inspiration</i></p> <ul style="list-style-type: none"> <li>-Ventilator application</li> <li>-Communication</li> <li>-Improves swallowing</li> <li>-May reduce aspiration</li> <li>-Improves olfaction</li> <li>-Improves oxygenation</li> <li>-Facilitates secretion management</li> <li>-Expedites weaning and decannulation</li> <li>-Restores positive airway pressure</li> <li>-Facilitates infection control</li> <li>-Psychological</li> </ul> | <ul style="list-style-type: none"> <li>-Cheaper alternative</li> <li>-High risk of incident reporting</li> <li>-Will need absolute close monitoring as the humidity and secretions can cause the valve to block off</li> </ul> |



= 22mm standard connection to ventilator circuit

*Passe-Muir® Valve = semi-transparent one way valve membrane, which remains closed until inspiration is initiated.*

### Weaning and decannulation principles

The term 'weaning' in the ICU environment usually means a gradual reduction in support from mechanical ventilation/ assist ventilation. The speed of weaning will be individual to circumstances. There are no definite criteria that accurately predict a patient's readiness for decannulation, but success is likely if the patient is neurologically intact (with adequate sleep and nutrition), has a 'stable' lung status (i.e. the primary cause for ventilation has resolved, FiO<sub>2</sub> < 40%, and secretions not excessive) and able to clear their own airway. Removal of the tracheostomy tube will cause an increase in the anatomical dead space which may result in an increase in the patient's work of breathing - some ventilatory reserve is needed for this step. Ways of assessing readiness for decannulation include:

- Spontaneously breathing off the ventilator for 24-48 hours continuously and the primary cause for ventilation has resolved (e.g. bronchopulmonary infection)
- With cuff deflation patient is able to **cough and clear** effectively
- Speaking valve initially 15 min with 30 min rest progressing to **4 hours** or more, although not to be worn whilst sleeping
- Decannulation cap\* (capping off) - the cuff must always be deflated. This practice usually follows downsizing to smaller tracheostomy tubes and is uncommon within the CICU environment (**\*Requires MDT and senior medical involvement**).

### Tracheostomy tube types

Patient anatomy varies; therefore variable length tubes are available to accommodate patients with thick necks (e.g. obese patients). Tubes may be PVC (polyvinylchloride), or silicone (+ reinforced) and will differ accordingly to the degree of flexibility they provide. Almost all tubes should be non-fenestrated, as fenestrated tubes are associated with ventilator leak, damage to the tracheal wall on suctioning (resulting in granulation tissue, obstruction, fistula +/- infection), and surgical emphysema. They tend only to be used for difficult/



prolonged weans. A cuffed tube allows positive pressure ventilation and protects against aspiration. Cuff pressure should be maintained at 15 – 25cm H<sub>2</sub>O to avoid tracheal necrosis, arterial erosion and stenosis.

### Discharge

Most patients from CICU with tracheostomies in situ will be discharged to regional critical care units, but occasionally to rehabilitation facilities or wards. Transfers should take place in day light hours, and standards of paperwork should include a detailed summary of care within the CICU discharge letter.

### References

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# Intra-Aortic Balloon Pump (IABP)

Authors: Dr Tom Pierce & Dr Kirstin Wilkinson

Revised: June 2015

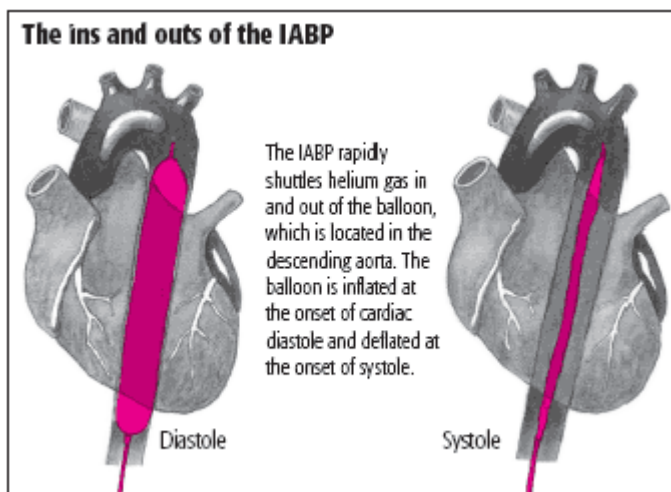
Sometimes referred to as intra-aortic balloon counter-pulsation, intra-aortic balloon pumps aim to help increase myocardial oxygen supply and reduce oxygen demand. They may be placed in patients who have resistant ischaemia before surgery or can be inserted intra or postoperatively. You will gain most experience from the post-operative patient in the CICU. The perfusionists are on-call for any technical problems, the surgeons for insertion and positioning problems. A perfusionist should always be present when patients with an IABP in situ are moved.

## Positioning

Via the femoral artery with its x-ray marker tip just below the aortic arch. Volumes 40 ml for males and 32 for females. Driven by helium. Contain a pressure channel for aortic pressure measurement.

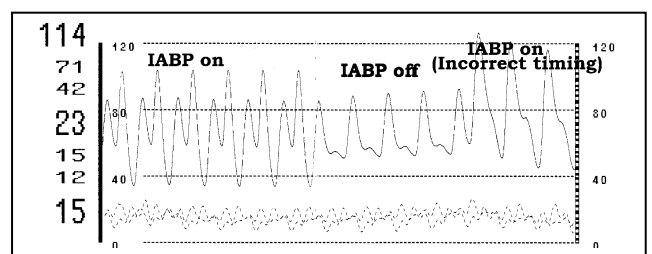
## Timing

Balloon inflation and deflation are generally timed with the R-wave and may be advanced or delayed. Sometimes timed on the pressure wave. Inflation on the dicrotic notch. Deflation immediately prior to patient's systole. Optimal timing as illustrated by good augmentation is shown in the first part of the accompanying trace.



## Effects of an intra-aortic balloon pump

- Balloon diastolic augmentation pressure  $\uparrow$
- Assisted aortic end diastolic pressure  $\downarrow$
- Patient's own systolic BP  $\downarrow$
- Mean arterial pressure  $\uparrow$
- Coronary perfusion pressure  $\uparrow \uparrow$
- Little effect on stroke volume (approx 400ml to CO)



## Complications of IABP

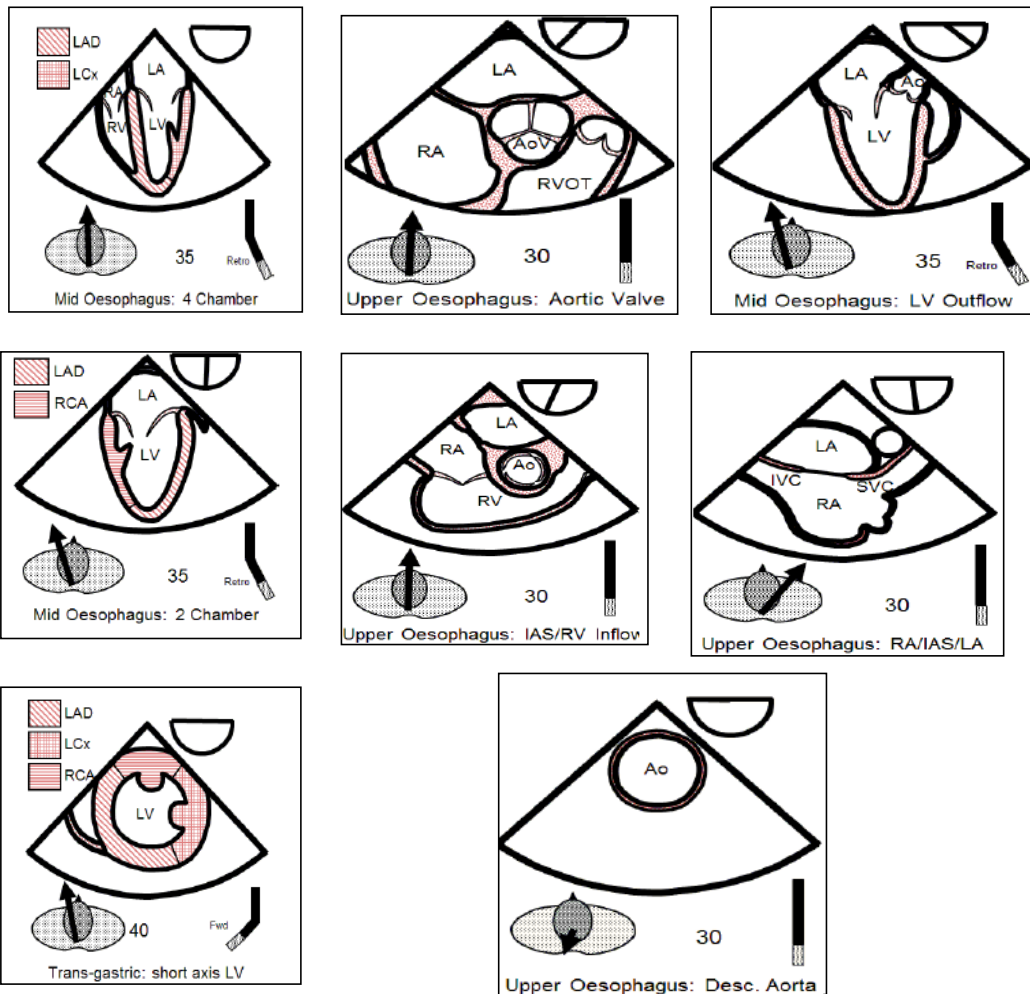
- Of the balloon: Rupture (rare), dislodgement of aortic atheroma, inflation over the renal arteries
- Of the vessel puncture: sepsis, dissection of the femoral artery, leg ischaemia (examine distal pulses)
- To cellular elements: thrombocytopenia
- Technical problems: Gas loss via a leak, difficulty tracking tachycardia and atrial fibrillation, incorrect timing, pacing spike artefacts may affect timing (select this option)

# Southampton CICU Focused TOE Examination

Authors: Dr Paul Diprose & Dr Kirstin Wilkinson

Revised: May 2015

This TOE examination protocol can be used to identify serious pathology in the CICU in the cardiovascularly unstable patient. There are 8 standard views as demonstrated below. It should only be carried out by those competent in the performance and interpretation of TOE or under appropriate supervision. The aim is to identify cases of hypovolaemia, LV or RV systolic failure, signs of potential massive PE, cardiac tamponade, severe AS or aortic dissection. Other pathologies including pleural collections or new regional wall motion abnormalities may also point to causes of cardiovascular instability.



Version 1.1 May 2015 PD/KW – Based on St George's FIT scan by Fletcher/Edsell/Leech

Once the examination is completed, the findings must be documented either on the CIS or using the form reproduced overleaf.

# Focused TOE Exam Findings

|   |   |   |   |
|---|---|---|---|
| Date: _____ Time: _____<br>Echocardiographer 1: _____<br>Echocardiographer 2: _____<br>Ease of insertion: _____ Machine no.: _____  | <p style="font-size: 1.2em;">Please affix patient label</p>                     |   |   |
| <ul style="list-style-type: none"> <li>• <b>Left Ventricle</b> <ul style="list-style-type: none"> <li>– Size               <ul style="list-style-type: none"> <li>• Normal / Dilated</li> <li>• LVIDD (    mm)</li> </ul> </li> <li>– Systolic function               <ul style="list-style-type: none"> <li>• Good / Moderate / Poor</li> </ul> </li> <li>– RWMA (territories affected)               <ul style="list-style-type: none"> <li>• LAD / RCA / Circumflex</li> </ul> </li> </ul> </li> <li>• <b>Right Ventricle</b> <ul style="list-style-type: none"> <li>– Size               <ul style="list-style-type: none"> <li>• Normal / Dilated</li> </ul> </li> <li>– Systolic function               <ul style="list-style-type: none"> <li>• Normal / Impaired</li> <li>• TAPSE &gt;20mm: Yes / No</li> </ul> </li> </ul> </li> </ul> | <b style="writing-mode: vertical-rl; transform: rotate(180deg);">Ventricles</b> | <ul style="list-style-type: none"> <li>• <b>Left atrium</b> <ul style="list-style-type: none"> <li>– Normal / Dilated</li> <li>– Thrombus in LAA: Yes / No</li> </ul> </li> <li>• <b>Inter-atrial septum</b> <ul style="list-style-type: none"> <li>– ASD/PFO present: Yes / No</li> <li>– Pressure overload               <ul style="list-style-type: none"> <li>• None / Rt high / Lt high</li> </ul> </li> </ul> </li> </ul> | <b style="writing-mode: vertical-rl; transform: rotate(180deg);">Atrium</b>             |
| <ul style="list-style-type: none"> <li>• <b>Mitral valve</b> <ul style="list-style-type: none"> <li>– Regurgitation               <ul style="list-style-type: none"> <li>• None / Trace / Mild / Moderate / Severe</li> </ul> </li> </ul> </li> <li>• <b>Tricuspid valve</b> <ul style="list-style-type: none"> <li>– Regurgitation               <ul style="list-style-type: none"> <li>• None / Trace / Mild / Moderate / Severe</li> </ul> </li> </ul> </li> <li>• <b>Aortic valve</b> <ul style="list-style-type: none"> <li>– Structure               <ul style="list-style-type: none"> <li>• Normal / Abnormal</li> </ul> </li> <li>– Regurgitation               <ul style="list-style-type: none"> <li>• None / Trace / Mild / Moderate / Severe</li> </ul> </li> </ul> </li> </ul>  | <b style="writing-mode: vertical-rl; transform: rotate(180deg);">Valves</b>     | <ul style="list-style-type: none"> <li>• <b>Pericardium</b> <ul style="list-style-type: none"> <li>– Effusion: Present / Absent</li> </ul> </li> <li>• <b>Pleura</b> <ul style="list-style-type: none"> <li>– Right collection: Present / Absent</li> <li>– Left collection: Present / Absent</li> </ul> </li> </ul>  | <b style="writing-mode: vertical-rl; transform: rotate(180deg);">Pericardium/pleura</b> |
| <ul style="list-style-type: none"> <li>• <b>Ascending aorta</b> <ul style="list-style-type: none"> <li>– Normal / Dilated (    mm)</li> <li>– Dissection Yes / No</li> </ul> </li> <li>• <b>Descending aorta</b> <ul style="list-style-type: none"> <li>– Normal / Dilated (    mm)</li> <li>– Dissection Yes / No</li> <li>– IABP Position NA / OK / High / Low</li> </ul> </li> </ul>   | <b style="writing-mode: vertical-rl; transform: rotate(180deg);">Aorta</b>      | <p style="font-size: 1.2em;">Signed: _____</p>  |   |
| <ul style="list-style-type: none"> <li>• <b>Notes</b></li> </ul>  |   | <ul style="list-style-type: none"> <li>• <b>Notes</b></li> </ul>  |   |

Version 1.1 May 2015 PD/KW – Based on St George's FIT scan by Fletcher/Edsell/Leech

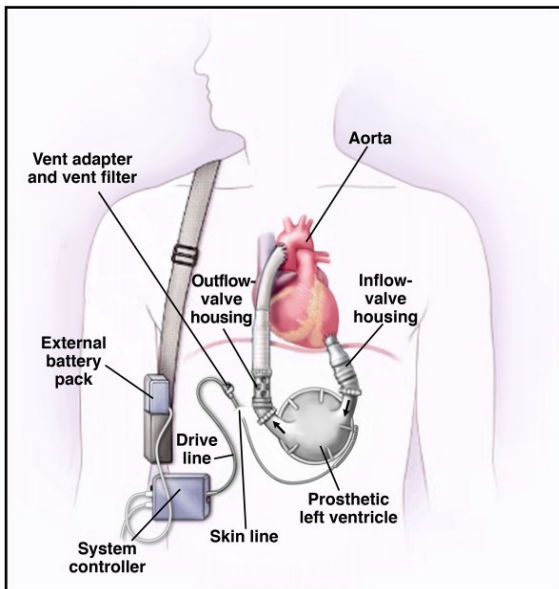
# Extra-corporeal assist devices and ECMO

Authors: Dr Andrew Richardson & Dr Kirstin Wilkinson

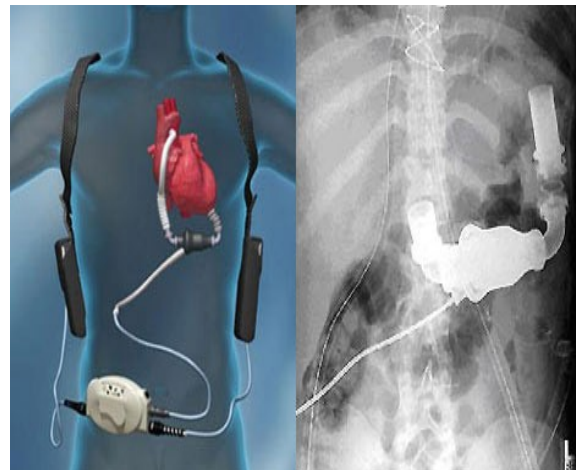
Reviewed: January 2016

## Ventricular Assist Devices

Patients facing imminent death due to cardiac failure who still have preserved function of other organs may be candidates for the implantation of ventricular assist devices (VADs). These consist of pump, inflow and outflow cannulae and a power lead, and may support the left, right or both sides of the heart. They also have a driveline exiting through the skin and connecting the pump to a console that provides controls and power supply. VADs may be pulsatile or non-pulsatile, implanted (intra-corporeal) or external (extra-corporeal), and short-, medium- or long-term. Cannulation sites for these devices are usually the left atrium or LV apex, with return to the ascending or descending aorta for an LVAD, and right atrium and pulmonary artery for an RVAD.



HeartMate XVE ventricular assist device.



EXCOR ventricular assist device by Berlin Heart

VADs may be used as a bridge to recovery, a bridge to cardiac transplantation, or as destination therapy. As we are not a transplant centre, and no local program with funding for destination VAD therapy exists, the use of VADs in this centre is exceedingly rare. However, caseloads can and do change over time, and it is possible that we will begin to encounter these devices in the future. The majority of patients only require support with an LV assist device; some require RVAD or even BiVAD therapy.

Four categories of patients are seen for VAD treatment:

#### Cardiogenic shock following cardiac surgery

Cardiogenic shock may occur rarely (<1%) post-cardiac surgery, as a result of severe ventricular dysfunction or intractable arrhythmias (e.g. VF). Much of this may be due to myocardial stunning, and if so, will frequently show recovery within 48 - 72 hours. Recovery is unlikely if it has not taken place after 5 - 7 days. Poor pre-operative ventricular function, significant perioperative MI and uncorrected cardiac lesions (valvular disease or coronary stenoses) all decrease the chances of a good outcome. Survival for patients requiring VAD therapy post-cardiac surgery ranges from 20 – 40%, but is significantly lower in those over 65 years of age (17% vs. 36%).

#### Cardiogenic shock following myocardial infarction (MI)

When optimal management using inotropes, revascularization and intra-aortic balloon counter-pulsation proves inadequate to deal with cardiogenic shock post-MI, there may be a role for VAD therapy. If recovery is going to occur, it will usually take place within 5 –7 days. In one study, 38% of these patients survived to hospital discharge.

#### Myocarditis and cardiomyopathy

Acute cardiomyopathies (e.g. acute peripartum cardiomyopathy) and myocarditis may produce haemodynamic compromise of sufficient severity to require VAD support. These cases show good survival rates (up to 70%) and high chances of recovery, with approximately half of acute myocarditis survivors recovering without transplantation.

Decompensated chronic heart failure is the most common indication for long-term VADs, either as a bridge to transplant or as destination therapy. Bridging to transplant is a highly effective treatment, with a 60 – 90% survival, and VAD therapy may improve success rates in comparison to treatment with inotropes.

### **Patient Considerations**

Patients should be on maximal medical treatment before assessment is made regarding the appropriateness of VAD therapy. The selection criteria for this treatment differ between acute and chronic heart failure. In this centre we are most unlikely to use VAD therapy for chronic heart failure patients.

Patients in acute heart failure should have a cardiac index < 2 l/min/m<sup>2</sup>, a PCWP > 20 mmHg, and a systolic blood pressure < 90 mmHg and signs of actual or impending organ dysfunction (e.g. a rising lactate, declining urine output), or be a surgical patient unable to be separated from cardiopulmonary bypass. They should already be receiving mechanical ventilation, inotropic and IABP support.

Once it is established that the above criteria are met, two crucial questions must be answered:

Is there a reasonable chance of recovery?

Is the patient a candidate for heart transplantation?

If the answer to both these questions is no, then VAD therapy should not be offered in the absence of a destination VAD program. It currently remains questionable as to whether transplant centres will readily accept referral of patients on VAD treatment from non-cardiac transplant centres, which may further limit the appropriateness of this intervention.

Consider early referral to transplant centre for advice.

### **Right Ventricular Assist Devices (RVADs)**

Although up to a third of patients with severe LV impairment have significant coexisting RV failure, less than 10% of patients receiving LVAD therapy require an RVAD. Generally RV function can be supported with inotropes, vasopressors and judicious fluid loading, whilst targeting a low pulmonary vascular resistance by avoiding hypoxaemia, hypercarbia and acidosis, high peak and mean airway pressures. Sometimes pulmonary vasodilators such as inhaled nitric oxide, nebulised prostacyclin or oral sildenafil are used to offload the RV,

whilst maintaining right coronary perfusion pressure. Tricuspid regurgitation usually resolves with the fall in pulmonary pressures that occurs as the left side of the heart is offloaded. The use of an RVAD increases the incidence of bleeding post procedure and reduces the chances of survival in comparison to an LVAD alone.

Risk factors for requiring an RVAD include a CVP > 16 – 20 mmHg, high right heart volumes and poor contractility, female gender, non-ischaemic cause of RV failure, and needing inotropic support preoperatively.

Severe ventricular arrhythmias were once thought to require BiVAD therapy, but if the pulmonary vascular resistance can be kept low enough; even VF may be tolerated with an LVAD alone. However, temporary RVAD support may be needed for intractable rhythm disturbances.

## **Other Considerations for VAD Therapy**

### Valvular disease

Aortic incompetence must be excluded before VAD placement, as the subsequent increase in aortic pressure combined with the lower intra-cavity pressure of the offloaded ventricle will worsen the regurgitation, causing ventricular distension and poor systemic flow. If aortic regurgitation is present, either valve replacement or valve closure with a pericardial patch will be needed at the time of VAD placement.

Where there is a diseased or prosthetic mitral valve, the drainage cannula of the VAD has to be placed in the left ventricular apex rather than the left atrium, as poor flow across the mitral valve leads to thrombosis and potential embolisation.

### Intra-cardiac Shunts

Any intra-cardiac connection (PFO, ASD, VSD, etc) that will allow right-to-left shunting when left sided pressures fall as the VAD pumps must be closed during the implantation procedure.

### Non-cardiac Issues

Severe liver dysfunction carries a very poor prognosis, and may contraindicate a VAD. Severe renal failure, in contrast, is no contraindication, as renal function usually improves following VAD treatment. Neurological function must be carefully assessed, especially in the context of cardiac arrest, pre-VAD implantation. Generally, an upper age limit of 65 years has been used for most VAD programs, as age above 65 years is a predictor of poor outcome.

## **Complications of VADs**

### Bleeding and thrombosis

VAD implantation is usually a bloody affair, requiring aggressive use of blood products and anti-fibrinolytics, with early re-operation if these are unsuccessful in procuring haemostasis. However, patients will often require anticoagulation after haemostasis is achieved to reduce the chance of thrombosis in the VAD.

### Low Pump Flows

Hypotension with poor systemic flow is a common problem in the post-implantation period. The differential diagnosis includes:

- RV dysfunction (the most common cause)
- Hypovolaemia
- Cardiac tamponade
- Obstruction of the pump inflow cannula

Whilst a low CVP might suggest hypovolaemia and a rising CVP one of the other problems, the definitive investigation is TOE, which will reveal the cause. The response of the VAD to hypovolaemia varies according to the device. Pulsatile devices may simply cycle slower, whereas non-pulsatile VADS may generate large negative left sided pressures, sucking the myocardium onto the inflow cannula, and stalling flow. Stalled flow is treated by reducing the pump speed to a minimum, and then filling the patient whilst slowly increasing the pump speed.



### Arrhythmias

Arrhythmias are common in these patients, and carry a high risk of thromboembolism: they should be treated aggressively with drugs, cardioversion and anticoagulation.

### Infection

Patients who are functionally immunocompromised by critical illness, who then receive extensive prosthetic implants are at huge risk of septic complications. Unsurprisingly, device-related infection has an incidence of over 25% by 3 months, and sepsis is one of the commonest causes of death in VAD patients. Prophylactic antibiotics and antifungals should be continued for at least 48 hours after implantation, and aggressive culturing and empirical antibiotic use should follow the mere suspicion of sepsis thereafter.

### Neurological Injury

Early neurological problems are usually caused by hypotension/hypoperfusion, and relatively rare. Late (weeks to months) neurological events are, however, common, and are usually related to thromboembolic complications of the device. VAD thromboembolism is frequently related to infection in the device, and the only effective may be to remove the device and perform cardiac transplantation.

### Device Malfunction

More than 10% of devices placed will malfunction. This may occur due to inflow valve regurgitation (in pulsatile devices), where blood leaks backwards into the LV, producing ventricular distension and poor forward flow. Alternatively, obstruction of either cannula may occur, either by the ventricular wall, or by thrombus.

## **Extracorporeal Membrane Oxygenation (ECMO)**

ECMO is the use of a modified cardiopulmonary bypass circuit to support failing respiratory or cardiac function, or both. It has been most widely used in the context of severe ARDS, and data from the Cesar trial suggests that ECMO increases survival among adult patients with severe but potentially reversible respiratory failure compared with conventional ventilatory support. It can also be used to provide biventricular support. However, its use for either purpose is generally restricted to certain specialist centres, although is on occasion used here.

### **Indications**

It is indicated for acute severe respiratory or cardiac failure that is potentially reversible, but has a very high predicted mortality (>80%) with maximal treatment.

The criteria for cardiac failure are the same as for VAD therapy, although for left ventricular failure, an LVAD is preferable to ECMO due to the immune activation and potential for complications that a circuit oxygenator brings with it. However, almost all VADs require sternotomy for implantation, whereas ECMO may be commenced via peripheral cannulation. Thus ECMO may preferable to VAD therapy where there is (1) concomitant significant lung injury or RV failure; (2) no recent sternotomy and urgent cannulation is required; (3) there is no institutional experience with VAD implantation.

### **Contraindications**

Absolute contraindications include:

- Contraindications to systemic anticoagulation
- Moderate or severe chronic lung disease
- Significant CNS injury
- Severe immunosuppression
- Very advanced multi-organ failure
- Underlying terminal disease

Relative contraindications would be:

- Age greater than 60 – 70 years
- Mechanically ventilated for > 10 days already (death on ECMO rises dramatically when ventilation duration exceeds 5 days)

## ECMO Circuits

There are two types of ECMO – venovenous (VV) and venoarterial (VA).

VV ECMO is generally used for respiratory failure with preserved cardiac function (even if dependant on inotropic support). Blood is drained from systemic veins, passed through the oxygenator, and returned to the right side of the heart, where it then passes through the heart and lungs as normal. It therefore requires adequate cardiac function, and, since not all the venous return will have passed through the oxygenator, the arterial oxygenation may be lower than normal.

VA ECMO is used for severe cardiac failure, and drains blood from systemic veins, returning via the circuit to systemic arteries, bypassing the heart and lungs completely. Normal blood gases and haemodynamics may be obtained in this way. There are, however, a number of potent disadvantages:

Complications of surgical cut down and arterial cannulation

- Risk of bleeding when heparinised
- Distal limb ischaemia
- Risk of gas embolism

Retrograde flow from the arterial cannula towards the heart may impose a high afterload, which compromises ventricular function and possibly recovery

Pulmonary perfusion is significantly reduced, potentially impairing recovery from acute lung injury

If the LV is completely non-functioning, any blood that does pass through the lungs is not ejected into the aorta, and progressive LV distension and pulmonary oedema occur, causing permanent ventricular damage

Upper body hypoxaemia may occur if there is reasonable LV function (i.e. VA ECMO is inappropriate), as there will be some blood passing through the non-functioning lungs and ejected via the LV, predominantly to the head and neck vessels

The ECMO circuit comprises:

- Drainage (venous or inflow) and return (arterial or outflow) cannulae
  - For VV ECMO, usually placed in the femoral (venous) and jugular (arterial) veins
  - For VA ECMO, usually placed in the jugular vein (venous) and femoral, axillary or carotid arteries (arterial)
- Pump
  - May be roller or centrifugal; roller pumps require a reservoir or bladder, centrifugal pumps can in certain conditions generate high negative venous pressure, causing haemolysis and increasing the risk of air embolism
  - Must be able to generate a minimum of 50 – 60 ml/kg/min, and potentially up to 100 ml/kg/min in septic patients (in children higher flow rates per kg are frequently used - often around 150ml/kg/min)
- Oxygenator
  - A PMP (Poly-Methy-Pentene) or Silicone oxygenator membrane is used
- Fresh gas flow (sweep speed) controls pCO<sub>2</sub>; determinants of pO<sub>2</sub> are more complex, but chiefly depends on circuit flow
- Heat exchanger
  - Integral to the oxygenator
- Tubing and bridge
  - The bridge is a line which bypasses the patient, allowing blood to be re-circulated within the extra-corporeal circuit and de-airing to occur in the event of an air embolism



## Management of ECMO

After cannulation, flow is gradually increased to the maximum that venous return allows. Once adequate flow is achieved, mechanical ventilation is reduced to “rest” settings (e.g.  $\text{FiO}_2$  0.3 – 0.5, PEEP 10 – 15 cm  $\text{H}_2\text{O}$ , rate 5/min). The reduction in mean airway pressure usually improves circuit flow. We will usually aim for full oxygenation. Oxygenator fresh gas flow is set to produce a  $\text{PaCO}_2$  of 5 – 6.5 kPa.

Blood gases are measured hourly, and anticoagulation monitored regularly with ACTs (hourly) and APTRs. Anticoagulation is adjusted by means of a heparin infusion to maintain an ACT of between 180 and 200 seconds. Anti-Xa levels should also be monitored as per the ECMO protocol. Platelet counts should be kept greater than  $100,000 \text{ mm}^3$ , and haematocrit above a minimum level of 30%. Haemolysis may be detected by regular measurement of free haemoglobin. Some authorities recommend routine daily circuit blood cultures.

## Complications and Problems during ECMO

### Circuit disruption

Massive problems require immediate clamping of the cannulae and removal from ECMO  
The ventilator is kept ready to be restarted for this reason

### Clot in the circuit

Small clots may not cause a problem, but larger ones require a change of pump head, oxygenator or of the entire circuit

### Air embolism

May occur due to excessive negative venous line pressure, partial venous cannula withdrawal exposing a side hole, or an oxygenator failure

Small volumes may be aspirated from the circuit, but larger volumes require clamping the cannulae while the air is removed

### Oxygenator failure

Oxygenator or circuit must be changed

### Recirculation

Occurs when oxygenated blood from the return cannula is promptly siphoned off by the venous cannula, resulting in shunt and hypoxaemia

Revealed by the venous  $\text{SaO}_2$  being greater than the patient's  $\text{SaO}_2$

Placing the drainage cannula in the IVC and the return cannula in the IJV reduces this problem, as does withdrawing both cannulae slightly

### LV distension

Discussed above; if increasing pump speed to empty the heart fails, then urgent LA venting is needed, either via thoracotomy or balloon atrial septostomy

### Upper body hypoxaemia

Discussed above; increasing pump speed to empty the heart may help, whilst changing to VV ECMO, or adding an additional return cannula to return to the IJV definitively solve the problem

### Bleeding

Minor bleeding is managed by keeping the platelet count  $> 100,000 \text{ mm}^3$

Major bleeding requires a higher platelet count ( $> 150,000 \text{ mm}^3$ ),  $\text{INR} < 1.5$ , fibrinogen  $> 2.0$  and consideration of anti-fibrinolytics

If bleeding continues then consideration should be given to reducing the heparin anti-coagulation

All invasive procedures can cause significant bleeding whilst on ECMO, and so require careful consideration of the risks and benefits

Procedures such as chest drain insertion should be performed using the smallest drain available, and with diathermy available

### Cardiorespiratory deterioration

With VA ECMO, sudden deterioration implies loss of circuit flow, possibly due to cannula displacement or hypovolaemia

With VV ECMO (and some patients on VA ECMO with significant contribution from their native cardiac output), it implies deteriorating cardiovascular function

Tension pneumothorax, haemothorax, cardiac tamponade, haemorrhage, myocardial ischaemia, arrhythmias and sepsis should be sought and excluded

### Sepsis

Bloodstream infection and UTIs are more prevalent in the ECMO population, increasing with duration of treatment

Lower respiratory tract infections occur with similar frequency to other ventilated patients

Daily blood cultures are recommended by some

Early use of empirical antibiotics should be considered

### **Weaning from ECMO (in adults)**

#### VV ECMO

When a progressively lower  $FiO_2$  in the circuit is needed to maintain oxygenation targets, it suggests that the lungs are improving.  $FiO_2$  low is gradually weaned down to .21%, and then in combination with invasive ventilatory settings the oxygenator fresh flow is turned off. If the arterial blood gases are satisfactory for a period of time the patient may be decannulated. During this trial period blood remains circulating within the ECMO circuit but no artificial gas exchange is performed. Respiratory recovery usually occurs by 1 to 3 weeks, but may take longer. Diuresis should be attempted in patients who fail to show improvement in respiratory parameters.

#### VA ECMO

Six to 12 hours prior to attempted weaning, a modest inotrope regime is commenced (equivalent to 0.1 mcg/kg/min adrenaline). Circuit flow is reduced to approximately 1 l/min over a period of between 12 and 24 hours during which cardiac function reviewed by regular TOE and haemodynamic parameters. If cardiac function has not recovered by 5 to 7 days, then recovery is unlikely.

# Specific Patient Groups on CICU

## Medical Patients Following Cardiac Arrest

Author: Prof Charles Deakin

Date: June 2015

The cardiac intensive care unit is receiving an increasing number of patients who have been successfully resuscitated from cardiac arrest, both from in-hospital and out-of-hospital arrests. These numbers are likely to increase as Southampton develops as a heart attack centre and resuscitation science improves the numbers of patients surviving the initial cardiac arrest. The intensive care burden from these patients is significant; they are usually extremely unwell and more than half will die before leaving the unit. These guidelines addressing post-resuscitation interventions are categorized into the following areas: (1) ventilation, (2) temperature control (therapeutic hypothermia and prevention and treatment of hyperthermia), (3) seizure control and sedation, and (4) other supportive therapies (blood glucose control, coagulation control, prophylactic anti-arrhythmic therapy). Careful post-resuscitation care has until relatively recently been overlooked in treatment guidelines, but now forms the final link in the Chain of Survival for these patients.



'Chain of Survival' for cardiac arrest

Intensive care units implementing management strategies as documented in these guidelines have seen a doubling in neurologically intact survivors leaving their intensive care units. This short document is written as an evidence-based management guide for doctors and nurses caring for these patients on the cardiac intensive care unit. It presents a summary of the science relating to specific areas of management and summarises guidelines for the management of these patients.

### MANAGEMENT OF AIRWAY AND BREATHING

- Consider intubation and ventilation of any patient unable to protect their airway, maintain normal gas exchange or maintain normal acid-base balance.
- Aim for PaO<sub>2</sub> in the range of 8.0 - 15.0 kPa and PaCO<sub>2</sub> in the range of 4.5-6.0 kPa.
- Insert a nasogastric tube.
- Obtain a chest X-ray and check for:
  - Correct tracheal tube placement.
  - Correct positioning of central line, Swan Ganz sheath/catheter, intra-aortic balloon pump, nasogastric tube etc.
  - Correct positioning of chest drain(s).
  - Evidence of rib fractures or haemo/pneumothorax.
- Ensure adequate depth of sedation.
- Consider neuromuscular blockade if high airway pressures or patient coughing.

Ensure adequate pain relief in patients with rib fractures secondary to external chest compression.

## **CIRCULATORY SUPPORT**

- Liaise with the on call cardiology registrar to ensure the appropriate administration of heparin (or its derivatives), aspirin and clopidogrel.
- Consider organising a baseline ECHO (if not recently performed) to assess structural and functional myocardial performance and exclude any correctable causes.
- Consider invasive cardiac output monitoring (LiDCO or Swan Ganz) if the patient requires high doses of inotropes or continues to deteriorate.
- Optimise potassium levels (4.0 – 5.0 mmol.l-1).
- Optimise magnesium levels (0.8 – 1.2 mmol.l-1).
- Consider intravenous amiodarone (loading dose then infusion) to control malignant or tachyarrhythmias (remember to check if a loading dose of amiodarone was given during the cardiac arrest).
- Check the CXR and urine output to ensure that the IABP is correctly positioned.

## **TEMPERATURE CONTROL**

### **Management of hyperthermia**

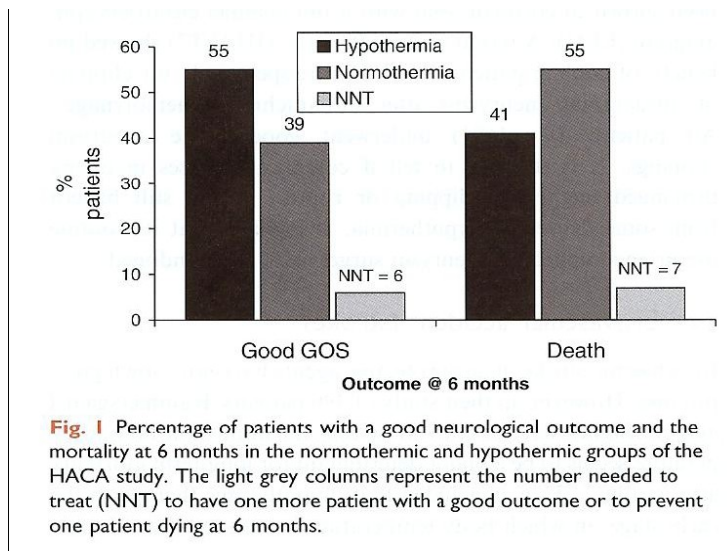
The risk of unfavourable neurologic outcome increases for each degree of body temperature above 37°C. Hyperthermia is associated with increased morbidity and mortality in post-stroke patients. Post-stroke pyrexia is not treated effectively by antipyretics such as paracetamol or ibuprofen. However, antipyretics or physical cooling methods have been associated with decreased infarct volumes in animal models of global ischemia.

- Avoid pyrexia (core > 37.0 °C) for the first 72 hours post-arrest.
- Measure and record core temperature at least hourly.
- Ensure core temperature is recorded at a suitable anatomical site (NOT axilla).
- If antipyretics are ineffective, use active cooling (tepid sponging, water blankets, cold i.v. fluids etc).

### **Therapeutic Hypothermia**

#### **Scientific Evidence**

Mild therapeutic hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury. These reactions include free-radical production, excitatory amino acid release, and calcium shifts, which can in turn lead to mitochondrial damage and apoptosis (programmed cell death). Two randomized clinical trials have shown improved outcome in adults who remained comatose after initial resuscitation from out-of-hospital VF cardiac arrest and who were cooled within minutes to hours after return of spontaneous circulation. Patients in these studies were cooled to 33°C (HACA Study group, *NEJM* 2002;346: 549-56) or to the range of 32 - 34°C (Bernard SA et al. *NEJM* 2002;346:557-63) for 12 to 24 hours. The Hypothermia After Cardiac Arrest (HACA – see graph below) study included a small subset of patients with in-hospital cardiac arrest. One study documented improved metabolic end points (lactate and O<sub>2</sub> extraction) when comatose adult patients were cooled after ROSC from out-of-hospital cardiac arrest in which the initial rhythm was PEA/asystole (Hachimi-Idrissi S, et al. *Resuscitation*. 2001;51:275-81). A small study has also shown benefit after therapeutic hypothermia in comatose survivors of non-VF arrest (Bernard SA, et al. *Ann Emerg Med*. 1997;30: 146-153). A recent study has suggested cooling between 32- 36°C gives better outcomes. The European Resuscitation Guidelines are currently being updated to incorporate this latest study. Current CICU guidelines are to cool between 32 - 36°C for 24 hours.



External or internal cooling techniques can be used to initiate cooling. The only studies documenting improved outcome with therapeutic hypothermia after cardiac arrest used external cooling. An infusion of 30 mL/kg of 4 °C saline achieved a decrease in core temperature of approximately 1.5 °C. One study in patients with cardiac arrest and 3 other studies have documented that intravascular cooling enables more precise control of core temperature than external methods.

Multiple studies in animals document the importance of initiating cooling as soon as possible and for adequate duration (12 to 24 hours). In an animal study looking at the effects of delays in initiating cooling (Carroll et al. Metab Brain Dis 1992), the effects were dramatic. Whilst caution must be used applying animal studies to clinical situations, this and several other studies do demonstrate the time-dependent nature of neuroprotection from cooling. Optimal cooling parameters, including onset, depth, and duration of cooling, are unknown.

GOS = Glasgow Outcome Score

### Treatment Recommendations

Current treatment recommendations were published in 2003 by the International Liaison Committee on Resuscitation (ILCOR) and are shown below. (As mentioned earlier, these guidelines are currently being updated. CICU guidelines will be updated as needed.)

***Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32°C to 34°C for 12 to 24 hours when the initial rhythm was VF. Cooling to 32°C - 34°C for 12 to 24 hours may be considered for unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest from any other rhythm or cardiac arrest in hospital. [Circulation 2003;108:118-12]***

At the time these guidelines were written, insufficient evidence existed to recommend the use of therapeutic hypothermia in rhythms other than VF. Since 2003, further studies have shown similar benefit in patients resuscitated from PEA and asystole and these guidelines therefore refer to patients resuscitated from all arrhythmias. These studies have reported the use of therapeutic hypothermia in relation to patients resuscitated from out-of-hospital cardiac arrest, but it is logical to apply the same treatment to patients remaining comatose following resuscitation from in-hospital cardiac arrest too.

### Methods of cooling

External and/or internal cooling techniques can be used to initiate cooling. An infusion of 30 ml/kg of 4°C N.saline decreases core temperature by 1.5°C. Although this equates to approximately 2000ml, studies have specifically reported that this volume does not cause pulmonary oedema secondary to acute heart failure. This technique should be considered as a method of rapid induction of hypothermia as cooling blankets that are currently used on CTICU are slow to achieve their effect. Intravascular cooling catheters are the most effect method of cooling. They are not currently available routinely on CTICU. Intravascular cooling enables more precise control of core temperature than external methods, but it is unknown whether this improves outcome compared with cooling blankets.

### Complications of cooling

Complications of mild therapeutic hypothermia include:

- Increased infection risk (particularly chest infection)
- Cardiovascular instability, coagulopathy, hyperglycaemia
- Electrolyte abnormalities such as hypophosphataemia and hypomagnesaemia

Shivering will necessitate sedation and intermittent or continuous neuromuscular blockade. Use of continuous neuromuscular blockade could mask seizure activity.

#### Rewarming from therapeutic hypothermia

When the 24–48 hr period of hypothermia is finished, the patient can be rewarmed. When using an intravascular cooling catheter, aim for 0.25–0.5°C/h. A similar rate can be achieved by allowing passive rewarming if the patient has been cooled using a water blanket. Remember not to let the temperature overshoot (> 37.0 °C), particularly in the first 72 hours post-arrest.

- Unconscious adult patients with spontaneous circulation after cardiac arrest should be cooled to 32–34°C. Cooling should be started as soon as possible and continued for at least 12–24 h.
- DO NOT DELAY INSTIGATION OF COOLING. COMMENCING COOLING IS OF THE UTMOST URGENCY.
- Rewarm the patient slowly (0.25–0.5°C.h<sup>-1</sup>) and avoid hyperthermia.
- Consider neuromuscular blockers if the patient is shivering.
- Correct hypophosphataemia and hypomagnesaemia.
- Be ready to treat hyperglycaemia as per CTICU protocols.

### Neurological Management

#### Prevention and Control of Seizures Scientific Evidence

Seizures and/or myoclonus occur in 5%–15% of adult patients who achieve ROSC, and in approximately 40% of those who remain comatose. Prolonged seizure activity may cause cerebral injury, and can cause life-threatening arrhythmias and respiratory arrest. Seizures following cardiac arrest should therefore be treated promptly and effectively.

Anti-epileptic drugs such as benzodiazepines, phenytoin, propofol or barbiturates are all suitable choices to control epilepsy. Maintenance therapy should be started after the first event once potential precipitating causes (e.g., intracranial haemorrhage, electrolyte imbalance, etc) are excluded. Have a high index of suspicion for status epilepticus in any patient who is slow to wake following cessation of sedation and beware of neuromuscular paralysis masking seizure activity.

- Seizures should be treated promptly and effectively. Involve the neurology team if there is any doubt as to the management of this condition. Consider benzodiazepines, phenytoin, propofol or barbiturates.
- Treat any hypotension arising from the use of these drugs.
- Beware of neuromuscular paralysis masking seizure activity.

#### Other Supportive Therapies

##### Blood Glucose Control:

- Monitor blood glucose frequently and treat hyperglycaemia as per protocols
- Inotropes and hypothermia may trigger or worsen hyperglycaemia.

##### Coagulation Control

- Anticoagulation may be required in patients following PCI or thrombolysis.
- Liaise with the cardiology registrar to ensure that appropriate anticoagulation is prescribed.
- In patients not receiving formal anticoagulation, subcutaneous heparin is usually indicated as DVT prophylaxis.

# Management of Cardiology Patients requiring Emergency PCI

Author: Dr Paul Diprose

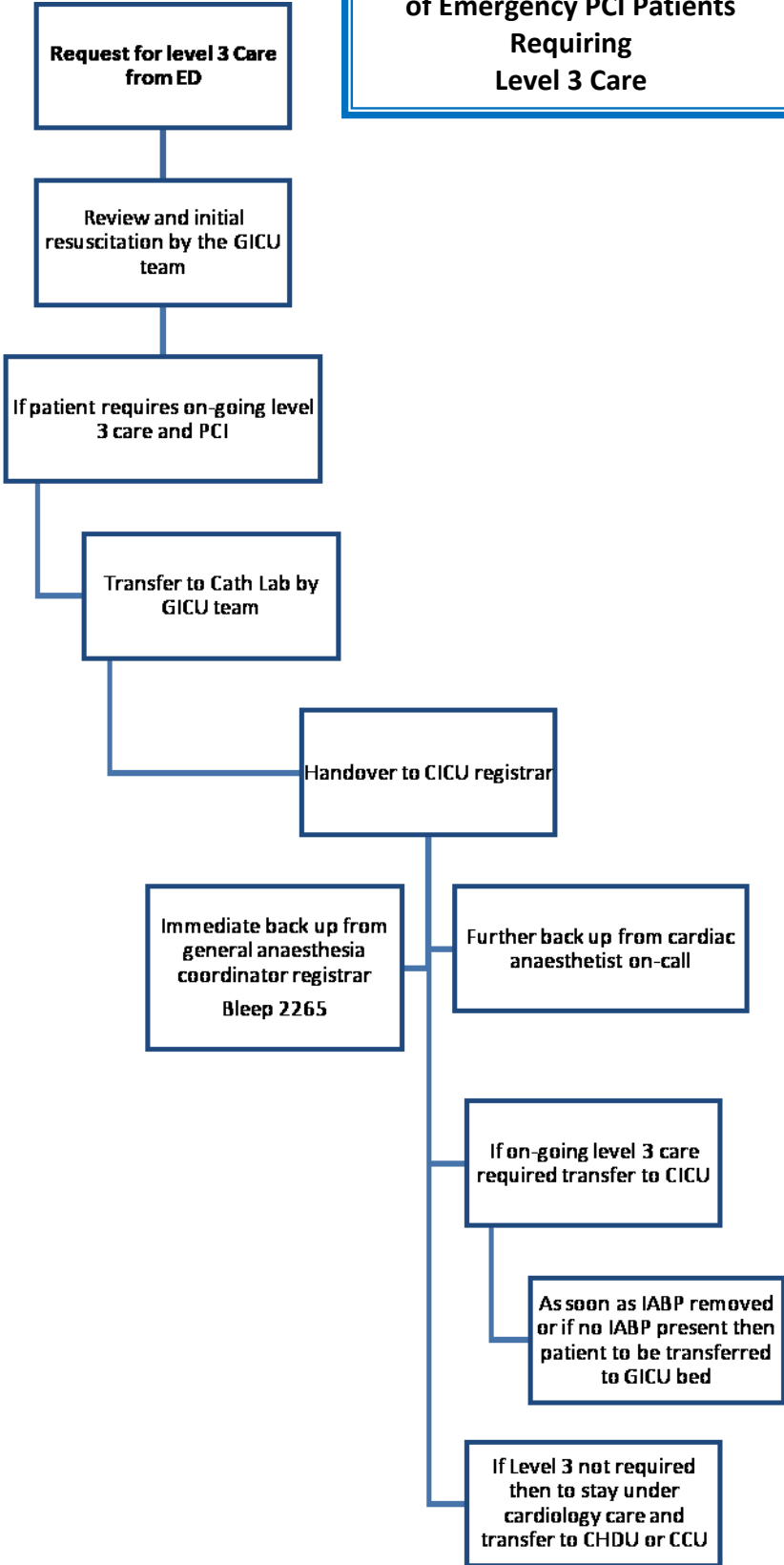
Revised: April 2015

Patients presenting in cardiac arrest in consequence of an acute coronary syndrome are managed in the Emergency Department (ED) by the ED and General Intensive Care (GICU) teams. If successfully resuscitated, they frequently require emergency percutaneous coronary intervention (PCI). In order to avoid any confusion, the agreed protocol for the management of these patients is detailed below.

1. Patients will be reviewed and resuscitated in ED as normal by the GICU team.
2. If decision is made that emergency PCI is required then GICU team will transfer the patient to the catheter lab. The patient will remain under the clinical care of the GICU team until arrival in the catheter lab.
3. At this point the care will be handed over to the CICU registrar on duty who will take over care after a handover from the GICU team.
  - An ODP will be made available (if necessary by the postponement of other theatre cases).
  - An appropriate ventilator and a fully stocked cardiac anaesthesia trolley must be available in any lab taking emergency PCI patients needing or likely to need level 3 care.
  - During office hours senior clinical support while in the catheter lab will be from the CICU consultant on duty.
  - Out of hours the on-site senior clinical support will be from the on-call coordinating SpR (bleep 2265).
  - If further assistance is required out of hours then the duty cardiac anaesthetist will be called.
4. If a patient requires level 3 care they will be transferred from the cath lab to CICU if an IABP has been inserted. If no balloon pump, the patient should go to GICU.
  - Otherwise, patients will remain under the care of the cardiologists and transferred to either CCU or medical CHDU.
5. The patient will stay under the care of the CICU team until the IABP is removed. Once the IABP is removed then the patient will be transferred to the care of the GICU team in a 'general' badged bed.

Anaesthetic management of these cases follows the principles of managing any patient with critical coronary disease, i.e. controlling the balance of myocardial oxygen supply and consumption through manipulating heart rate, blood pressure, haemoglobin, oxygen saturation, sedation and body temperature. Management of dysrhythmias is also integral to the care of these patients, as is safe provision of 12 to 24 hours of therapeutic hypothermia (see chapter on care of the post-cardiac arrest patient), which should start during the cardiac arrest.

**Algorithm for the Management of Emergency PCI Patients Requiring Level 3 Care**





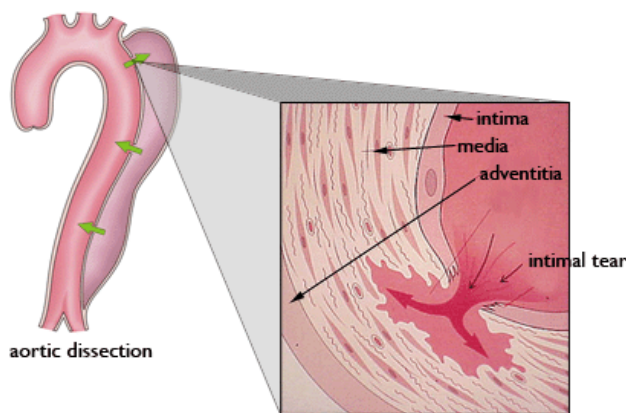
# Aortic Dissection

Author: Dr Nick Goddard

Revised: Sept 2015

## Introduction

Aortic dissection is a spontaneous intimal tear of the aorta with subsequent false passage of blood under systemic pressure between the layers of the tunica media. Extension may be proximal or distal. Blood may re-enter the true lumen at any point. When this involves aorta proximal to the left subclavian it is always classified as Stanford Type A. Where the tear is limited to beyond the origin of the left subclavian artery, it is classified as Stanford Type B. [1] Type A dissection is one of the most common cardiac surgical emergencies. In contrast Type B dissection is usually managed conservatively.



In the DeBakey classification [2], Type I and II dissections originate in the ascending aorta (Type II = no extension, Type I = distal extension to arch/descending aorta), where Type III is confined to the descending aorta. Unlike the Stanford classification, the additional subdivision into Type I and II may suggest involvement of distal organs and potential for malperfusion. The European Society of Cardiology Task Force on Aortic Dissection has also produced an etiological classification [3] further defining subtypes of 'aortic syndrome' (these may or may not be treated as a surgical emergency depending on circumstances – see bottom).

*Layers of the Aorta. From: The Marfan Syndrome, by Reed E. Pyeritz, M.D., Ph. and Cheryll Gasner, M.N., C./F.N.P. Fifth Edition, July 1999, Revised September 2001 Publisher: The Marfan Foundation (with permission)*

## Risk factors

Aortic dissection is often an isolated condition, frequently occurring in older men with essential hypertension and usually at the greater curve of the aorta (mechanical stress is greatest at this point), usually less than 10cm from the aortic valve. It may also arise in association with other conditions and syndromes:

- Bicuspid aortic valve (associated with aortic stenosis)
- Marfan's syndrome
- Ehlers-Danlos syndrome
- Rarely complicating difficult aortic cannulation prior to CPB

## Clinical Presentation

The typical story for acute Type A (< 2 weeks from onset) is central chest pain with or without haemodynamic instability. Instability usually arises following proximal rupture of the adventitia into the pericardium (causing tamponade) or distal rupture into the mediastinum (causing haemorrhagic shock). A variety of mechanisms may also lead to an incompetent aortic valve. In addition, where the dissection flap obstructs aortic branches, malperfusion of distal organs may occur. In fact, any branch of the aorta (including the brain vessels and coronary vessels) may be obstructed by the dissection flap. This may lead to:

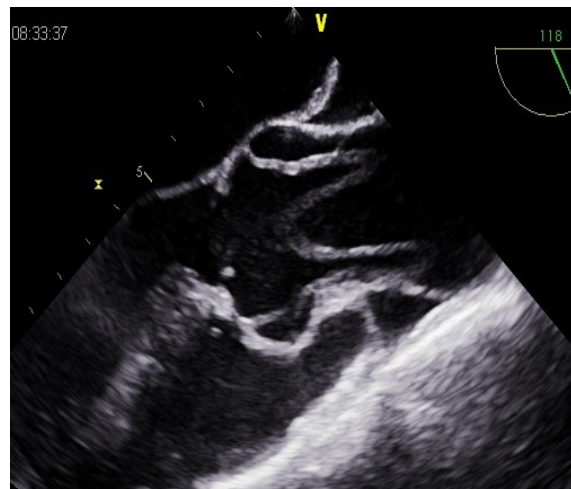
- Coronary Ischaemia (MI, arrhythmia, cardiac impairment), cerebral ischaemia (CVA), spinal ischaemia (paraplegia), renal ischaemia (AKI) or disparate distal pulses (i.e. pulse deficit in affected territory/ limb)

Malperfusion phenomena are associated with poorer prognosis. Overall for acute Type A dissection (<2 weeks from onset) mortality increases by 1-2% per hour delay to repair to around 90% at 30 days. In contrast, operative mortality for many centres is in the range 15-30%. [4]

### Imaging

Choice of imaging is based on practicality and availability as much as preference. Most stable patients are diagnosed following CT scan, but some may be picked up via echocardiography or as an incidental finding. Even when the diagnosis is confirmed, surgeons frequently will not know exactly what operation to perform until the chest and aorta are opened and the intima can be inspected. The entry point of the tear (which must be surgically excluded from the 'true' circulation) must be completely identified. If a portion of the tear remains above the cross clamp, circulatory arrest and more complex distal repair are likely. 'Live' imaging is undertaken in theatre by the anaesthetist with TOE and will give

helpful information in relation to confirming diagnosis, assessment of heart function and valves, as well as helping to determine the type and extent of operation to be undertaken. The Role of TOE in theatre is summarised in table 1.



*Acute Aortic Dissection as seen via transoesophageal echocardiography depicting a large circumferential dissection flap involving the proximal ascending aorta back to level of the coronary sinuses of Valsalva. Involvement of one or more sinuses is usually an indication to replace the aortic root rather than perform a supracoronary ascending aorta replacement only.*

### Role of TOE

| Question:   | Role:  |
|---|--|
| Correct diagnosis?                                    | Alternate diagnoses possible: leaking aneurysm? or intramural haematoma? – <i>type and timing of surgery will vary</i> |
| Where is the tear?                                    | i.e. - Can surgeon get clamp above?  |
| Aortic valve competent? + Is the aortic root dilated? | Native valve may be preserved + IP graft can be considered   |
| Tamponade/ RWMA's?                                    | Likely to result in CVS instability/ may involve coronary ostia if RWMA  |
| Track extent into descending aorta                    | Correct placement cannulae/ tubes in true lumen  |

### Anaesthetic Management:

*On arrival to SGH -*

- Be organised! – Crossmatch form ready for 6 units, bloods (clotting), consent, sticky labels

- ABG (+/- TEG/ multiplate may be useful) as many dissections are initially given concurrent dual antiplatelet agents +/- full dose LWMH – mistaken as acute MI
- Systolic BP control ~100-110 systolic (remember may require analgesia and/or have a full bladder after transfer)
- Examination (document neurology +/- GI exam)
- Check last oral intake
- Provide an opportunity (brief) to speak to relatives
- Transfer to Anaesthetic room

*Induction (Surgeon should be present) –*

- Two radial arterial cannulae (as right subclavian may be involved in dissection)
- PA sheath + CVC
- NIRS monitoring + TOE
- If full stomach – modified RSI (i.e. controlled haemodynamic transition with cricoid)
- Consider all line placements awake (including TOE in extremis) with surgeon and theatre team scrubbed pre-induction in theatre

**N.B.** - Type A Dissections may deteriorate at **any time** prior to or after induction of anaesthesia or following instigation of CPB. This may result in malperfusion of end organs which may or may not be detected by cerebral infrared spectroscopy NIRS). Superlative intra-arterial monitoring and attention to detail help in recognition and prevention. [5]

*Going on to CPB –*

- Access to chest via midline sternotomy
- Vigilance for clinical deterioration (particularly going on to CPB). Blood pressure may rise if a cardiac tamponade is released on chest opening.
- Cannulae placement depends on stability/ circumstances but clearly cannot be the ascending (diseased) segment of aorta -  
*[Historically, peripheral cannulation (fem-fem) was 'usual' as provides good access and flow rates. However, retrograde flow in the aorta may adversely affect flow dynamics between true and false lumens and also risks pressurisation of the false lumen if incorrectly sited. Retrograde emboli of aortic atheroma are also possible. Therefore, RA (venous) to right subclavian/ axillary inflow is a good alternative if coming off the 'true' aortic lumen. However, neither is perfect and all cannulation sites have the potential to result in malperfusion of distal organs]*
- Once on CPB, cool to 18-24 degrees (surgical preference) in anticipation of hypothermic circulatory arrest (HCA) which may be required for an open distal anastomosis

*Surgical Techniques –*

- Usually high aortic cross clamp + inspection of aorta internally
- Concurrent inspection of aortic valve and annulus (may need AVR or resuspension)
- Range of operations possible: AV resuspension/ replacement/ IP graft or aortic root replacement (proximal). For distal portion, straight anastomosis of root/ IP graft (if tear below cross clamp) +/- elephant graft or even full arch replacement if tear extends distally
- If composite graft used, coronaries will also need to be re-implanted
- If cannot get above tear with cross clamp, hypothermic circulatory arrest will be required (remember ice and steroids)

### While on CPB (= Organ Protection) –

- NIRS should be at least within 20% of baseline – look for differential readings + most will receive anterograde perfusion during circulatory arrest (discuss with surgeon/ perfusionist)
- Observe and maintain UO +/- frusemide infusion (bear in mind pre-op low perfusion state + CPB +/- haemoglobinuria with pump suction)

### Coagulation –

- Long CPB + lengthy suture lines under arterial pressure may be difficult to control
- Multiple blood products +/- novo 7
- 20-30ml 10% calcium gluconate during products (to counteract rapid citrate infusion)
- Use both lab and near patient tests
- Avoid hypothermia + hyperthermia (i.e. aim 36-37 degrees)

### Post-op/ICU -

- Systolic < 120 or surgical preference (can use nitroprusside 1-5mg/hour)
- Ongoing organ protection (as above) +/- low dose dopamine for AKI/oliguria
- UO > 200ml/hour if any signs haemoglobinuria
- 30 degree head up/ avoid tight ETT ties
- Normocapnea + avoid hyperthermia, Na 135-145, glucose 4-8
- Cefuroxime 48 hours + vigilance with surgical sites (chest and groin if femoral access)

## Aortic Syndrome

The focus of this article has been the anaesthetic and intensive care management of emergency Type A dissection. However, surgery is occasionally undertaken for type B dissection when medical therapy fails and where TEVAR is inappropriate. As the management of open surgery for type B dissection shares many of the issues associated with the management of both Type A dissection as well as the management of thoraco-abdominal aneurysms, no further discussion will take place here (as this will be discussed in a separate article on thoracoabdominal aneurysms.)

However, a range of emergency conditions with similar characteristics involving the aorta may also require surgical management. These are classified under the umbrella term 'aortic syndrome' and share in their pathophysiology the common pathway of breakdown of the intima/ media followed by haematoma formation/ separation of aortic wall layers +/- dissection, pseudoaneurysm and/or rupture. Aortic syndrome is classified according to table 2 below: [3]

|          |  |
|----------|--|
| Class 1: | Classic Aortic Dissection (with true and false lumens +/- communication) |
| Class 2: | Intramural haematoma   |
| Class 3: | Subtle or discrete dissection (with bulging of the aortic wall)          |
| Class 4: | Ulceration of aortic plaque (followed by rupture)                        |
| Class 5: | Iatrogenic or Traumatic dissection                                       |

Table 2: Aortic Syndrome Classification (European Society of Cardiology 2001) [3]

According to the 'Guidelines on the diagnosis and treatment of aortic diseases', produced by the European Society of Cardiology in 2014, [6] emergency surgery is indicated for intramural haematoma in the ascending aorta/arch if complications arise (e.g. pericardial effusion, periaortic haematoma or for large aneurysms). Otherwise, surgery can usually be undertaken 'urgently' (i.e. within 24 hours of diagnosis). For penetrating aortic ulcer, the aim of treatment is to prevent progression, and indications for intervention include recurrent,

refractory pain, or signs of contained rupture (e.g. rapidly growing aortic ulcer with associated periaortic haematoma or pleural effusion). Since establishing UHSFT as a major trauma centre in 2012, traumatic dissection/injury is also increasingly presenting to UHS and may urgently involve cardiac anaesthetists. Following trauma, the commonest injury site is the isthmus (descending aorta) following rapid deceleration. Intervention should take place urgently in order to avoid free rupture (not consistent with survival) and any treatment should take into account other injuries and their respective management.

For aortic syndrome affecting the descending aorta (and trauma in particular due to the frequency of aortic isthmus injury), TEVAR has lately become the preferred technique due to its lesser morbidity/ invasiveness when compared to open thoracotomy, one lung ventilation, partial cardiopulmonary bypass and deep hypothermic circulatory arrest. Current evidence suggests a survival advantage over open procedure (9% vs 19%) as well as decreased incidence of paraplegia, although endoleak is reported in up to 5.2% and stent collapse in 2.5%. [6] However, there may be technical reasons a stent cannot be deployed, which may include lower extremity artery disease, severe tortuosity of the iliac arteries, sharp angulation of the aortic arch and/ or the absence of a proximal landing zone for the stent graft. Where TEVAR is not possible or even available, open surgical repair/ intervention is the default. In the future as technology and experience improves, there is the potential for considerable overlap with hybrid open and stent approaches for complex lesions.

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# Thoraco-abdominal Aneurysm Surgery (CICU Care Guideline)

Authors: Dr Nick Goddard and Dr Tom Pierce

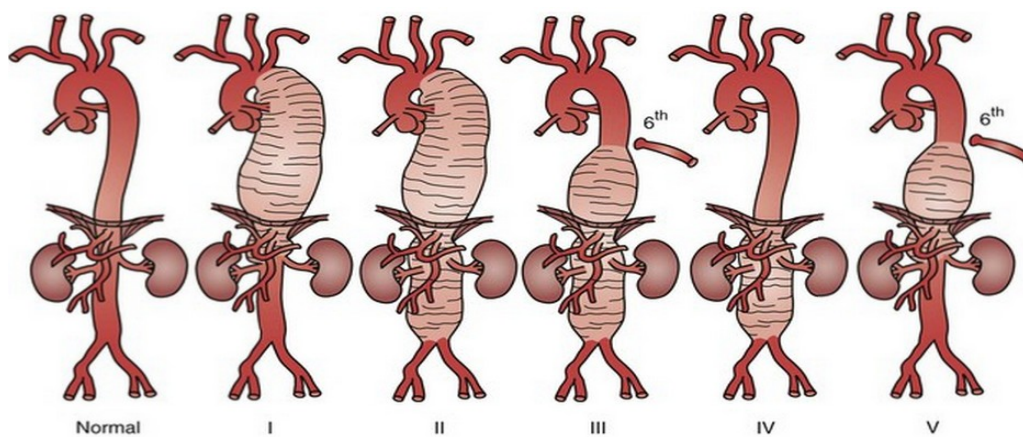
Date: Dec 2015

## Introduction

Aortic aneurysms can occur at any point along the thoracic or abdominal aorta. The majority of aneurysms are abdominal (accounting for about 75% of the total) and are managed by vascular surgeons. In contrast, thoracic aortic aneurysms (TAA) and thoracoabdominal aortic aneurysms (TAAA) are usually managed by cardiothoracic surgeons because access to the chest is required and because cardiopulmonary bypass (CPB) may be needed. In recent years stent technology has improved significantly such that in the near future there will be far greater overlap between open and stent procedures.

## Principles of Care

In open thoracic aortic surgery, the key aim of surgery is to minimise (or avoid) the adverse consequences arising from aortic cross-clamping. Where an aneurysm is more *proximal*, a higher cross-clamp will render a greater number of organs ischaemic and place more strain on heart ejection. Where an aneurysm is more *extensive*, a longer cross-clamp time will increase the duration of ischaemia. When planning the surgical technique, the Crawford Classification provides a useful reference. Created in 1986, and modified by Safi et al (to include 'Type V'), it describes all suprarenal aneurysms distal to the left subclavian: [1]



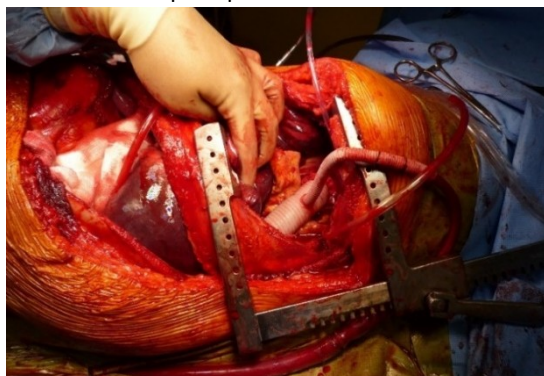
Modified Crawford Classification for thoracic and thoracoabdominal aortic aneurysms: Available at: [Clinicalgate.com](http://clinicalgate.com/thoracic-and-thoracoabdominal-aortic-aneurysms/).  
<http://clinicalgate.com/thoracic-and-thoracoabdominal-aortic-aneurysms/>. (Permission obtained)

|                             |   |
|-----------------------------|---|
| <b>Type I</b>               | Descending thoracic aorta to abdominal aorta (above renal arteries)   |
| <b>Type II</b>              | Descending thoracic aorta to below renal arteries +/- beyond to bifurcation                                 |
| <b>Type III</b>             | Mid/distal descending thoracic aorta involving most of abdominal aorta to bifurcation                       |
| <b>Type IV</b>              | Includes upper abdominal aorta +/- infrarenal aorta   |
| <b>Type V (added later)</b> | Mid/distal descending thoracic aorta to suprarenal aorta – includes coeliac and SMA but not renal arteries. |



## Outcomes

Historically, outcomes relating to thoracic aneurysm surgery were very poor but have improved dramatically in modern times. This is due to a combination of improved surgical technique, better organ protection and advances in perioperative care. Published mortality averages 5-12%, with paraplegia rates at 10% (up to 30% for Type II repairs), renal failure 13% and CVA risk around 8%.



*Lateral thoracotomy + deflation of lung to expose proximal descending thoracic aortic aneurysm and recurrent laryngeal nerve (tip of surgeons' forceps)*

[2,3] For all patients that were operated on between 2000 and 2015 at UHSFT (n=67), the mortality was only 3%, spinal cord injury 1.4% (2 patients – 1 with numbness, 1 with lower limb weakness but still able to mobilise), renal failure 10% (permanent renal replacement therapy in 2%) and CVA 10% (8% permanent). These figures include 25 patients with TAAA (37%) and 20 cases which were classed as emergency (30%). There have been no patients operated with resulting paraplegia. [personal communication]

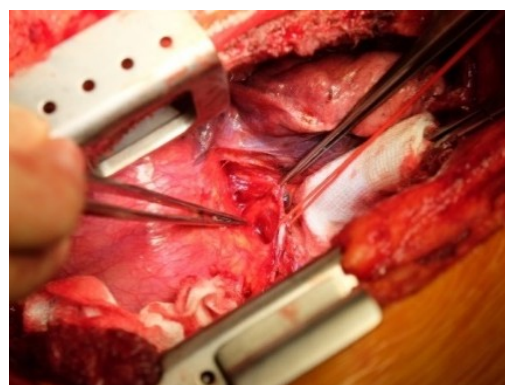
## Intraoperative Care

### Surgical Technique

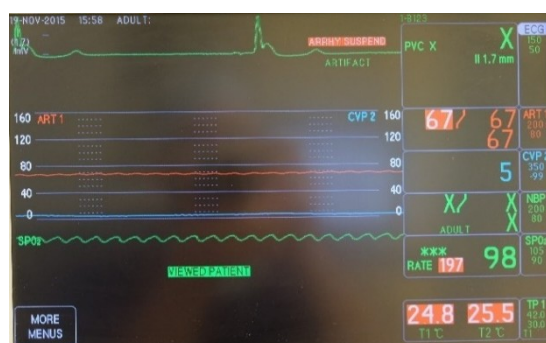
The aim of surgery is to remove the diseased section of aorta and replace it with a synthetic (usually Dacron) graft. Access depends on the location of aneurysm, but a full thoracoabdominal incision is usually necessary for Crawford types II, III, IV. The left lung must be deflated to expose the thoracic aorta. Following this, the muscular portion of the diaphragm is divided (avoiding the phrenic nerve), and abdominal organs are mobilized to expose the abdominal aorta.

A cross-clamp may then be applied to the aorta just proximal to the aneurysm. However, this will create unacceptably high obstruction (afterload) to left ventricular ejection, leading to cardiac distension, ischaemia and failure. Instead the heart can be offloaded using 'left heart bypass'

In this technique, pulmonary vein inflow to the left atrium is diverted through an external circuit before being returned to the distal aorta or common femoral artery. Temperature may be maintained at 32-34°C using a heat exchanger (lower would increase the risk of ventricular arrhythmias).



*Flow may be restored to the head and neck via a graft side branch. The distal aorta may then be sequentially clamped as major vessels are re-anastomosed. Note also the extent of incision – posterolateral thoracotomy through 6<sup>th</sup> intercostal space extending across the midline as far as the umbilicus.*



*Cooling via CPB to achieve DHCA. Note appearance of J waves on the ECG below 27°C*

An alternative (in the majority of cases at UHSFT) is to arrest the circulation in order to sew in the proximal anastomosis after the patient is rendered hypothermic (deep hypothermic circulatory arrest or DHCA). Full CPB is used to cool the patient to 18°C and then the circulation is turned off. Once the proximal graft is sewn in place, circulation to the head and neck may be resumed via the arterial cannula from the bypass circuit or via a

side branch from the new graft (see below). Perfusion to the lower limbs and/or viscera may be separately supplied from the bypass circuit during this time.

Following completion of the proximal anastomosis, major vessels are sequentially anastomosed, including the largest intercostal artery or 'arteria radicularis magna' (artery of Adamkiewicz) followed by the visceral ostia.

In practice, the coeliac, superior mesenteric artery (SMA) and right renal may be joined together for convenience as a single patch. The final stage is to complete the distal anastomosis followed by restoration of full flow to the lower limbs.

#### **Organ protection techniques:**

Long, complex surgery results in significant interruption of flow to organs including the spinal cord in particular. 'Adjunctive' techniques aim to minimise harm resulting from organ ischaemia. The following are accepted adjunctive techniques:

- Left heart bypass + distal visceral organ perfusion
- CSF drainage (indirect manipulation of spinal cord perfusion pressure)
- Hypothermia (Passive or via CPB circuit)
- Deep hypothermic circulatory arrest (DHCA)
- Reattachment of intercostal arteries
- Epidural cooling
- Monitoring of spinal somatosensory and motor evoked potentials

The particular techniques chosen will depend on the type of aneurysm and all the above may be appropriate for Crawford Types I and II aneurysms. For a full description of intra-operative anaesthesia, please refer to the detailed guide provided by Dr Pierce, located in the Theatre C anaesthetic room resource folder.

#### **Postoperative Care:**

Postoperative blood pressure should not be allowed to rise excessively (typically maintained < 110 – 120 systolic for first few hours). However, a balance must be struck between the risks of bleeding and adequate perfusion of organs recently rendered ischaemic. The principles of organ protection commenced in theatre will continue into the post-operative period on CICU. The most salient organs in need of protecting are:

- Spine (see below), kidneys and GI tract

Patients will typically arrive on CICU with a spinal (CSF) drain which should be left on free drainage and pressure limited to 10 mmHg (see below). A furosemide infusion +/- low dose dopamine (or dopexamine) infusion may help maintain renal perfusion pressure and urine output. N-acetylcysteine (NAC) is commenced in theatre and will still be running (when this finishes, there is no requirement to start another bag).

#### **CSF (Spinal) Drain:**

Blood flow to the spinal cord, and hence neurological function of the lower limbs is dependent on spinal cord perfusion pressure (SCPP) where  $SCPP = MAP - \text{extrinsic pressure on spinal cord}$ . The SCPP may be modified directly by increasing the arterial supply pressure to the spinal cord or indirectly by reducing extrinsic pressure on the spinal cord which in turn is largely dependent on CSF pressure in normal circumstances. CSF pressure is affected by postural changes and may be artificially controlled using a lumbar spinal drain. At UHS we currently use the 'Medtronic Becker Extraventricular Monitor and Drainage system' (figure 1). The system can be confusing to those unfamiliar with its layout.

The desired pressure of CSF is prescribed (or fixed) by adjusting the level of the black double arrow on the collection chamber ('pressure line' is written clearly - figure 2) relative to a zero point (figure 3). The height of the collection chamber is manually adjusted so that it is always set to 10 mmHg. This means that any pressure rise above 10 mmHg will result in CSF spilling over into the collection chamber. Therefore, the printed light-blue and white-background scales (mmHg and cmH<sub>2</sub>O respectively) that rise vertically alongside the collection



chamber are not for measuring the pressure of CSF but rather for reference when ‘fixing’ the pressure. The chamber can periodically be emptied from time to time. Where ongoing drainage is seen this is indirect evidence that the drain is patent and working. The zero point is set at the level of the atria. This is achieved by adjusting the height of the device (usually suspended from a drip stand). Once set up there is no requirement to change any settings and the drain should be left on free drainage.

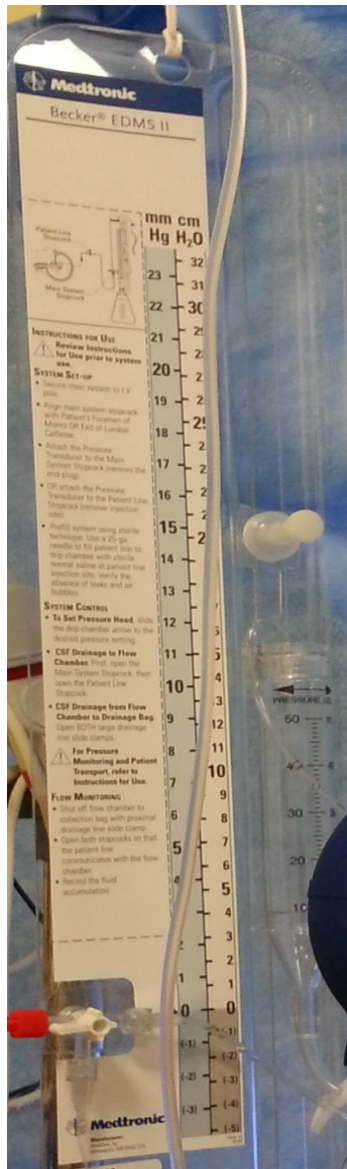


Figure 1 - Medtronic Becker spinal drainage system – CSF from the patient ascends the height of the thin drainage tube (arrowed). The prescribed pressure is aligned with the double arrow on the collection chamber.



Figure 2 - ‘Pressure line’ double arrow set (prescribed) to 10 mmHg by adjusting height of the collection chamber

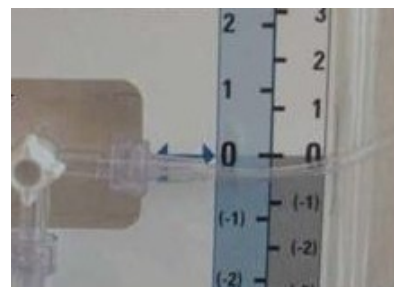


Figure 3- Medtronic Becker spinal drainage system – Zero point

### **Drain Management:**

Spinal drains are not without risk and may lead to epidural/spinal haematoma, meningitis, fistulation and subarachnoid haemorrhage. Drains may be confused with epidurals and/or venous lines resulting in inadvertent injection of drugs. Drains must always be clearly labelled and access ports should be taped off. Spinal drains should only be managed in the intensive care environment. [4]

Cord compromise may be immediate or delayed and vigilance in early detection is key to preventing paraplegia. An early sedation hold and assessment of neurology should take place the morning after surgery. If the patient can **convincingly** move their lower limbs, this is good evidence that cord perfusion is not compromised and the drain may be clamped from 24 hours onwards. However, if there is doubt the following actions should be considered and the patient reassessed:

- Lie the patient flat and check function of drain/ re-site if necessary
- Ensure adequate O<sub>2</sub> delivery (SATS 95%), cardiac index (>2.5L/min/m<sup>2</sup>), Hb > 100 g/dL
- Ensure MAP > 90 mmHg (SCPP > 80 mmHg)

Where paraplegia is evident or where there is a new, unexplained decrease in GCS, or where there is blood stained CSF drainage the following action should take place:

- Inform consultant on call and consider urgent CT scan head and spine

A CT scan of the spine will exclude cord compression due to an epidural haematoma and a CT brain will exclude intracranial haemorrhage. In contrast, if there have not been any neurological concerns, the clamped drain may be removed at 72 hours provided that neurology is normal, clotting corrected, platelet count is > 100 and S/C heparin has been discontinued for > 8 hours or more.

### **Further ICU care:**

An NGT will usually be sited in theatre, so a postoperative CXR is necessary to confirm the position. ETT position is also important, as in the event of accidental extubation, laryngeal oedema following a double lumen tube may make reintubation more challenging. Rifampicin/ vancomycin powder is applied before wound closure and a combination of cefuroxime and metronidazole given intraoperatively. A further dose should be administered after 8 hours.

Pain relief may be provided by remifentanyl infusion while intubated followed by a thoracic epidural prior to extubation. Thoracic epidurals are extremely effective. However, where the block is not perfect, topical lidocaine patches (5%) should be considered as supplementary analgesia. Up to three may be applied near to the wound with a 12-hour life span.

### **References:**

1. Safi HJ, Miller CC 3rd. Spinal cord protection in descending thoracic and thoracoabdominal aortic repair. *Annals of Thoracic Surgery* 1999; (67):1937-9; discussion 1953-8
2. Fann JL. 'Descending thoracic and thoracoabdominal aortic aneurysms' *Coronary Artery Disease*; **13**: 93-102
3. Zierer et al. 'Elective Surgery for Thoracic Aortic Aneurysms: Late Functional Status and Quality of Life' *Annals of Thoracic Surgery*; **82**: 573-8
4. Field M, Doolan J, Safar M et al. 'The safe use of spinal drains in thoracic aortic surgery' *Interactive Cardiovascular and Thoracic Surgery* **13**(6): 557-565

# Revision Fontan patients in CICU

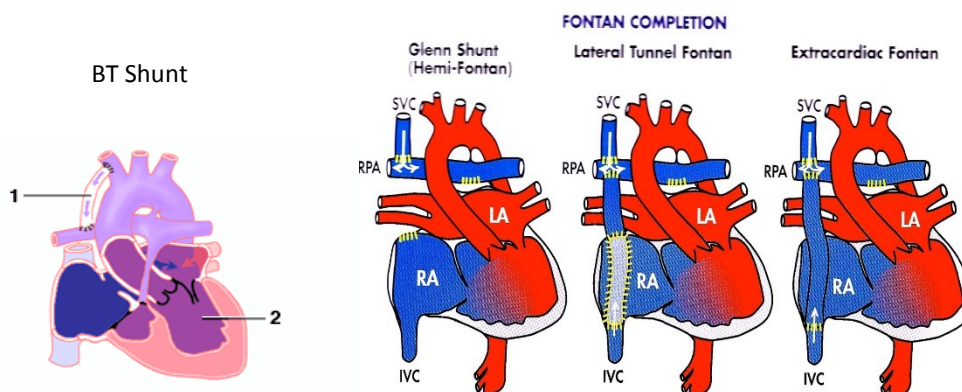
Authors: Dr Richard Cope & Dr Kirstin Wilkinson

Revised: January 2016

## Introduction

Single ventricle/ Fontan physiology comprises a large number of underlying lesions e.g. hypoplastic left heart syndrome, tricuspid atresia. Long term survival depends on the type of lesion and degree of co-existing cardiac malformations. Patients with a 'Fontan' type circulation have previously received multiple cardiac operations to permit survival with a single functioning ventricle.

The 3 main stages of converting the circulation into a Fontan physiology are usually Blalock- Taussig shunt, Glenn (hemiFontan) shunt and Fontan procedure (total cavopulmonary connection, TCPC). These are shown below,



As techniques developed, the operation changed from a classical Fontan (atriopulmonary connection) to a lateral tunnel Fontan to an extracardiac Fontan to try and prevent the complications of atrial arrhythmias and thrombosis. Patients with complications from one of the original types of Fontan operation may be offered revision Fontan surgery. Southampton is established as a centre with good results for this operation and has had a large caseload over the years. There are fewer patients suitable for this operation now but the odd case still happens.

The absence of an effective RV means that the RA pressure has to be substantially higher than the LA pressure in order to drive blood across the pulmonary vascular bed. The higher RA pressure is pivotal in the pathophysiology. Those patients that present to CICU will do so after revision surgery usually to manage the inevitable atrial dilation (causing arrhythmias and clot formation) that occurred with the classical type of palliative procedures.

These are complex difficult patients to look after and require a multidisciplinary approach. They are high risk cases and some are higher risk than others. The surgery is difficult and time consuming and so the patients have the potential to be sick on arrival in the Unit. The purpose of the surgery is to convert an older style 'atrial' Fontan circulation (in which the right atrium is connected directly to the pulmonary arteries) into a new modern one in which the SVC is connected directly to the pulmonary arteries and with an extra cardiac conduit running from the IVC to the pulmonary arteries. The older atrial Fontan patients end up with very larger right atriums that form a reservoir for clot and for arrhythmia formation. A bi-atrial maze and right atrial reduction is also performed with a permanent pacing system implanted at the end of the procedure, since the SA node is removed at the time of surgery.

### *Preoperative Problem List*

- Multiple previous cardiac surgeries
- Pre-operative medications (warfarin, beta blockers and ACE inhibitors)
- Borderline renal function
- Sometimes borderline myocardial function plus other issues (e.g. valve dysfunction)
- Borderline cardiac output and 'Fontan' circulation
- Cirrhosis with portal - systemic shunt
- Red cell fragility
- Low clotting factor levels and platelet counts
- Protein losing enteropathy with immune suppression from low immunoglobulin levels

### *Peri-operative Problem List*

- Risk of low output state during dissection which can take a long time
- Risk of bleeding during dissection
- Risk of low pressure on bypass due to cirrhosis, medications and inflammatory response
- Risk of haemolysis on bypass due to a long bypass time, the need for a lot of surgical suction and to red cell fragility
- Risk of bleeding after bypass due to raw surfaces, length of bypass and coagulopathy related to warfarin and possibly cirrhosis
- Risk of problems related to pacing (poor rhythm control)
- Risk of low output due to ischaemic period or preoperative ventricular dysfunction, bleeding, pacing issues and 'Fontan' circulation

### *Postoperative Problem List*

- Risk of low output due to ischaemic period or preoperative ventricular dysfunction, bleeding, pacing issues and 'Fontan' circulation
- Risk of bleeding (see above)
- Risk of inflammatory response from factors above including low output state and blood products
- Risk of renal failure due to preoperative and perioperative factors including cirrhosis
- Risk of lung injury
- Serious infection risk

### *ICU Management*

The commonest problems in the early postoperative phase are:

- Bleeding
- Haemolysis
- Poor urine output
- Poor oxygenation
- High trans-pulmonary gradient
- Pacing / rhythm disturbance
- Ventricular dysfunction
- Low cardiac output

Bleeding is managed in the conventional manner and the haemolysis (if present) and poor urine output is generally managed with furosemide usually by infusion. The big problem with these patients is optimising oxygen delivery to the tissues in the face of the remaining factors on the list above.

### *Ventilation*

This is a complex issue in these patients since cardiopulmonary interactions are exaggerated from normal. Ventilation can have a serious adverse effect on cardiac output and as a result of this respiratory complications have a much bigger impact than usual on the patient and greatly increase the risk of a poor outcome. To understand why this is it is important to review more normal cardiopulmonary interactions.

## Interactions Affecting the Right Ventricle

During normal ventilation the negative intrathoracic pressure generated during inspiration acts to draw blood into the thorax and thus improves right ventricular filling. During IPPV the reverse is true and this effect is worsened by hypovolaemia leading to a fall in blood pressure and cardiac output (very common on induction of anaesthesia followed by ventilation).

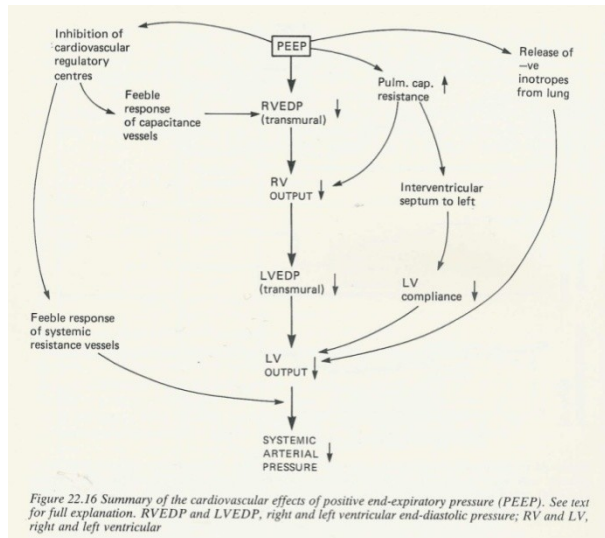


Figure 22.16 Summary of the cardiovascular effects of positive end-expiratory pressure (PEEP). See text for full explanation. RVEDP and LVEDP, right and left ventricular end-diastolic pressure; RV and LV, right and left ventricular

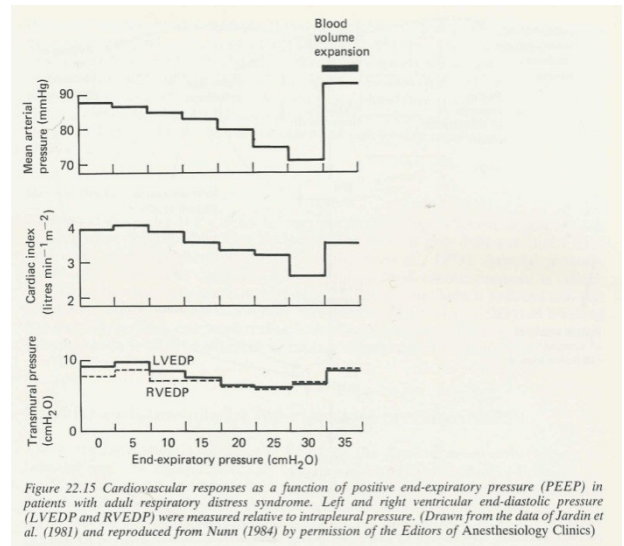


Figure 22.15 Cardiovascular responses as a function of positive end-expiratory pressure (PEEP) in patients with adult respiratory distress syndrome. Left and right ventricular end-diastolic pressure (LVEDP and RVEDP) were measured relative to intrapleural pressure. (Drawn from the data of Jardin et al. (1981) and reproduced from Nunn (1984) by permission of the Editors of Anesthesiology Clinics)

The first of the above figures demonstrates the effect of PEEP on cardiac index in the normal patient and the second one shows some of the reasons why this happens. As mentioned above this is often in the context of induction of anaesthesia as well, which can cause myocardial depression and vasodilatation in addition to the effects of positive pressure ventilation. The situation is made worse if the patient has poor right ventricular function and requires a higher than normal preload to maintain stroke output. Of course if you don't have a right ventricle as in the case of the Fontan circulation the effects are exaggerated greatly because there is no pump to drive blood through the lungs, only venous pressure.

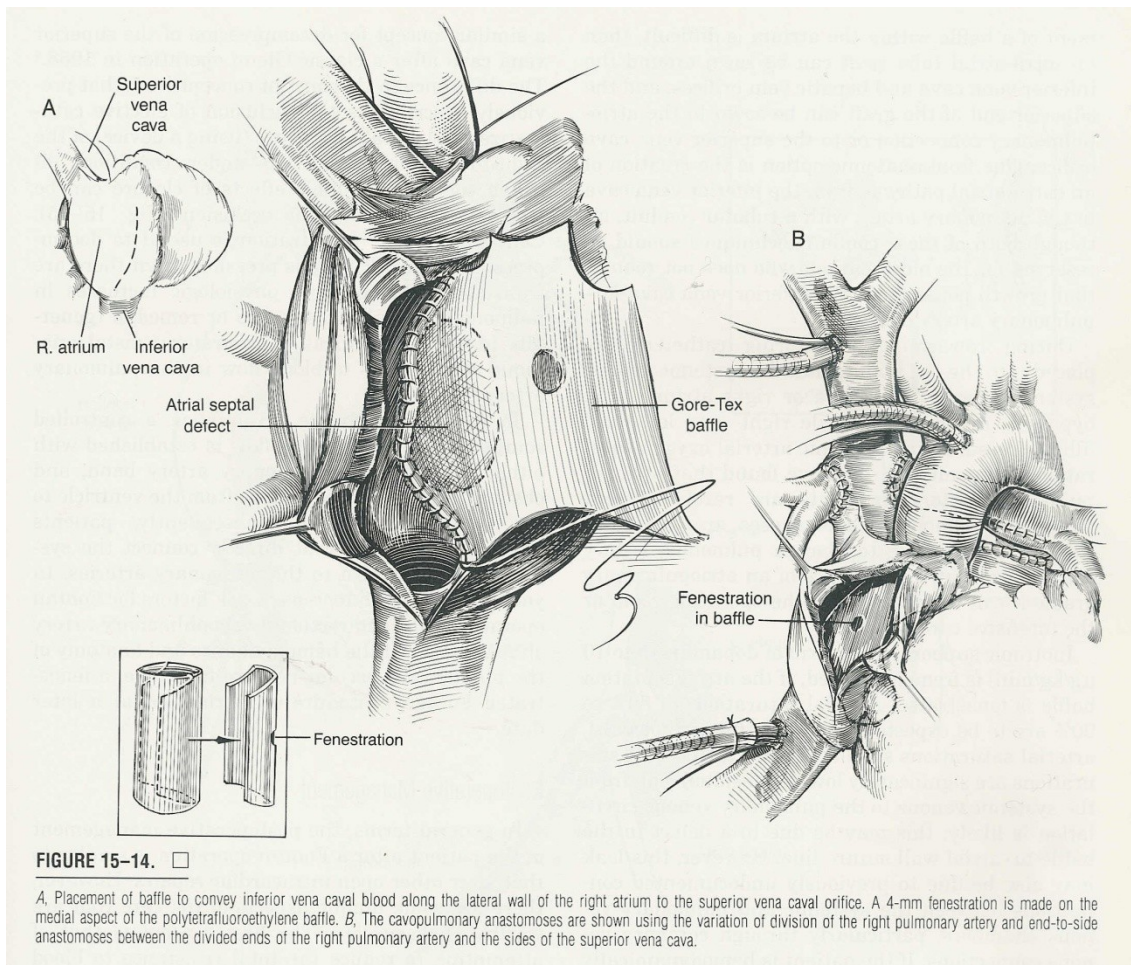
## Interactions Affecting the Left Ventricle

In patients with normal left ventricular function the impact of preload on the right side generally predominates. However in the setting of left ventricular failure there is often a high left atrial pressure and a 'back pressure effect on the right side (sometimes leading to high systemic venous pressures as well). This will tend to reduce the impact of positive pressure ventilation on the right ventricular preload. The effect that predominates in this situation is the reduction of left ventricular after load. When the pressure in the thorax is raised the 'pressure gradient' between the arterial system inside and the arterial system outside is reduced. This reduces the pressure that the left ventricle has to generate for a given blood pressure. This explains the beneficial effects of positive pressure ventilation and CPAP in patients with left ventricular failure.

## Interactions in the Patient with a Fontan Circulation

In these patients 'right sided' interactions predominate as ventricular failure severe enough to raise the atrial pressure significantly is very poorly tolerated and the patient will not survive for long under these conditions. Reasonable ventricular function is required for survival in the Fontan patient. Hence IPPV can reduce cardiac output by up to 30%. Also factors effecting PVR, such as hypoxia, hypercarbia and acidosis have a bigger impact in the Fontan patient than in patients with two ventricles. One way of protecting the patient from this effect to a degree is the surgical technique known as a fenestration. This is a hole or a channel between the IVC conduit and the common atrium. This is perhaps easier to understand if we take the extreme example of a very high PVR. In this situation pulmonary blood flow will be dramatically reduced and so the ventricle will become empty and cardiac output will fall. The systemic venous pressure however will be very high. The fenestration acts as a 'blow off' valve allowing blue blood to enter the atrium directly thus reducing the venous pressure, allowing filling of the atrium and the maintenance of some cardiac output, albeit at a lower oxygen saturation level.





The above picture shows this in an older style 'lateral tunnel' Fontan in which the IVC is connected to the pulmonary arteries using a baffle up the inside of the right atrium.

### Causes of Hypoxia

Poor oxygenation is a common problem after this surgery and the difficulty is deciding whether this is due to a 'normal' respiratory complication or due to right to left shunting within the Fontan circulation. A low cardiac output will make the effect of this shunting greater. Other causes of hypoxia include all the usual respiratory complications that can occur after cardiac surgery.

#### Causes of right to left shunting:

- Fenestration
- Obligatory shunt from coronary sinus within the right atrium
- Abnormal collateral vessels which connect the venous system to the arterial system with in the lungs or more directly (atrial or arterial - venous connections)

#### Other causes of hypoxia (not an exhaustive list):

- Malposition of ET tube
- Collapse – atelectasis
- Effusions
- Pneumothorax
- Consolidation
- Lung injury

These patients are more prone to respiratory complications than most as well as being subject to right to left shunts.

### The Decision to Extubate

This is extremely difficult and requires careful discussion and planning. The advantages of early extubation are very significant, but the high risk of respiratory complications after this difficult and time consuming surgery makes this a risky strategy in itself. There is no right or wrong answer here and every case must be judged on its merits. On balance it would seem that overnight ventilation is a good idea rather than the conventional approach of extubating early. By the next day it may be more obvious as to whether extubation is appropriate.

### Respiratory Complications

Collapse, consolidation and pneumonia can lead to very severe hypoxia (due to obligatory shunts) and can massively increase trans-pulmonary gradient and reduce cardiac output due to an effect on PVR. This happens because of hypoxia and hypercarbia, increased mean airway pressure and interstitial oedema collapsing pulmonary vessels.

### Ventilation Strategy in the Event of Respiratory Complications

This is problematic because modern protocols for RDS will not work well due to the effect on cardiac output. There is a need to keep the mean airway pressure as low as possible (minimal peep, high peak airway pressure) bearing in mind that hypoxia and hypercarbia can worsen cardiac output.

### Management of Oxygen Delivery

- Optimise oxygenation (may not be easy)
- Extubate when appropriate to optimize cardiac output
- Optimise preload and afterload
- Optimise rhythm, heart rate & contractility
- Optimise haematocrit

Preload in the Fontan circulation is very important and needs to be much higher than normal. Immediately out of theatre the venous pressure may well need to be 20 mmHg for an adequate cardiac output because of the raised pulmonary vascular resistance (and hence trans-pulmonary gradient). Afterload needs to be appropriate but is not as critical. A very high afterload will increase atrial pressure and so increase the venous pressure needed to produce an adequate cardiac output. A very low afterload will lead to a very low blood pressure and possibly to organ failure. Optimizing rhythm usually means sorting out any issues that there are with pacing and this may require a return to theatre if this is not right. The rate is generally set a little higher than normal at around 100 beats per minute. Contractility is optimized usually with the aid of echo. Inotropes used include dopamine, dopexamine, milrinone, and adrenaline. Noradrenaline may be used when the afterload is inappropriately low, but has the disadvantage of probably increasing the PVR the most.

Since these patients are generally hypoxic and have borderline cardiac outputs it is important to maintain a good haematocrit for oxygen delivery. This is usually between 35 and 40 depending on oxygenation.

Measuring cardiac output in these patients has proved difficult.

### Sepsis

The risk of this is quite high due to immune suppression and it is very hard to treat because there is a very limited ability to increase cardiac output. Vasoconstrictors increase pulmonary vascular resistance and can decrease cardiac output. Ventilation is often needed in addition which further reduces cardiac output and of course pulmonary sepsis is very poorly tolerated for all the reasons mentioned above. For these reasons close attention to antibiotic therapy is needed. It is possible that they should also be reverse barrier nursed although we have not gone down this route as yet.

### Renal Failure

There is a relatively high risk of this because:

- Some of these patients have poor renal function pre operatively
- There is a lot of potential for a low output state as discussed
- Increased red cell fragility and long bypass times lead to haemolysis
- There is a risk of hepatorenal syndrome as a result of the liver disease

Filtration may be needed when this occurs.

### *Summary*

One of the most important factors in determining the outcome for these patients is their preoperative state. Some of these patients are in very good condition preoperatively and generally they tolerate the surgery very well. The patients who are in a poor condition preoperatively have a very high risk, although exactly what this risk is uncertain. The worst cases tend to suffer from all of the problems mentioned above and the usual cause of mortality is renal failure and sepsis.



# Transcutaneous Aortic Valve Implantation

Author: Dr Paul Diprose

Revised: February 2016

The patients for these procedures have in the past been 'non-surgical' candidates with severe aortic valve disease; as such they will have multiple co-morbidities and will frequently be very elderly. However, the indications for TAVI are changing all the time and there are trials underway comparing standard AVR with a sternotomy to the transcutaneous route.

There are two kinds of technique, one called trans-apical where a surgeon passes the new aortic valve under image guidance from the apex of the LV via a mini-thoracotomy and one entirely trans-femoral approach (surgeons are still required for femoral wound closure). All the procedures take place in the catheter lab with CPB stand-by and cell salvage available. Post-operatively, the trans-apical cases are managed on CICU; some selected trans-femoral cases will be sent direct to CHDU.

## Care for all TAVI Patients

- Limb perfusion should be assessed regularly (as for IABPs) since wide bore femoral access would have been made and surgical reconstruction (or percutaneous closure with the 'ProGlide' device) of the femoral artery is performed at the end of the procedure
  - This is particularly important after the trans-femoral approach but should also be performed for the trans-apical patients because they will also have femoral artery cannulation
- All patients should be kept warm with standard cardiovascular medications
- Prescribe standard medications including the following
  - Paracetamol 1g PO/IV Qds prn
  - Morphine 1-2mg IV boluses prn
  - At least one anti-emetic
  - Aspirin 75 mg PO od (to start on first post-op day)

## Trans-femoral cases

- 1g Paracetamol would have been given in the cath lab
- 10-20ml 0.5% bupivacaine to wound would have been given by surgeon
- Many of these patients would have had no anaesthetic and minimal sedation
- Those patients that have had a GA will usually be extubated before admission to CICU/CHDU
- Patients should have standard cardiac monitoring
- Patients can eat and drink as normal

## Trans-apical cases

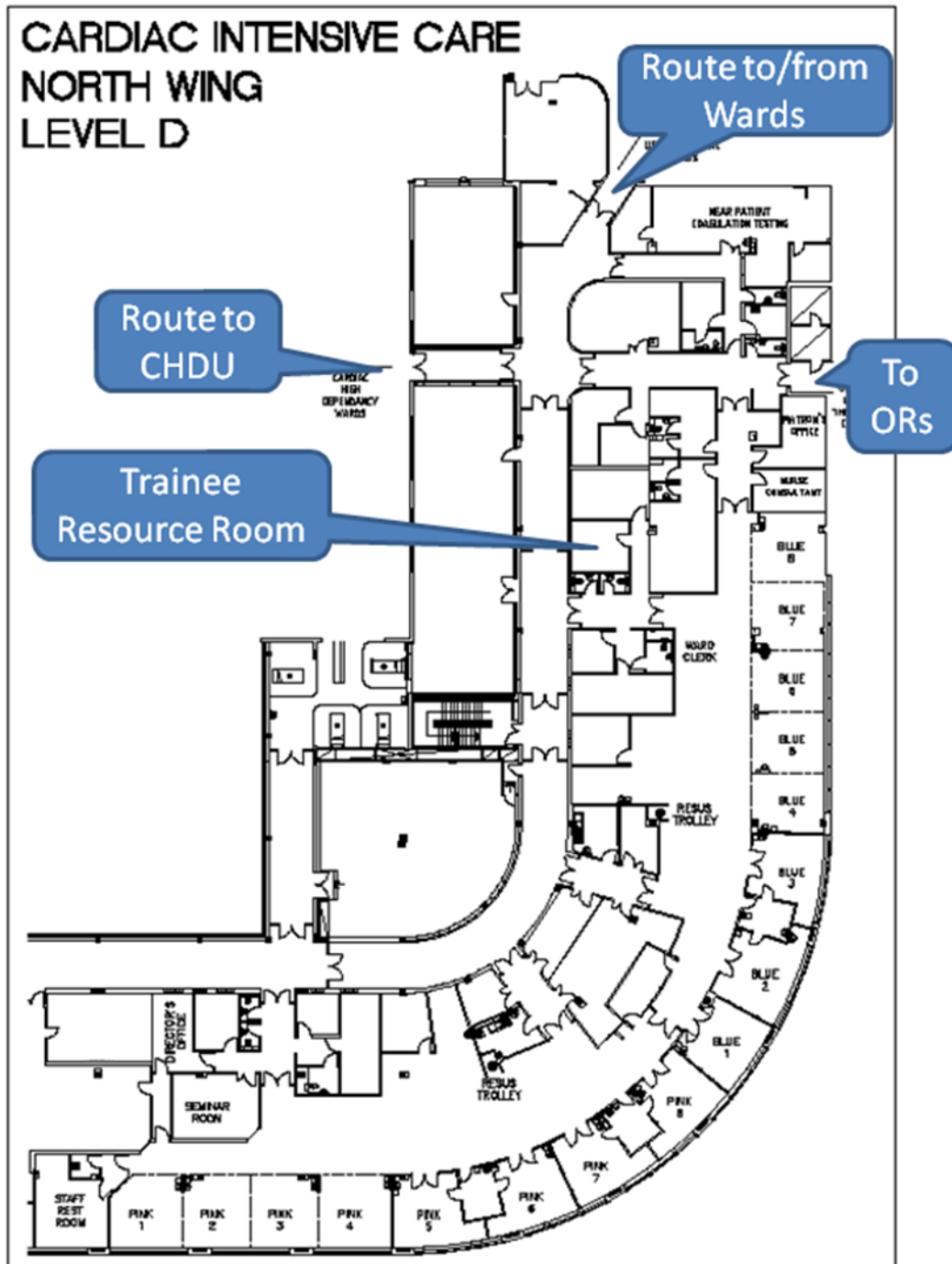
- Will have an extra-pleural catheter sited by the surgeon
- 10ml 0.5% bupivacaine bolus is given before leaving lab
- Extra-pleural infusion of 0.125% or 0.25% bupivacaine
  - Start infusion at 8ml/hr
- They are transferred to CICU on propofol IV infusion for sedation
- Should be extubated when warm and stable (usually within 1-2 hours)
- Before extubation
  - Give 1g IV Paracetamol
  - Ensure that the extra-pleural infusion is running
  - Have IV Morphine 1mg/ml ready for titration
- Extra-pleural infusion to run at 8ml/hr for first 24-48 hours, and then can be removed
- Consider paravertebral or thoracic epidural if intercostal catheter not working well
  - Although this is not usually required

### *Complications after TAVI*

- Bleeding
  - This can occur with either TAVI approach.
  - Trans-femoral cases - bleeding may be concealed with blood tracking into the retroperitoneal space
  - Trans-apical cases – bleeding most likely to occur from the left ventricular apex access site
  - Management
    - Avoiding excessive spikes in blood pressure (particularly for T-A cases)
    - Detection and correction of coagulopathy
    - Maintaining a haemoglobin concentration >9g/dl
  - Occasional patients will need to return for surgical re-exploration
- Heart block
  - AV block can develop in 5% or more of patients after TAVI
  - If this develops then the on-call cardiology team should be contacted urgently
  - Patients will have a central venous sheath in place that can permit (with x-ray guidance) the pacing of a temporary venous pacing wire
  - Occasional patients may require urgent placement of a PPM to restore a sequential pacing (DDD) – particularly important for patients previously in sinus rhythm with diastolic dysfunction
- Stroke
  - The incidence of stroke appears to be higher with TAVI than with conventional AVR
  - If identified, then this should be discussed with the CICU consultant on-call and an urgent CT brain +/- referral to stroke team organised
- Para-valvular leaks
  - This is much more common after TAVI than conventional AVR
  - They will usually be picked up intra-operatively
  - Associated with a poor long term outcome
  - Becoming less likely with more modern valve developments

# Appendices

## Layout of cardiac Intensive Care Unit



## Equipment list for CICU

All trainees starting on the unit should be familiar with the equipment used. If further training is required then the trainee should discuss with their educational supervisor. Use the checklist below to identify training needs.

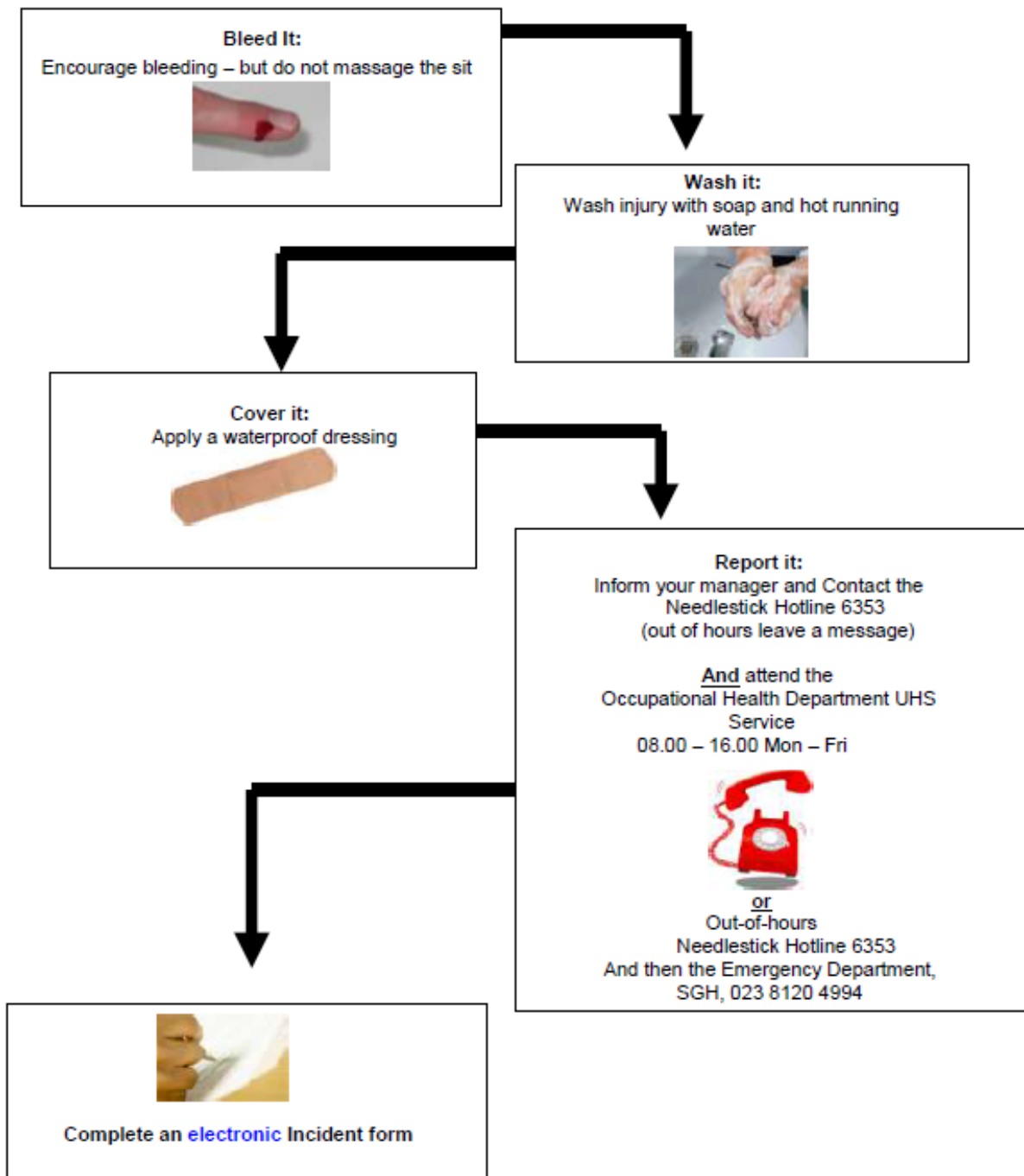
| Cardiac ICU   |                   |                         |           |                                    |                            |                  |
|---|-------------------|-------------------------|-----------|------------------------------------|----------------------------|------------------|
|   | Explanatory notes | Use / might need to use | Never use | Competent through use / experience | Had training (date / type) | Require training |
| <b>Ventilators</b>                                  |                   |                         |           |                                    |                            |                  |
| GE Carestation                                      |                   | Yes                     |           |                                    |                            |                  |
| Oxylog 1000   | Transport vent    | Yes                     |           |                                    |                            |                  |
| Oxylog 3000   | Transport vent    | Yes                     |           |                                    |                            |                  |
| <b>Syringe drivers</b>                              |                   |                         |           |                                    |                            |                  |
| Baxter Flo-guard                                    |                   | Yes                     |           |                                    |                            |                  |
| <b>Defibrillators</b>                               |                   |                         |           |                                    |                            |                  |
| Phillips Heartstart MRx                             |                   | Yes                     |           |                                    |                            |                  |
| <b>Monitoring</b>                                   |                   |                         |           |                                    |                            |                  |
| Vigilance II  | CO monitor        | Yes                     |           |                                    |                            |                  |
| LiDCO   | CO monitor        | Yes                     |           |                                    |                            |                  |
| Solar 8000  | GE                | Yes                     |           |                                    |                            |                  |
| Dash 3000   | GE                | Yes                     |           |                                    |                            |                  |
| Transport pro                                       | GE                | Yes                     |           |                                    |                            |                  |
| <b>Miscellaneous</b>                                |                   |                         |           |                                    |                            |                  |
| Clinical Information System                         |                   | Yes                     |           |                                    |                            |                  |
| Intra-aortic balloon pump                           |                   | Yes                     |           |                                    |                            |                  |
| PACE/ Osypka/ St Jude/ Cardio Logic Pace pacing box |                   | Yes                     |           |                                    |                            |                  |
| Fibrescope/stack                                    |                   | Yes                     |           |                                    |                            |                  |

# Sharps and Contamination Incidents

'Sharps Safety Policy' from Staffnet February 2016

## Flow Chart – If a sharps injury does occur

If a sharps injury does occur, the following action must be taken **IMMEDIATELY**:



More detailed guidance can be found in the following policies; Policy for the Management of Sharps and Contamination Incident

## Cardiac anaesthetic and ITU weekly teaching



Week commencing x/x/xxxx programme:

|           | Topic                                  | Time  | Location                       | Presenter | Cons Chair |
|-----------|--|-------|--------------------------------|-----------|------------|
| Monday    | M+M 1 <sup>st</sup><br>Monday of month | 07:30 | CICU seminar room              |           |            |
| Tuesday   | TOE teaching                           | 07:30 | CICU Seminar Room              |           |            |
| Wednesday | Cardiac anaesthetic/<br>ITU teaching   | 15:00 | CICU Seminar Room              |           |            |
| Friday    | Cardiac anaesthetic meeting            | 07:30 | Anaesthetic dept. seminar room |           |            |