Wessex Cardiac Intensive Care Unit

University Hospital Southampton NHS Foundation Trust

Anaesthetic trainee and clinical fellow handbook





3rd Edition 2023

Edited by Dr Paul Diprose & Dr Kirstin Wilkinson

University Hospital Southampton NHS
NHS Foundation Trust

Introduction

Welcome

Welcome to the cardiac intensive care unit we 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 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

April 2023

General Housekeeping

Authors: Drs Issa Ashhab & Paul Diprose *Revised: March 2023*

Previous authors: Drs Wilkinson & Diprose

Staff List

A large number of staff work on the intensive care unit. All consultant cardiac anaesthetists provide cover for the cardiac intensive care unit. Contact details for all consultants can be found in the flip folders on the nursing stations on each side of the unit.

Consultant Medical Staff	
Dr Mike Herbertson	Consultant Cardiac Anaesthetist
Dr Nick Goddard	Consultant Cardiac Anaesthetist & CICU Lead
Dr Jonathan Huber	Consultant Cardiac Anaesthetist
Dr Andy Curry	Consultant Cardiac Anaesthetist & Congenital anaesthesia lead
Professor Charles Deakin	Consultant Cardiac Anaesthetist
Dr Paul Diprose	Consultant Cardiac Anaesthetist
Dr David Hett	Consultant Cardiac Anaesthetist
Dr Steve Sandys	Consultant Cardiac Anaesthetist & Lead for cardiac anaesthesia
Dr Peter Wicks	Consultant Cardiac Anaesthetist
Dr James Montague	Consultant Cardiac Anaesthetist
Dr Crispin Weidmann	Consultant Cardiac and Neuro Anaesthetist
Dr Kirstin Wilkinson	Consultant Cardiac Anaesthetist
Dr Omar Al-Azzawi	Consultant Cardiac Anaesthetist
Dr Francois Wessels	Consultant Cardiac Anaesthetist
Dr Joshi Chandra	Locum consultant cardiac anaesthetist
Dr Sean Bennett	Locum consultant cardiac anaesthetist
Other Medical Staff	
Dr Issa Ashhab	Specialist Cardiac Anaesthetist
Dr Kausalya Raman	Associate Specialist
Dr Vivek Koul	Specialty Doctor
Dr Neda Bakalova	Specialty Doctor
Senior Nursing Team	
Michaela Jones	CICU Matron
Other Key Staff	
Julie Robinshaw	Ward Clerk
Laura Holden-Parker	Ward Clerk
Sally Barnes	CICU Pharmacist
Ryan Beacham	CICU Metavision support
Bethan Jenkins	Dietician
Dr Tatshing Yam	Consultant Microbiologist

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.

The HCAs and nursing teams can help you in setting up the equipment. However, you are expected to be able to prepare all the equipment needed for any specific procedure, including CVC and arterial line insertion. It's worth visiting the storeroom a few times and familiarising yourself with the location of different items. This will save you time and effort when you need to perform any procedure in the CICU. Always return equipment to its dedicated place.

TOE probes and machines need careful handling and can be damaged easily by dropping them to the floor, the transducers hitting hard objects, and contact of the plug with any fluid or moisture. TOE probes are very expensive. Always clean the probes and machines them according to the trust policy, complete the decontamination and tracing forms, and cover the TOE with the red plastic bag and return them to the endoscopy decontamination shelf between theatre 5 and 6 in E level.

Supervision and Tutorial Programme

All specialist anaesthetic and intensive care medicine 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 Peter Wicks (anaesthesia), Dr Francois Wessels (ICM) or the clinical lead for CICU. The responsibility for fellows on the CICU rests with Drs Huber and 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 Huber/ 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 30 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.

Resource Room

There is a trainee/clinical fellow resource room on the ‘blue side’ of the cardiac intensive care unit. It can be accessed via a keypad. 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 Goddard or Wilkinson.

Hospital Library

The Hospital Library is in A level in the south Academic block (SAB). You will find a diverse collection of resources and books in different specialties and dedicated spaces for reading and research and you can also apply for book loans

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 always act in a professional manner under the principles of the GMC guidance for good medical practice. http://www.gmc-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, your hair should be off your shoulders
- 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 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 always making clear and legible clinical records. Always login with your username to ensure all entries are assigned to you.

Pay particular attention to:

1. 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 under the appropriate sections.
2. Documenting all relative and family discussions and updates under the relatives' communication section. Make sure you note the names of all parties involved in the discussion and all the details discussed.

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.

ICU daily tasks

The patient care and progress in the CICU depends mainly on ward round plans and team discussions. All discussed and approved plans must be acted upon as soon as possible after they have been approved. This includes:

Prescribe/ change drugs and medication on Metavision as soon as it is discussed and approved.
Request all procedures needed on eQuest immediately after being approved.
All medical team's referral needs phone call discussions and should be done immediately after request.
All CT scans, whether urgent or routine, need to be approved by calling the radiology registrar.

All requests need to be submitted on eQuest, but some requests, including radiology, neurophysiology, electrophysiology and specialty team referrals need also to be discussed and approved by the appropriate clinicians.

Try to never leave plans unrequested, drugs unprescribed or procedures uncompleted before the end of your shift.

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.

Essential and Important apps useful for your stay in the UHS

CLWROTA: this is the UHS official rota management app. You need to contact the anaesthesia Admin office to have a login username and password first.

Induction: provides you with all the important phone and bleep numbers in the UHS and any other hospital in the UK.

MicroGuide: provides you with the infection control and critical care policies.

Logbooks: you need to keep records of all the cases you were involved in during your stay in the UHS. The RCOA specified data required when submitting a case in a logbook, the RCOA also provides access to the lifelong learning platform for a yearly paid subscription, you also can use other platforms or other validated software that use the same features and are recognised and accepted by the RCOA. Please contact Dr Huber for a copy of an electronic handbook that is suitable for clinical fellows.

MyESR: or my electronic staff record, you can access your payslips and P60 and pension details from this app. This requires first-time login username and password. Can be requested from the app itself

Healthroster Employee Online: is a desktop platform where you apply for locum jobs and submit your job claims

Important emails and bleep and phone numbers:

CICU consultant bleep: 2251
Anaesthetic office number: 6135
Medical HR number: 6525

Anaesthetic admin office: AdminAnaes@uhs.nhs.uk
Medical HR: MedicalHR@uhs.nhs.uk
Cardiac case manager: Christina.Bannister@uhs.nhs.uk (provide the case manager with your trust's email to have access to the daily surgical list)

Common tasks you might face on a CICU shift

Patient reviews, ward rounds and discharge: This is the everyday job for all patients. This needs thorough assessment of patients, review of all the blood results and ongoing issues, and previously requested jobs.

Referrals from and to other hospitals: Some patients need to be referred to treatment centres close to where they live if they are not in the catchment area to the UHS. This needs discussion with the referral centres and sometimes needs medical escort.

Fast track patients managed on CHDU need to be extubated by the CICU team. On a few occasions, some patients fail to meet the criteria for fast-track pathway and need to be transferred to CICU. This decision-making needs discussion with the consultant on call.

Drug prescriptions: There are 2 main drug prescription systems in the UHS. Metavision for CICU and other critical care areas, and JACs for the wards and high dependency areas. After patient discharge from CICU, all chronic and essential medication needs to be re-prescribed on JACs to ensure the continuity of care in the ward and other high dependency care areas.

Catheter lab anaesthetic support: Cath lab jobs can be slightly challenging sometimes due to the acuity of cases, out of hours jobs, and unfamiliarity of location. Always pay a visit to the catheter lab before attending the case if possible and arrange for an ODP cover (bleep 9266), good preparation prior to starting the case will help you tremendously.

CT patient transfers: Transferring patients to CT scanners needs good preparation, our critical care technicians will provide you with support and help in transferring intubated and ventilated patients to the CT scanners. Make sure to go through all the transfer checklists with the nursing team and CICU technicians. All CT scan patients need dedicated peripheral IV access for contrast imaging, CVC lines should not be used due to the risk of line fracture in a major vessel, or the heart associated with the high infusion pressure of the contrast machine.

Useful contact numbers

Author: Paul Diprose *Revised February 2023*

		Extension number (Outside calling: 023 81 20+ext)	**Bleep number** (Dial: 15+bleep+your ext)
Cardiac theatres	A	1986	
	B	1987	
	C	1988	
	3	2475	
	4	8949	
	Hybrid lab	5600 or 5601	
	Coffee room	4528	
	ICU technicians		2317
	Perfusion office	6930	
	Cardiac theatre nurses' office	4531	
	Cardiac theatre team		9217
Cardiac ICU	Blue side	6121 or 6122	
	Pink side	5080 or 4394	
	Coffee room	1240	
	CICU Consultant		2251
	CICU SpR		2310
	CICU Fellow		1660
	Cardiac surgery on-call		9211
	Cardiology on-call		2390
	Pharmacist		2140
	CICU nurse in charge		1183
	CICU bed coordinator		1491
Cardiac HDU	Main bay	6836	
	Beds 1-6	6835	
Anaesthetic Dept.	Admin team	6720 or 6135	
	Coffee room	3367	
Wards	E1 (Paediatrics)	6470 or 3267	
	E2	6473	
	E3 Blue	4111	
	E3 Green	6472	
	E4	6498	
	D4	8468	
	A&E Resuscitation room	4979 or 3807	
Others	Nurse case managers	8686 or 5333	
	Transfusion lab	4620	
	PACS office	4390	
	Metavision/CIS	4496	1794
	ICU Technician office	6890	
	Theatre coordinator		2897

Professionalism and Conduct

Dr Issa Ashhab & Paul Diprose *November 2022*

Appraisal and Revalidation

Every 5 years, you will need to revalidate your registration with the GMC. The GMC monitors your progress and professional conduct by examining the annual appraisal process undertaken at your workplace. Hence, you are expected to provide 5 appraisals in every revalidation cycle.

How do I do my appraisal?

For anaesthetic and ICM trainees, appraisals will be carried out as per the RCOA and FICM guidelines with your formal educational supervisor.
For clinical fellows, specialty doctors and post-CCT fellows, UHS has moved to an electronic, paperless appraisal platform called SARD (https://uhs.sardjv.co.uk). The website is very user-friendly and self-explanatory. It also provides live help chat service online. There are different sections and tasks you need to fulfil before submitting your appraisal form, you also will nominate an appraiser to review the evidence you provided and set a meeting with him or her (appraisal meeting). For clinical fellows, your educational supervisor will be your appraiser. For specialty doctors and post-CCT fellows, your educational supervisor must be a UHS approved appraiser. There are a number in the general department with Drs Wilkinson, Goddard and Hett available in the cardiac anaesthesia department. After successful review of evidence, the appraiser will approve your appraisal process for that year.

Some of the things you need to prepare for each appraisal:

Evidence of attendance of educational activities with reflection on the contents of these activities
Logbook of all the cases you were involved in that year
360 feedback - needed only once every revalidation cycle
Patient feedback forms. You need a minimum of 35 feedback forms in 5 years
Feedback from colleagues on teachings and educational activities
Signed off competency forms
Any complaints or complications
Declaration forms on health and probity

Professional Conduct

You are expected to adhere to all aspects of the General Medical Council's guidance on Good Medical Practise. This can be accessed through the GMC website and should be read carefully if you are not already fully familiar with its contents (https://www.gmc-uk.org/). Your conduct will be judged according to the standards set out in this document and the other documents related to it.

You should ensure that you enrol with a Medical Defence Organisation (MDO) whilst practising in the UK. They can provide excellent advice should you encounter complaints against you whilst working in the UK, and if necessary, can provide you with legal assistance in the event of fitness to practise issues. Currently, there are 3 main organisations in the UK that provide these services. (MDU, MPS, MDDUS). You should register with one of them at the first available opportunity; they will not provide legal cover retrospectively.

Professional misconduct is taken very seriously at UHS and in the UK generally. Any misconduct can put your career at risk and potentially affect your ability to work as a doctor for a long time.

Common examples of professional misconduct:

Dishonest or inaccurate note keeping
False information in financial status and other Jobs
Inaccurate claims for overtime or locum payments
Non-disclosure of previous work sanctions
Breaches of professional and personal boundaries with colleagues or patients
Inaccurate declarations of previous experience, jobs, and medical qualifications

Where possible, and depending on the seriousness of the allegations, all professional misconduct investigations are aimed to be resolved internally at the UHS. Some will be referred to the GMC and the GMC will decide the actions needed to resolve the issues. In more serious cases, the GMC may also decide to refer the cases to the Medical Practitioner Tribunal Service (MPTS). The MPTS is responsible for deciding on the fitness to practise of doctors in the UK. If a doctor is found to have an impairment of their fitness to practise for any reason, they have a variety of powers to restrict, or (in the most extreme cases) to remove, a doctor's right to practise.

General Patient Management

Admission and reviews on CICU

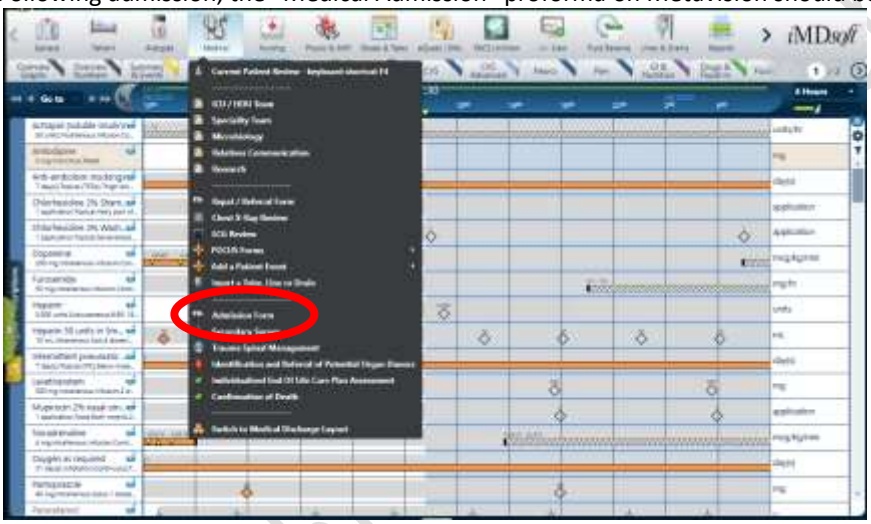
Author: Dr Alisha Allana *November 2022*

Previous author: Dr Wilkinson

Most patients will be admitted to CICU directly from cardiac theatres. However, patients may also be admitted from the cardiac high dependency unit (CHDU), the emergency department, cardiac wards, the catheter labs, or transferred from other local hospitals. We may also be asked to admit occasional patients from GICU (but only following agreement with the duty CICU consultant.
A verbal handover should be taken by the patient's bedside together with the nurse looking after the patient, and the nurse in charge if available.

Admission to CICU

Following admission, the "Medical Admission" proforma on Metavision should be completed.



The admission form should clearly state the reason for admission to CICU, relevant past medical history and any known limitations of care for the patient.

Age and gender of the patient

Presenting complaint:	Operation performed (if applicable) – the operation note can be found on CHARTS for exact details to be documented onto Metavision.
History of presenting complaint:	Brief summary of the reasons for the operation Pre-operative investigation results include TTE and coronary angiogram (specifically LV function, valves and coronary arteries affected) Pre-operative pulmonary/renal function Pre-operative medication including anticoagulants and if/when they were stopped.

Operation details:

- Surgical complications
- Cardiopulmonary bypass (CPB) time and cross-clamp (XC) time
- Grade of intubation
- Inotrope and vasopressor support pre/post CPB
- Balloon pump pre/post CPB
- Blood products administered
- TEG/clotting results
- Pacing and perioperative shocks delivered

Past medical history, treatment and allergies
Smoking and alcohol history
Function and mobility pre-operatively
Next of kin/family

If there are any queries, please discuss these with the surgical/anaesthetic team that cared for the patient in theatre in the first instance. If they are not available, please discuss any concerns with the cardiac anaesthetic consultant on CICU or the cardiothoracic registrar on call (bleep 9211).
Following admission to CICU a full clinical examination should be carried out and documented on Metavision. This should focus on:

- Ventilatory settings and arterial blood gas trends
- Drainage from the chest (if post-surgical)
- Urine output
- Inotrope and vasopressor support
- Sedation
- Lines (patency and location)
- Pacing (mode, underlying rhythm if present)

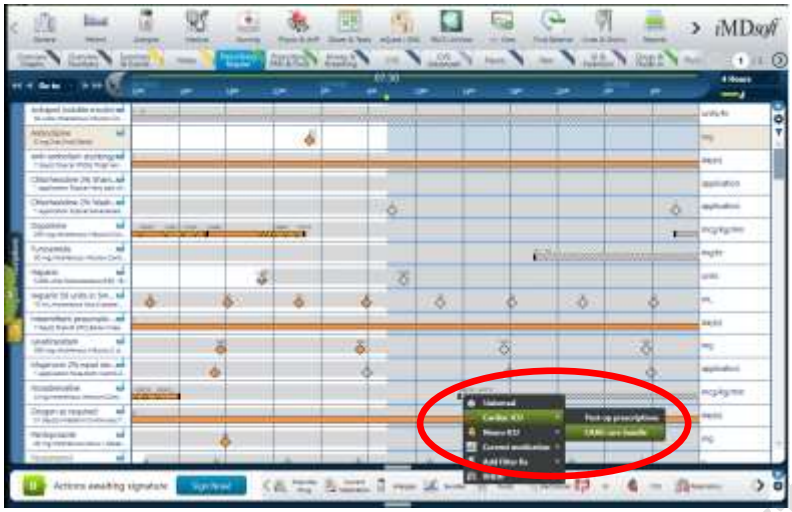
Clinical plan

The plan is normally handed over by the anaesthetist that has transferred the patient from theatre. This should include:

- Physiological aims – MAP, SPB, CVP, Hb target and urine output
- Warm, wean wake and extubate if there have been no perioperative complications
- Bloods/other investigations required e.g. clotting, TEG
- A chest x-ray is not routinely indicated, but is required in certain scenarios e.g. in the presence of a pulmonary artery catheter or intra-aortic balloon pump

Prescribing

Following admission to CICU patients should be prescribed postoperative medication (this can also be done by the theatre anaesthetist). On Metavision, select the bundle for "Cardiac ICU", and this should include the necessary prescriptions. Please review these based on the needs of individual patients. Remember specifically that postoperative patients will need a further dose of antibiotics, and the relevant anti-platelet agents, as well as VTE prophylaxis when appropriate.



Daily review

The daily reviews are documented on the review forms on Metavision accessed either by pressing F4 or by selecting the form from the Toolbar. Remember to click "new" in the top right corner of the form, or you will override the previously documented review!

The daily review should be thorough but concise. Use the free text boxes in the form to clearly document any important findings.

The review should include:

- Age of patient
- Number of days on CICU
- Reason for admission (in 1 or 2 sentences)
- Main issues – as a simple list e.g. renal failure, acute liver injury, low CO state
- Past medical history

Every patient should be examined daily:

Airway/breathing	Airway – if intubated, size of ETT/tracheostomy Ventilation mode, tidal volume, respiratory rate, FiO_2 (trends), peak/mean airway pressures Arterial blood gas results Chest signs, presence of sputum, trachea
Circulation	Pulse – rate, rhythm (underlying), pacing mode and thresholds Blood pressure – MAP, SPB, CVP and vasopressor support (doses/trends) Heart sounds and presence of oedema Inotropes (doses/trends) and IABP settings if present Cardiac output studies if using PAC or LiDCO Chest drainage
Disability	Sedation, GCS/RASS (or CAM ICU) score, neurology and blood glucose Presence of seizures
Gastrointestinal	Abdominal examination and bowel sounds Method of feeding, whether absorbing and bowel movements Ulcer prophylaxis (IV or PO PPI) Liver function tests
Renal	Urine output and fluid balance (total and 24h)

Use of furosemide boluses/infusion
Investigations – urea, creatinine, Na, K
Renal replacement therapy

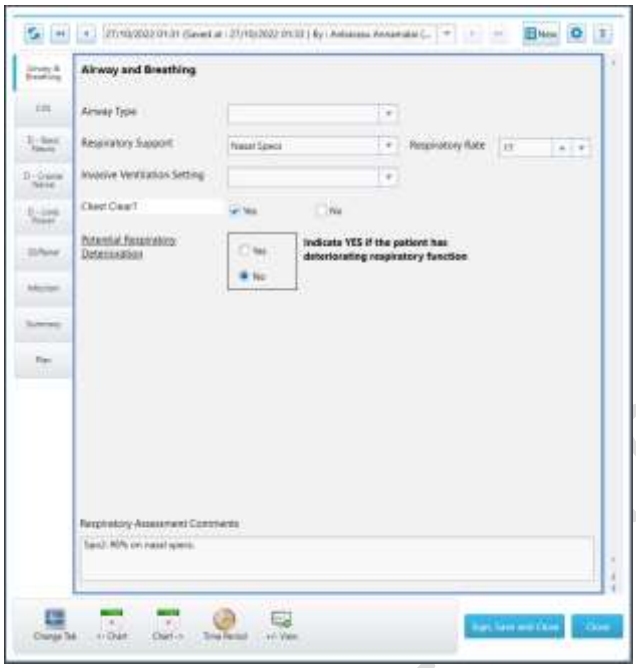
Haematology

Hb, clotting and anticoagulation.
The plan for anticoagulation should be confirmed with the surgical team. This may include:

- Aspirin – post-CABG
- Warfarin/DOAC – post-metallic valve surgery
- Subcutaneous heparin for postoperative VTE prophylaxis

Micro

Temperature
Culture results if appropriate
Current antibiotics
Duration of invasive lines/catheters



Any recent investigations should be followed up and documented onto Metavision e.g. chest x-rays, blood cultures or CT scans.
Enter a brief impression of ongoing patient issues and formulate a plan on the last tab of the form. This can be updated directly by subsequent members of the team.

Ward round

The daily consultant ward round should be documented as a clinical note on Metavision.
The outcome of the microbiology ward round should also be entered as a note (specified as "Microbiology").
All entries and discussions with specialist teams should be documented and should include the name and contact number of the individuals who were present.

Night reviews

The night review can be entered either as an F4 form, or as a free text entry on Metavision. This should include any pertinent issues, basic systems review and any discussions that take place with family or seniors overnight.

Infection Issues

Authors: Sr Melanie Griffiths *Revised: October 2022*

Previous authors: Dr Richardson and Dr Wilkinson

Important Contacts

Microbiologist: Dr Tat Yam
Lead Infection Prevention Link Sister, CICU: Melanie Griffiths (bleep 1086)

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

Hand Hygiene

The transfer of micro-organisms between humans can occur directly via hands, or indirectly via an environmental source. Performing hand hygiene is widely accepted as a key strategy of infection prevention and control to prevent Healthcare associated infection (HCAI), as healthcare workers' contaminated hands are the vehicle most often implicated in the cross-transmission of pathogens in health care.

Hands must be decontaminated at critical times before, during and after patient care activity to prevent cross-transmission of micro-organisms. Hands must be decontaminated:

- Immediately before each episode of direct patient contact/care, including clean/aseptic procedures
- Immediately after each episode of direct patient contact/care

- Immediately after contact with body fluids, mucous membranes and non-intact skin
- Immediately after other activities or contact with objects and equipment in the immediate patient environment that may result in hands becoming contaminated
- Immediately after the removal of gloves.

(Epic 3, WHO)

There are some taps and sinks within CICU that have been identified as being colonised with pseudomonas or legionella. All are treated and many have filters in situ. To minimise risk of contact transmission after hand washing, please also decontaminate hands with alcohol gel.

Personal Protective Equipment

- Disposable plastic aprons must be worn when close contact with the patient, materials or equipment is anticipated and there is risk that skin or clothing of the healthcare worker may become contaminated with pathogenic organisms, blood, body fluids, secretions and excretions.
- Gloves and aprons are available in each bed space.
- Full sleeved gowns are to be worn for a risk of splash/ contamination of your uniform and when directed by the infection prevention team.
- Full facial visors should be worn for any risk of splash to the face, when with Covid 19 contact and Covid 19 positive patients, and when wearing a valved FFP3 mask.
- A surgical face mask should be worn for droplet precautions and when advised by the Trust dependent upon local infection rates; and a FFP3 mask for aerosol generating procedures (AGPs – see appendix), including for all suspected or known respiratory viruses (including Covid 19) and TB. You must be fit tested for a FFP3 mask when you arrive and every two years. A member of the senior nursing team will be able to assist you with this. An appointment is also bookable via VLE.
- No valved masks are to be worn when carrying out sterile procedures e.g. central venous access device insertions due to the risk of condensation droplets from the HCP landing on the patient via the valve (NPSA alert 2021).
- All PPE (except for a mask as determined by Trust policy) must be removed before leaving the bed space.

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 Staffnet, 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) to facilitate hand hygiene.
- Follow the uniform policy including hair tied above the shoulders, no jewellery or watches, no lanyards during clinical care.
- 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 of this manual).
- All personal stethoscopes are to be cleaned between patients with either a green Clinell universal wipe or Sochlor if the patient is in isolation. There are unit stethoscopes available in each bedspace.
- All reusable equipment must be cleaned before and after patient use with a green Clinell universal wipe or Sochlor if in isolation. Clinell yellow detergent wipes can be used to clean screens. Clinell red sporicidal wipes will be used for commodes and bedpans only. Transoesophageal echocardiogram equipment has a separate policy for decontamination (See details on TOE room and on the machines). The machine itself will be cleaned with Tristel Solo wipes and Lyrecol screen cleaner. A green 'I am clean' sticker or tape label should be used to identify when the item has been cleaned. All products are readily available on CICU.
- There are different waste streams bags for waste, please use the appropriate one:

Black bag – domestic waste (no clinical waste)
Orange bag – clinical infectious waste
Tiger stripe (yellow and black) bag – clinical, not infectious waste
Recycling – mixed
Grey bags – confidential waste

MRSA Screening and Risk Reduction Measures

Almost all adult admissions to UHS are screened for MRSA and receive daily whole-body washes and hair shampoo with 4% chlorhexidine gluconate (Hibiscrub®) or 2% chlorhexidine wash cloths and shower cap 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®). If Bactroban is not available, Naseptin is used for 10 days (NB contains chlorhexidine).
- The treatment must be prescribed on the drug chart on Metavision
- If the patient is sensitive to chlorhexidine, Octenisan washes can be prescribed.

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 will be screened for carriage on a weekly basis, on all adult ICUs this is done every Sunday night/Monday morning. 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 4% (Hibiscrub) only 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. 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® or Tegaderm CHG, chlorhexidine-impregnated dressings are generally considered for use on all central venous access device insertion sites in patients in ICU, including all patients who have MRSA.

Carbapenem Producing Enterbacteriaceae Screening

Almost all adult admissions to adult ICUs are screened for carbapenem producing enterbacteriaceae (CPE) on admission and weekly, alongside the MRSA screening. This is usually a rectal swab. In addition, all patients will also have additional screening 48 hours after commencing any carbapenamase antibiotics e.g. meropenem or ertapenem. You should verify that this has been done and be aware of the result, when available. All patients who have been admitted from another ICU or have been an inpatient in another (not local) hospital within the last 12 months will be isolated until they have had 2 clear CPE screens 48 hours apart.

Covid 19

Guidance is frequently changing.
The most up-to-date information is available on Staffnet – please refer to this information or check with the infection prevention nurse.
All staff displaying any respiratory symptoms (no matter how mild) are to take a lateral flow test and follow Trust guidance if positive (available on Staffnet or speak to your line manager).

Infection Prevention Audits

CICU partakes in the UHS infection prevention audit programme. Observations of practice, surveillance and auditing is continuous on the unit. They include audits on high impact interventions such as VAP, surgical site infection, insertions and ongoing care of central venous catheters, peripheral venous cannulas and urinary catheters, and cleaning and decontamination.

All local audit results are available on both Staffnet and emailed monthly via the critical care infection prevention newsletter.

Central venous access device insertions

Auditable elements for insertion include hand hygiene, aseptic non touch technique, wearing a head covering, face mask and eye protection, sterile gown and gloves; skin decontamination using 2% chlorhexidine with 7% alcohol (typically Chloraprep 3ml, tinted and non-tinted applications are available, transparent dressing (this is often Tegaderm CHG for ICU), disposal of sharps, and documentation of the insertion on the dedicated sticker enclosed in the insertion packs or on Metavision. Catheter associated device infection (CADI) scores will be documented by nursing staff 8 hourly. All lines must be reviewed and documented daily for continued use.

Peripheral cannula insertions

Auditable elements for insertions include hand hygiene, aseptic non touch technique, skin decontamination (typically Choraprep 1ml), transparent dressing, and documentation on dedicated form or Metavision (one for each cannula). Visual inspection phlebitis (VIP) score will be documented by nursing staff 8 hourly. All lines must be reviewed and documented daily for continued use. Less than 3 days (maximum 5 days) is the typical length of time a cannula is to remain in situ in UHS.

Surgical site infection

Auditable elements include optimising temperatures pre-op, shaving hair where required using surgical clippers, a chlorhexidine wash pre-op and post op for 2 days, initial interactive dressing is to be undisturbed for a minimum of 48 hours after surgery (24-48hrs for caesarean section wounds) unless there is leakage from the dressing and need for a change, hand hygiene.

Urinary Catheters

ANTT for all insertions and documentation on Metavision. Daily review for requirement and prompt removal if no longer required or patient anuric.

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 consequently.

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, several 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

- **Oral hygiene:** Teeth are brushed 12-hourly with standard toothpaste and documented or regular mouth care to be carried out if no teeth present and documented.
- 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.
- **Tracheal tube cuff pressure:** Cuff pressure is measured a minimum of 4 hourly, maintained between 20-30cm H2O (or 2cm H2O above peak inspiratory pressure) and recorded on ICU chart/ Metavision. This can be monitored continuously via the *intellicuff* on the Hamilton ventilator or 4 hourly manually with a cuff manometer.

Minimising the Duration of Mechanical Ventilation

- **Assess for extubation:** Assess for weaning and extubation at least daily (unless contraindicated) and document.
- **Daily sedation hold** - accumulation of infusions delay weaning, increase complications and thus sedation is stopped until it is deemed necessary to recommence for ventilation & safety issues.
- **Sedation level assessment:** Aim for RASS (Richmond agitation sedation score) of 0 to -1 unless clinically indicated and documented.
- 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.

Ultrasound Gel

Only sterile gel can be used in all high care and intensive care settings as per PHE due to outbreaks of Burkholderia associated with bottles of non-sterile gel.
Ref: CA-2022-001.pdf

Catheter-related or Catheter-associated Blood Stream Infections (CRBSI or CABSI)

CRBSIs and CABSIs 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 chlorhexidine, 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 PaCO2 < 32 mmHg (4.27 kPa)
- WBC > 12 x 10^6 cells/mL or < 2 x 10^6 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 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

- All patients prescribed a carbapenamase antimicrobial e.g. meropenem, ertapenem, will require carbapenem screening (usually a rectal swab) 48 hours after the commencement of the antibiotic. You should verify that this has been done and be aware of the result, when available.

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 (typically 1ml Chloraprep applicator)
- **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 (typically a wipe), 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
- Link for ANTT action card on Staffnet for **taking peripheral blood cultures**: http://Staffnet/Media/DepartmentsAndCareGroups/DepartmentOfInfection/InfectionControl/ANTT/Proceduralcards/ANTT-blood-culture-collection-v2.pdf
- Alternative methods for **taking blood culture samples from a CVAD** with or without a transducer is available on Staffnet (links: http://Staffnet/Media/DepartmentsAndCareGroups/DepartmentOfInfection/InfectionControl/ANTT/Proceduralcards/ANTT-blood-culture-sampling-from-CVAD-**with-transducer**-action-card.pdf and http://Staffnet/Media/DepartmentsAndCareGroups/DepartmentOfInfection/InfectionControl/ANTT/Proceduralcards/ANTT-blood-culture-sampling-from-CVAD-**without-transducer-**action-card.pdf

Stool Sampling

If a patient has an unexpected type 5, 6 or 7 stool, it is the Trust policy to have a review and the Good Practice Guide (diarrhoea proforma) to be completed. A medical review must be carried out and documented within 8 hours of the onset of symptoms. If deemed potentially infectious, the patient is to be isolated within 2 hours and a specimen sent for appropriate testing.

The Good Practice Guide can be found on Metavision under the Nursing tab along the top row.

Mandatory Daily Review

As part of the Saving Lives High Impact Interventions, it is necessary to review and document whether a patient still requires invasive devices such a urinary catheters, CVCs and peripheral cannulas, and sedation and antibiotics have also be reviewed.
On Metavision within the Medical Icon under 'current patient review' and the 'plan' tab you will find a simple checklist to complete rather than write this out.

Appendix – Aerosolised Generating Procedures (AGPs)

The current National infection prevention and control manual for England (April 2022) has amended its guidance on what is considered an AGP. UHS has now updated its' guidance to match this (October 2022):

UPDATED PPE IPC guidance for AGPs v4.0 10th October 2022.pdf

Bleeding & Coagulopathy

Author: Dr Kirstin Wilkinson *Revised: January 2023*

Previous authors: Dr Tom Pierce & Dr Kirstin Wilkinson

Every patient after CPB develops a coagulopathy, which for the most part, is sub-clinical. Very often, the patients will have abnormal coagulation and you will see and manage 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 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 of bleeding and coagulopathy

Patient

- Pre-existing coagulation abnormalities INR and APTR
- Thrombocytopaenia
- Anti-platelet therapy including aspirin, clopidogrel, dipyridamole, prasugrel, ticagrelor
- LMWH, warfarin, NOACS (rivaroxaban, apixaban, edoxaban, dabigatran),
- 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
- Thrombocytopaenia (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 - repeat ACT – a further dose of protamine may stop the bleeding
  - INR, APTR, platelets, fibrinogen
  - Hb
  - Thrombelastogram: plain and heparinase
- Crossmatch 4 units, if large blood loss, consider 6 units and think about clotting products
- Inform consultant anaesthetist/ intensivist early

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

A recent ACTACC national audit found the resternotomy rate for bleeding or cardiac tamponade to be 0.69-7.6%, overall rate 3.6% with a mortality rate (95%CI) of 15.0% (12.7–17.5). In patients who underwent resternotomy, the median (IQR [range]) length of stay on ICU was 5 (2–10 [0–335]) days, and time to tracheal extubation was 20 (12–48 [0–2880]) hours. A total of 89.3% of patients who underwent resternotomy were transfused red cells, with a median (IQR [range]) of 4 (2–7 [1–1144]) units of red blood cells. The rate (95%CI) of needing renal replacement therapy was 23.4% (20.6–26.5). This UK-wide audit has demonstrated that resternotomy after cardiac surgery is associated with prolonged intensive care stay, high rates of blood transfusion, renal replacement therapy and very high mortality. (1)

The surgeon should contact the theatre team (including the perfusionist). Consider whether safe to return to theatre or whether patient needs to have chest opened in CTITU.
In a number no surgical cause is found. Most patients 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.

References

S. Agarwal, S. W. Choi, S. N. Fletcher, A. A. Klein, R. Gill, Contributors The incidence and effect of resternotomy following cardiac surgery on morbidity and mortality: a 1-year national audit on behalf of the Association of Cardiothoracic Anaesthesia and Critical Care. Anaesthesia 76(1) 2021 19-26.

Epicardial Pacing

Authors: Dr Mike Purkiss, *Revised October 2022*
Previous authors: Drs Pierce & Wilkinson

This is required for the intra- and post-operative management of patients undergoing cardiac surgery. Although most 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%.
Those with a greater need to have a backup pacing system around the time of surgery are patients with pre-existing conduction disease such as bundle branch block or profound 1st heart block. The type of surgery is also relevant as the His Purkinje system runs past the non-coronary cusp. Therefore aortic valve replacement, mitral valve surgery and infective endocarditis specifically effecting the aortic root all have a higher chance of heart block. TAVI has also been shown to carry a higher risk of heart block post procedure.

External Pacing Box

At Southampton, we currently use 2 types of external pacing box. The Osypka and St. Jude boxes which are identical in function, just differing in colour and the Cardio Logic Pace T20 Box.
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.

External pacing wires:
Pacing wires are lightly sutured to the surface of the myocardium. There are 2 wires attached to the RA and 2 attached to the RV. They then penetrate the chest wall and are secured to the skin before running off to the pacing box.
By convention in Southampton, **white** wires are connected to the **atria**. These insert into the 2 ports in the left side of the pacing box and the **blue** wires are connected to the **ventricles**, these ports are located on the right side of the pacing box. **YOU MUST CHECK YOU HAVE INSERTED THE WIRES CORRECTLY INTO THE PACING BOX.** The ports are marked + and – and which atrial wire is in which port is of no consequence; this is the same for the ventricular leads.

On the front of the Osypka and St. Jude 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. If not pressed, the box will lock after a short period of time.

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 to optimise cardiac output. In some cases, DDD may not be appropriate e.g. in chronic atrial fibrillation where VVI or DVI may be chosen instead.

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.

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 (pacemakers have an internal battery to allow continued pacing while the external battery is safely changed. You do not need to panic while changing the pacemaker battery on a pacing dependant patient)
- 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 to assess the underlying rhythm, as follows:

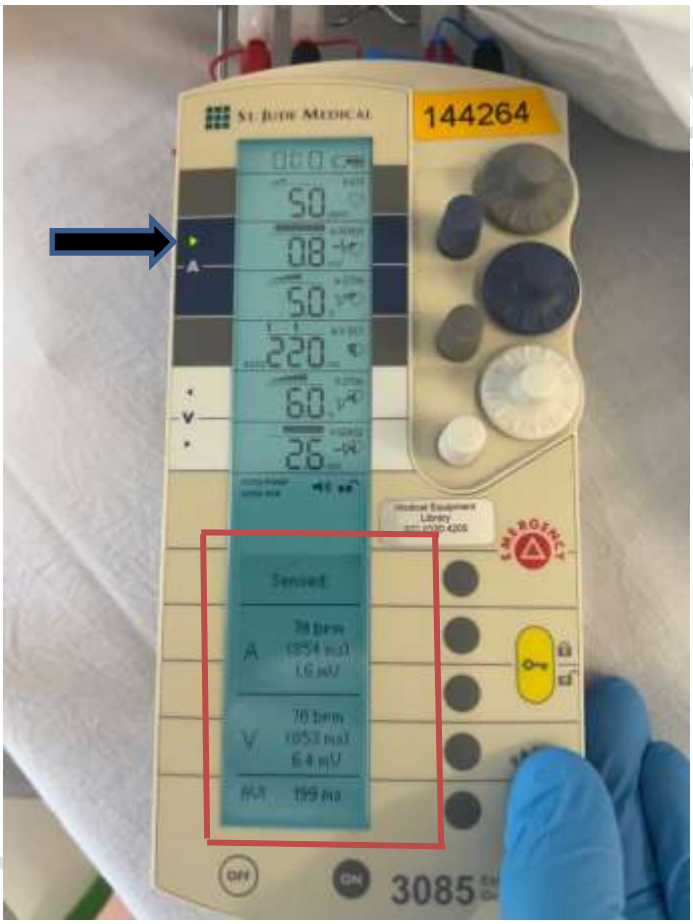
On the St Jude/Osypka Box Press the PAUSE key until pacing is inhibited. Assess the rhythm. To restore pacing, simply release the PAUSE key. This will result in return to the normal status display. Holding the pause key on these particular devices is particularly useful as the box performs a small "EP" Study showing you the parameters the box has sensed.
This can then be used to set up the pacing box more effectively.

The Green light indicates it is sensing input from the atria at the time the photo was taken.

The bottom of the picture shows what is being sensed. In this case 70bpm from the atria and an A wave of 1.6mV.

It also shows the ventricles are beating at 70bpm indicating probable sinus rhythm with a V wave of 6.4mV.

The AV interval (AVI) is 199ms showing the time delay between sensing the atrial signal and then the ventricular signal.



Unusual Readings from the pacing box:
Atrial Fibrillation present: atria light flashes quickly and randomly and the atria sensing screen comes up with random high numbers it indicates the atria are fibrillating.
Atrial Flutter present: atria light flashes fast and regular with normal AV delay.
Junctional Rhythm present: ventricular light seems to light before the atrial with a very short time period between the two flashing.

If no significant underlying rhythm is present, then pressing the pause button will result in a blank screen and loss of cardiac output. This will continue as long as the button is pressed or for 10 seconds whichever is sooner.

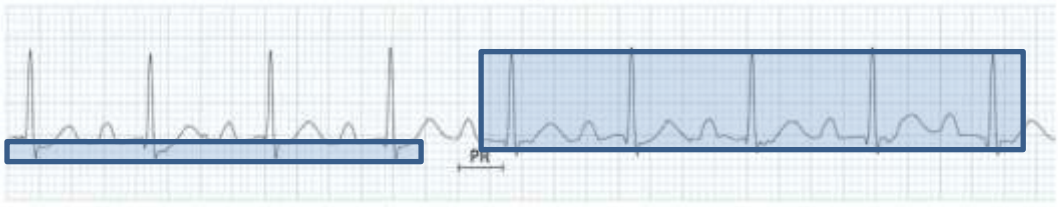
Sensitivity

This is the minimum current that the pacemaker can sense – a lower number thus corresponds to a greater sensitivity.
It is like a wall and the pacemaker can only see what is above the wall.

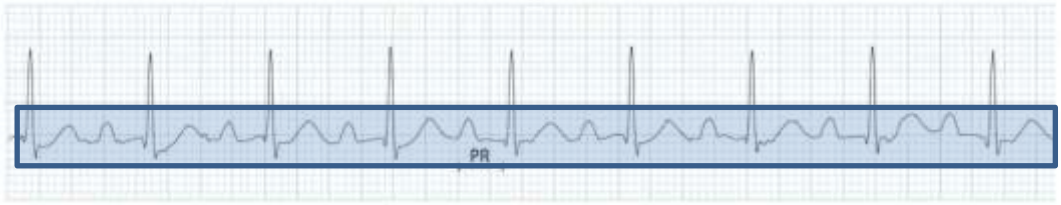
If set too low, the pacemaker will pick up background noise as well as cardiac activity. This will cause the pacemaker to think there is allot of intrinsic electrical activity and can cause the pacemaker to pause.
If set too high the pacemaker will not be able to see the electrical activity of the heart and think there is no underlying rhythm. This will cause the pacemaker to pace over the top of the patients own rhythm and can cause an R on T phenomenon.

Sensitivity set too Low. Pacemaker sees too much electrical activity.

Sensitivity Set too High. Pacemaker unable to see the underlying rhythm



Pacemaker set correctly and sees the spike of the QRS Clearly but screens out unwanted activity.



Setting and identifying the correct sensitivity in other pacing boxes providing there is adequate underlying rhythm:

Atria: Set the pacing box to AAI

1. Turn down the pacemaker rate and sensitivity so the patient's own rhythm is present.
2. The box should now display it is sensing. It will show flashing green arrows pointing towards the pacing box one flash for the atria and one for the ventricles.
3. Slowly turning the atrial sensitivity value up will now screen out background noise and then the atrial signal. The pacemaker will then start to pace the atria as it no longer detects the underlying rhythm.
4. The sensitivity should then be turned down until atrial pacing sensing is occurring all the time. The sensing value shown will indicate the size of the P-Wave coming from the atria is just larger than the value sensed. This is the sensing threshold.
5. To ensure the atria is always effectively sensed, the sensing value should be turned down to roughly half the sensing threshold but ideally not to less than 1mV.

Ventricles: Set the Pacing box to VVI

1. Turn down the pacemaker rate and sensitivity until the patient's own rhythm is present

2. Follow the steps for the atria sensing as above.

If there is no endogenous rhythm this cannot be done. In this situation the sensitivity is typically set to 2mV for the Ventricles and 1mV for the Atria.

Capture threshold

This is the minimum pacemaker output required to stimulate an action potential in the myocardium.
If there is no underlying rhythm, please ask a senior to guide you through the process of testing the threshold. If no underlying rhythm is present, we usually keep a spare pacing box by the bed and test the threshold twice a day to ensure the threshold is not rising to dangerous levels without our knowledge.

The best way to check rhythm and threshold when the pacemaker is pacing dual chamber:

1. Turn the pacemaker setting to AAI.
2. If the heart rate stays the same, then the patient has atrio- ventricular conduction. This immediately rules out complete heart block and means significant damage to the conducting system is unlikely.
3. If the rate drops, then there is conduction delay between the two chambers. Depending on what the drop is tells you the issue.
    a. This could be occasional missed beats with lengthening PR = Wenckebach.
    b. Half the rate of the atria = 2:1 Heart block.
    c. The ventricles own rhythm independent from atrial activity = complete heart block
4. If the rate stays the same: slowly turn down the atrial rate until the underlying rhythm appears. If you get to 30 and no intrinsic rhythm appears then do not go any further. Turn the rate back up to 90 to allow cardiac output to recover.

Now we have identified the underlying rhythm we can check the threshold:

Checking atrial threshold when there is underlying rhythm:

1. Turn the pacemaker to AAI.
2. Ensure the pacing rate is above the patient's own rate by 10 beats/minute.
3. Slowly turn down the threshold until the heat rate drops. This represents loss of capture and is usually a lower figure than when the pacing spikes disappear on the monitor. If you use when the pacing spikes disappear, you're actually checking how good the monitor is at picking up the pacing signal…. Interesting but not very useful!
4. Turn the output back up until you recapture the myocardium, and the rate increases back to your set pacing rate. This is the capture threshold, and it may be higher than when you lost capture.
5. Setting the capture threshold with a safety margin: Continue to increase the output to either 3x the capture threshold or add 5V to the threshold (Whichever is greater). If the threshold is high so it cannot be tripled, then simply add 5V.

The safety margin is used as the wires can become micro dislodged by blood clot from the myocardium and if pacing for a long period fibrosis can set in where the electrical stimuli occurs both of which will cause the threshold to rise. 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.

Checking the Ventricular threshold

This can be done in DDD or VVI:
VVI is simply the same as the AAI check except it will often cause the cardiac output to drop slightly if the patient is in sinus rhythm. DDD will not tend to do this but you have to pay more attention.

1. Set the DDD rate at least 10 beats above the patients underlying rhythm or just leave at 90.
2. Slowly turn down the ventricular stimuli until you see a change in PR interval and/or a change in morphology of the QRS complex.
3. As pacing the Right ventricle causes the QRS complex to appear as LBBB the point of loss of capture usually is when the QRS becomes narrow. This may be challenging if the patient has bundle branch

block as their usual rhythm. In that case you are usually looking for a subtle change in morphology and PR interval. If you find it difficult to see then check using the VVI method or ask a senior.

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

= Dual Chamber Paced, Dual Chamber Sensed. If faster underlying rhythm sensed, then don't pace.

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 ventricular depolarisation. If it senses one, then it will not pace but simply watch the ventricles. If no ventricular depolarisation is seen, then it will deliver a pacing spike.
A problem with this type of pacing is that having the wires sewn on the outside of the heart increases the time it takes for the pacemaker to see ventricular activity. This means it often paces the ventricles despite normal AV conduction as it simply does not see the activity in time.
A further problem is when the patient goes into atrial flutter or atrial tachycardia. As the pacemaker can sense the atrial activity it can then try to deliver pacing spikes to match the atrial rate. To get around this issue pacemakers have an upper tracking rate so will not deliver high atrial rates but max out at the set upper tracking rate – often 130bpm.
If atrial arrhythmia is an issue such as AF, then DVI is a better mode as it will not sense the atria and not try to track it.

Indications: All indications for pacing, except for atrial tachyarrhythmia.

AAI

Used in patients with an intact and reliable atrio-ventricular conducting system. Safe for post CABG surgery where you don't expect post op swelling around the electrical conducting system to cause heart block within the next 24 hours.

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 CICU 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 if no underlying rhythm has returned. If there is an underlying rhythm but intermittent pausing or heart block, then quite often the EP team will conduct a 24-hour tape at day 10. If the conduction system has not recovered by then it is unlikely to. Further delay in placement of a permanent system would be ongoing infection or raised inflammatory marker as new metallic implants are prone to infection if implanted in someone with bacteraemia.

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

Pacing Box Tips

- **Lights on the pacing box**
  - Green light (arrow in) indicates the box is sensing the heart.
  - Yellow light (arrow out) indicates the box is sending a pacing spike to the heart.
- **Lots of pacing spikes on the patients monitor**
  - Look at the pacing box lights to see if they correspond… They don't tend to and its usually interference being picked up.
  - Check the leads and stickers on the patient's chest – ensure everything is well connected and there is good contact with the patient. Then check the ECG on the monitor.
  - At this point you can change the ECG lead the monitor is looking at and pick an alternative such as lead I, II or III.
  - Next look at Advanced settings and check what filter has been applied and change it.
  - I always make the ECG bigger if it's hardly showing on the monitor at this point as it makes assessing the rhythm and QRS that much easier.
- **Rising thresholds**
  - Always check the connectors to the pacing box to ensure they are adequately attached. Simply swapping the wires over from one port to another can bring about a reduction in threshold as electricity is now being sent to a different part of the myocardium.
- **Capture threshold**
  - This made up of a combination of Voltage delivered to the myocardium and duration of stimulation. Higher voltage is required if the myocardium is only stimulated for a very short period of time.
  - A lower voltage may be able to adequately stimulate the myocardium if the duration of the "pulse" is longer.
    - Default setting for pulse duration is 0.75ms.
    - This can be altered on the Osypka and St Jude boxes up to a value of 1.5ms which often lowers the voltage required to capture the myocardium.
    - It is altered by going to MAIN MENU at the very bottom of the screen.
      - Then PARAMETERS & OPTIONS also near the bottom of the screen.
      - Then PULSE DURATION.
      - You will then see the option for increasing or decreasing the pulse duration for the atria and ventricles.
      - Increase the duration to 1.5ms and recheck the threshold. You should find it to be lower by several Volts.
- **Ectopics**
  - These can be a real issue when pacing and is usually the result of irritable myocardium or scar.
  - Simple things are to ensure the magnesium is well replaced and usually greater than 1.
  - Ectopics will also be suppressed when the K is >5 but replacing it to this level can make staff nervous (it should not be an issue with normal renal function however).
  - Ideally, they may settle with time but if not B-blockers work well.
  - If bisoprolol cannot be given due to inotropes, then turning the pacing rate down.
  - AAI pacing or pacing slightly faster may suppress the ectopic beats.

Pain, agitation, sedation & delirium on CICU

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This chapter deals with the typical sedation strategies, pain management, and approach to post-op delirium on CICU.

The uncomplicated patient

All patients admitted to CICU from cardiac theatres arrive sedated and ventilated. Sedation is usually achieved by a propofol infusion at a rate of about 4mg/kg/hr (20-40 ml/hr). If there are no indications for keeping the patient sedated for a longer period, sedation is quickly weaned off once the patient is stable, warm and not bleeding - typically within 4-8 hours. The nurse looking after the patient might check with you before they start weaning off the sedation.

For pain relief during this period patients are given intravenous morphine in boluses of 1-2 mg up to a total dose of 16mg. (This is the standard CICU prescription for 20mg/20ml morphine, however 4mls are used to prime the line, thus the maximum dose that can be given is in effect 16mg). It is rare that all 16 mg are used and the nurse looking after the patient will let you know if there is a high morphine requirement.

Pain scores for intubated patients are recorded using the Critical Care Pain Observation Tool (CPOT) based on facial expression, body movements, compliance with the ventilator, and muscle tension. The score is recorded under the Pain tab on Metavision. Just before extubation the patient also receives 1g intravenous paracetamol.

Once extubated, patients receive regular oral paracetamol 1g QDS and dihydrocodeine 30mg QDS. For breakthrough pain, they have oral morphine on the PRN side. Pain scores for extubated patients are recorded using the self-reporting scale of 1 to 10 and the CPOT tool for extubated patients.

Pain management

The mainstay of pain management in post-op cardiac patients are opiates. Opiate use, however, comes with untoward side effects, namely respiratory depression, increased risk for delirium, and reduced gastric motility. Multimodal analgesia should be used whenever possible to decrease opiate requirements and provide better pain relief.

Paracetamol: all patients on CICU receive 1g intravenous paracetamol before extubation and 1g oral paracetamol QDS thereafter. When transferring a patient to the ward make sure no double dosing of paracetamol occurs as prescriptions are changed from Metavision to JAC.

Opiates: In theatre, patients usually receive up to 1mg fentanyl during their surgery. On arrival on CICU while still intubated they receive intravenous morphine in 1-2 mg boluses. Once extubated, they receive regular dihydrocodeine with oral morphine on the PRN side. Other opiates such as oxycodone and tramadol are also available and can be used instead of morphine.

Exchanging one opiate for another rarely leads to better analgesia as in equipotent doses they should have a similar analgesic effect. It might be more effective to think of adding a different type of drug rather than changing a medication within the same group. Fentanyl, morphine and oxycodone can also be set up as a PCA. Opiates are potent respiratory depressants and should be used carefully in patients at risk and in the elderly. Make sure laxatives are also prescribed to prevent opiate induced constipation. In ventilated patients who need longer sedation, an opiate is given as an intravenous infusion - usually fentanyl or morphine but remifentanil can also be used (but it should be time limited and for a definite indication as expensive).

NSAIDs: NSAIDs are very effective for alleviating musculoskeletal pain. Unfortunately, their use in cardiac surgery patients is limited by their potential for precipitating acute kidney failure in susceptible patients. They

can still be used judiciously in younger patients with normal renal function. Diclofenac is available as a suppository and as a tablet.

Others:
Gabapentin, Pregabalin: Neuropathic pain medications should be restarted as soon as possible in patients who usually take them for chronic pain. Moreover, the current PADIS guideline suggests using neuropathic medications with opioids for pain management in adults after cardiac surgery. Small trials have shown significant reduction in opiate consumption with their use.

Clonidine is an alpha-2 agonist with weak analgesic and sedative properties. It can be used as an adjunct in patients with poorly controlled pain and agitation. Be mindful of its side effects which include bradycardia and hypotension. It can be given as an oral tablet or an intravenous infusion.

Ketamine: the current PADIS guideline suggests ketamine at a dose of 1-2 mcg/kg/hr as an adjunct to opioid analgesia. It is rarely used on CICU for that indication.

Lidocaine patches - transcutaneous lidocaine patches are available on the unit and tend to be used in patients who complain of severe postoperative pain. The data as to their effectiveness is not compelling but their use is safe. However, they are expensive and often not stopped before leaving CTITU so can be continued, without need, whilst on HDU or the wards. Please make sure they are stopped when no longer needed.

Also, a simple non-pharmacological measure can be the timely removal of chest drains. If the drains have low output liaise with the surgical registrar to see if they can be taken out.

Sedation

Some patients might need sedation for longer periods of time due to neurological, respiratory or cardiovascular issues. If they are expected to remain intubated for more than 24-hours, sedation will be usually maintained with at least two agents - a sedative-hypnotic agent and an opiate - usually propofol and fentanyl.

The sedation level is evaluated by the nursing team and recorded under the Neuro tab on Metavision. The GCS, the RASS score and the AVPU response can be found under this tab along with pupil size and reaction. The latest PADIS guideline recommends keeping patients under light rather than deep sedation which corresponds to a RASS score of -2 and up. In general terms we aim for a patient that is calm, cooperative and communicative.

Propofol is an anaesthetic agent with no analgesic properties. Usual dose on CICU is about 4-6 mg/kg/hr and it is often combined with a fentanyl infusion. It has a negative inotropic effect and reduces blood pressure in a dose dependent manner.

Midazolam and morphine are sometimes used when sedation is expected to continue for longer. Midazolam is given at a loading dose of 0.01-0.05mg/kg followed by an infusion of 0.02-0.2mg/kg/hr. Both midazolam and morphine may accumulate with prolonged use and especially in patients with renal or hepatic dysfunction.

Dexmedetomidine is an alpha-2 agonist typically used for conscious sedation. It is useful in patients in whom delirium might be precluding extubation or in extubated patients that are agitated and delirious. It causes minimal respiratory depression. Very common side effects are bradycardia (sometimes extreme) and hypotension. The usual dose is 0.7 mcg/kg/hr with a dose range of 0.4-1.4 mcg/kg/hr. It cannot be stopped abruptly due to withdrawal symptoms. There is a protocol regarding its use available on Metavision.

Clonidine is another alpha-2 agonist but less specific and less potent than dexmedetomidine. It can be used as a weak sedative agent via an infusion. Clonidine can cause significant blood pressure drop and may not be tolerated by all patients.

An opiate infusion is always used when patients are being under prolonged sedation to provide analgesia and tube tolerance on the ventilator. Fentanyl is the usual agent of choice as it causes relatively little hypotension

and histamine release and does not accumulate in patients with renal impairment. The typical rate is 50-200 mcg/hr (1-3 mcg/kg/hr).

Morphine can occasionally also be used as an infusion but is usually avoided due to its tendency to accumulate especially in patients with renal impairment.

Remifentanil is an ultra-short-acting opiate with a context-sensitive half-time of 3 minutes. It can be used in patients with severe renal/hepatic dysfunction, in the extremely obese patients, when frequent neurological assessment is required, or when other agents have failed. When stopped it provides no residual analgesia so make sure to get longer acting pain medications on board before discontinuing it. It is expensive and its use is limited to the special circumstances listed above. It is run as a continuous infusion without a loading dose as boluses can precipitate severe bradycardia. The rate range is 0.1-0.3 mcg/kg/min. There is a protocol available on Metavision.

Some patients may require paralysis on CICU either due to severe respiratory failure with challenging ventilation or when they are left with an open chest. Paralysis is achieved via a cisatracurium bolus (0.15mg/kg) followed by an infusion (usually 0.18 mg/kg/hr). The degree of paralysis is evaluated with a peripheral nerve stimulator. Cisatracurium is cleared by ester hydrolysis in plasma and has no active metabolites so it can safely be used in patients with organ failure. There is a protocol available on Metavision.

Delirium

Delirium is a frequent postoperative complication after cardiac surgery. According to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) it represents a disturbance in attention, awareness and cognition that develops over a short period of time, represents a change from baseline and tends to fluctuate during the day. The change cannot be better explained by another pre-existing, evolving or established condition and it is usually caused by a medical condition, substance intoxication or a medication side effect.
Delirium is a serious complication that is associated with increased mortality, prolonged length of stay, and long-term worse cognitive outcome. Therefore, its recognition and prompt treatment are a priority. It usually presents in three forms - hyperactive delirium, hypoactive delirium and mixed being the most common.

The risk for developing delirium can be evaluated by examining the predisposing risk factors (related to the patient) and the precipitating risk factors that trigger the onset of delirium. Common predisposing factors are advanced age, pre-existing dementia and cognitive disturbances, Parkinsonism, co-morbidities such as heart failure, vascular disease, diabetes, anaemia etc. Precipitating factors can be sepsis, heart failure, metabolic disturbances, medications, major surgery and any other critical illness. All those predisposing and precipitating factors are commonly encountered in the cardiac surgery patient population. Delirium incidence post cardiac surgery has been estimated between 26-52% with significant incidence of hypoactive delirium.
The recommended tool for identifying delirium in intensive care patients is the Confusion Assessment Methods – ICU (CAM-ICU). The nurses complete a CAM-ICU for every patient and record it under the Neuro tab on Metavision.

Strategies to reduce the incidence of delirium include early mobilization, avoiding medications that can precipitate delirium, frequent orientation of the patient as to time and place, family visits, hearing and visual aids for patients with impairments, decreasing noise levels and improving the natural sleep/wake patterns. Addressing medical issues that precipitate delirium such as pain, hypoxia, infection, dehydration, and poor nutrition is crucial. Medications that can precipitate delirium are benzodiazepines, opioids, digoxin, diuretics, many antibiotics, antidepressants, antiemetics and many others. The use of some of those drugs cannot be avoided, however, whenever possible try to minimize polypharmacy and use a multimodal opioid sparing analgesia. To date, no drug has been identified that can prevent the occurrence of delirium and current guidelines do not recommend the prophylactic use of antipsychotics, statins or any other medication for the prevention of delirium.

The treatment of delirium targets two things: the treatment of the underlying cause and the treatment of symptoms. Whenever there is new onset delirium in a postoperative patient, first re-evaluate the patient for any acute change in their condition. Hypoxia, sepsis, heart failure, arrhythmia, ischaemia, metabolic

disturbances, bleeding, tamponade and many other conditions can manifest with delirium. Always exclude and treat pain before treating delirium symptoms with antipsychotics.

The latest PADIS guideline does not recommend routine treatment of delirium with antipsychotics. However, in patients in significant distress who might pose a risk to themselves or others, the guideline suggests using either Haloperidol or an atypical antipsychotic (olanzapine is available on CICU) to treat delirium. Treatment should be discontinued immediately after resolution of symptoms. Dexmedetomidine is also recommended for the treatment of delirium in mechanically ventilated patients when agitation is precluding weaning and extubation.

Haloperidol is a typical antipsychotic medication. The initial dose is 2.5-5.0 mg iv and the maximum daily dose should not exceed 10-12 mg. Haloperidol increases the QT interval and can precipitate Torsade's de pointes in susceptible patients.

Olanzapine is a new, atypical antipsychotic. It has been evaluated in several trials and seems to be non-inferior to haloperidol for the treatment of active delirium symptoms. The usual dose is 5mg but 2.5mg can be given initially to patients at risk of respiratory depression.

Dexmedetomidine infusion can also be started and titrated to effect. It causes minimal respiratory depression but might lead to bradycardia and a fall in blood pressure.
In challenging cases even, a propofol infusion can be used but be vigilant regarding respiratory depression with propofol in non-ventilated patients.

Benzodiazepines are not recommended for the treatment of delirium except in patients with alcohol withdrawal symptoms. Often patients with a history of harmful alcohol use are prescribed chlordiazepoxide on CICU together with thiamine/ascorbic acid (Pabrinex).

Sleep disturbance on CICU

Melatonin - melatonin is a hormone secreted by the pineal gland at night and plays a role in the control of the sleep/wake cycle. 2mg MR tablets are available on CICU and usually 1-2 tablets are prescribed at night to improve sleep. The disruption of circadian rhythms and especially sleep on CICU is quite common and is hypothesized to contribute to ICU delirium. Currently there is no recommendation for or against the use of melatonin to help promote regular sleep patterns and prevent delirium. However, given the excellent safety profile and the potential for improvement of sleep quality it is often prescribed at night.

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CICU Investigations

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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 any chest x-ray in the notes.
Rarely we may need a chest X-ray to find the correct location of the endotracheal tube and may avoid the consequences of endobronchial intubation and over-ventilation of right lung with under ventilated left one. Percutaneous tracheostomies should be x-rayed after insertion.

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 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.

Once you have decided to get CT, all requests should undergo a process of vetting by a radiologist, or through an agreed protocol with subsequent authorisation by a radiographer. This process is necessary for justification of the investigation: appropriate use of imaging, radiation safety, and judicious use of resources

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 unresolved sepsis, or to investigate patients with severe lung disease like ARDS preventing weaning from mechanical ventilation.
Abdominal CT scans are usually reserved for patients who develop signs of serious intra-abdominal pathology (perforated viscus or mesenteric ischaemia presenting as gastric ileus/coffee ground aspirates in NG).

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, suction unit as well as a single power supply to all equipment.

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 valvular dysfunction or regional wall motion abnormalities suggesting graft failure, or global LV dysfunction
- New murmurs
- Failure to wean from mechanical ventilation due to pulmonary oedema

During working hours, transthoracic 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 (Non-invasive cardiology, E level, opposite the perfusion department entrance to cardiac theatres). 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.

Bronchoscopy

Most ICU patients who require bronchoscopy are mechanically ventilated. The most common diagnostic indication is to collect lower respiratory tract samples for cultures, and most common therapeutic indication is broncho-alveolar lavage and removal of mucus plugs or bronchial secretions.

Bronchoscopy is of two types -rigid & fibreoptic. Fibreoptic bronchoscopy is the one commonly used in critical care.

Any suspected aspiration, lower lobe atelectasis, haemorrhage may hinder the weaning from ventilator. Usually, fibreoptic bronchoscopy is done by the consultant anaesthetist/ intensivist or registrar/ senior clinical fellow (under appropriate supervision). In very rare cases, like very severe haemorrhage, a respiratory specialist team might have to be involved. It can be done by placing a request on eQuest system. Rigid bronchoscopy is usually done only in theatres by cardiothoracic surgeons.

TEG & ACT

TEG & ACT are point of care tests of coagulation. Further details can be found in the sections on bleeding and point of care testing.

Nutrition on Cardiac ICU

Author: Maria Beja & Bethan Jenkins (Critical Care Dietitian) *Revised: February 2023*

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. Although MUST still needs to be completed as per of the trust policy, it is important to mention that this risk tool is not specific for critical care patients and the evidence says that all patients who have an ICU stay >48h will be automatically at high risk of malnutrition (ESPEN 2019).
The fact that most patients require enteral feeding, and considering the complexity of their condition, a dietetic assessment is often required even when the most score is low.
When in doubt, please do not hesitate to contact the critical dietitians on ext. 6072.

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. Sometimes, a post-pyloric route might be considered (NJ, JEJ, PEG-J) when gastric access is contraindicated or when patients are unable to tolerate gastric feed.
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

Guidelines suggest that enteral nutrition should be commenced early in the ICU admission (within 24-48 hours) and volumes gradually increased over the first week of admission (Van Zanten 2019).
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 consider other sources of energy, such as propofol, citrate and dextrose infusions, when prescribing the feeding regimen.

Initially, a wide bore NG tube is passed (usually 10-12fr 'Corflo®' tube). These are easier to aspirate, which is necessary to gastric emptying in sedated patients.

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 el 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 a Critical Care Nutrition guideline, which should ensure that all patients are fed using the best practice. This document is available on Staffnet.

All patients are usually started on Nutrison Protein Plus at 40 ml/hour, unless patients are at risk of refeeding and are any of the following:

- BMI less than 16 kg/m2
- No or minimal oral/enteral intake more than 5 days
- History of alcohol abuse or IVDU
- More than 10% weight loss in 6 months.
-

If patients are at refeeding risk, please start rate at 15 ml/h as per the critical care nutrition guidelines available on Staffnet and refer to the dietitians.
Patients at risk of refeeding syndrome will require either IV pabrinex for 3-5 days or thiamine 100mg tds for 10 days.

On ICU we have a volume-based feeding protocol – known as enhanced feeding protocol – which ensure patients meet targets on a regular basis and provides clear guidance for nurses on the rate the feed should be running on. This strategy is known to improve calorie delivery during ICU admission (McClave, S. et al 2015) and the rate should be checked and adjusted when required every 4h.

Gastric residual volumes should be monitored 4-hourly. If aspirates are 500ml or more then pro-kinetics and alternative enteral feeds should be considered. If aspirate is greater than 500ml IV metoclopramide 10mg tds should be prescribed. If 2 consecutive aspirates are over 500ml reduce feed rate by 25ml/hr to a minimum of 25ml/hr and contact the dietitian. If aspirates remain high after 24 hours of metoclopramide, consider prescription of erythromycin.

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 GI symptoms and sings of intolerance (vomiting, abdominal distension and bloating, diarrhoea and GRV over 500ml, 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 24hours 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, please refer to the enhanced feeding protocol as the feed rate will likely need to be adjusted 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 and 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.

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
Consider 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. We are also happy to attend the ward round if that is helpful, but this is not routine at present.

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.
- Are there any other drugs with motility or osmotic effect that need to be reviewed?
- 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 changed.

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

Progressing to Oral diet

Once enteral feeding is stopped, or patients start to eat and drink following their procedure, food record charts are kept to monitor their intake.
We do not advise to remove NG tube before patients are assessed by the dietitians as oral intake is often minimal after extubation or even during ICU admission and patients might still need NG feeding to meet their nutritional requirements.
If patients' intake is poor of if there are any issues related to their diet, allergies, or intolerances, 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 protein bd for the first 72 hours post operatively as oral intake is unlikely to meet nutritional requirements during this time (if there is no contraindication for this indicated by the medical team).

Gastrointestinal Disorders on CICU

Author: Dr Godfrey Nerupfunde *Revised: December 2022*

Previous Authors: Drs A Richardson & K Wilkinson

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), stool softeners (e.g. Movicol) and pro-motility agents (e.g. senna) should be used in patients who have not had a bowel motion for more than 72 hours. It should be noted that they may take up to 2 days to show effect and therefore should be prescribed pre-emptively. 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
- Preop fasting
- Prolonged mechanical ventilation
- Prolonged period on cardio-pulmonary bypass, complex procedures, emergency surgery
- High inotrope/vasopressor requirements, intra-aortic balloon pump use
- Renal replacement therapy
- Atrial fibrillation
- Coagulopathy/anticoagulation

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 recommenced 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 disassociated 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. Diagnostic delays may result from the high lactate associated with post op patients on inotropes. Continuous haemofiltration may mask the onset and extent of any lactatemia. 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. CT angiography can be done if the patient is stable, and endoscopic studies if they are too sick for transfer to the scanner.

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. Cholestasis from ischaemic injury impairs digestive function and predisposes the patient to cholangitis that accelerates liver failure. 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 dysfunction and liver failure

Liver dysfunction may affect up to 25% of cardiac surgical patients. 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.

The severity of liver dysfunction ranges from transient hyperbilirubinaemia to frank liver failure. Prolonged CPB time (>100 min) is associated with an elevation of aspartate transaminase on postoperative day 2. Postoperative unconjugated hyperbilirubinaemia may be a result of haemolysis on CPB, a mechanical valve or haemolysis of transfusion. This usually is transient and improves within 3 days. Late peaking or persistent hyperbilirubinaemia (beyond postoperative day 3.5) carries a higher risk of higher morbidity and mortality.

Severe ischaemic liver injury usually presents as metabolic acidosis, hyperlactataemia, coagulopathy, hemodynamic instability, and hypoglycaemia. Risk factors include history of cardiac failure, diabetes, hypertension and prolonged CPB time. It is important to commence supportive treatment early and minimise additional stress from ischaemia, haemorrhage, or sepsis.

Acute liver failure is a highly specific condition characterised by rapidly progressive liver dysfunction with coagulopathy (INR> 1.5) and encephalopathy in a patient with previously normal hepatic function. Without encephalopathy, it is termed acute liver injury and carries a better prognosis. Investigations are aimed at determining aetiology and assessment of severity and involvement of other organ systems, particularly hepatic encephalopathy. Initial tests include acetaminophen levels, serological markers of Hepatitis A (IgM VHA), HBsAg, anti HBc IgM, urine toxicology screen (amphetamine, cocaine), hepatic doppler ultrasound, triple phase CT scan of the liver and echocardiography, which may demonstrate congestive hepatopathy from right heart failure. Assessment of severity and disease progression includes assessment for encephalopathy, haemodynamics, coagulation (INR, Factor V), glycaemia, lactate level and arterial ammonia. The mainstay of management centres on early referral and supportive measures.

Thresholds for referral to a specialist centre generally include INR>3/ PT>50s, increasing hepatic encephalopathy, hyperlactataemia or hypotension despite resuscitation, acidosis, bilirubin> 300 micromol/L, AKI and shrinking liver volume.

Supportive measures in ALF include early tracheal intubation and ventilation for airway protection and avoidance of agitation using strategies to prevent increases in ICP. Haemodynamic goals include maintaining perfusing MAP >65mmHg, often higher in hepatic encephalopathy. Volume resuscitation using cardiac output monitoring with a balanced crystalloid is recommended, and although albumin has no effect on mortality, it can be used as a colloid volume expander. Starches based colloids are avoided to preserve renal function. Noradrenaline is the vasopressor of choice, avoiding terlipressin with its attendant splanchnic vasoconstriction. AKI accompanying ALF is associated with poorer outcomes and early RRT is recommended for renal support, hyperammonaemia, sodium imbalances, temperature control and metabolic control.

Monitoring and control of intracranial pressure is essential, and care should be taken to maintain ammonia levels below 100micromol/L. Other targets to prevent rise in ICP include maintaining Na+ between 145-150 and serum osmolality below 320. Hypertonic saline may be used to treat surges in ICP, as well as mannitol. Fever should be avoided.

Coagulopathy poses a bleeding risk, the most sensitive markers being platelet count and fibrinogen, which should be corrected as required. Changes in INR/PT reflect the synthetic function of the liver and should be corrected for invasive procedures like invasive ICP monitoring.
Sepsis is multifactorial, accelerates HE, increases mortality and delays transplantation. Prophylactic antimicrobials should be given with micro advice.
Electrolytes derangements are common and should be corrected promptly. Nutritional support, preferably enteral, should be given with a target of 25-30 kcal/kg/day.

Cardiac surgery associated acute kidney injury

Authors: Dr Guillaume Bousquet-Dion & Dr Kirstin Wilkinson *Revised: November 2022*

Previous author: Dr Kirstin Wilkinson

Incidence

Acute kidney injury (AKI) is a severe complication occurring in up to 30% of patients after cardiac surgery and requiring renal replacement therapy in 1 to 5% of patients. Its incidence has been stable since the 1960s, but its associated mortality decreased. Renal failure continues to carry a 64% 30-day mortality compared to 4% in its absence. Audit data from our unit confirm these figures, revealing a median survival of only 20 months. Acute renal failure (ARF) following cardiac surgery increases the risk of post-operative sepsis, gastro-intestinal 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.

Staging

Stage	RIFLE (2004)	AKIN (2007)	KDIGO (2012)
Stage 1/ Risk	SCr 1.5x baseline **or** GFR decrease > 25%	SCr 1.5-2x baseline **or** ≥ 26mmol/L increase	SCr 1.5-1.9x baseline **or** ≥ 26mmol/L increase
Urine output < 0.5mL/kg/hr x6h			
Stage 2/ Injury	SCr 2x baseline **or** GFR decrease > 50%	SCr 2-3x baseline	SCr 2-2.9x baseline
Urine output < 0.5mL/kg/hr x12h			
Stage 3/ Failure	SCr 3x baseline **or** SCr > 352 mmol/L (with acute rise > 44mmol/L) **or** GFR decrease > 75%	SCr 3x baseline **or** SCr > 352 mmol/L (with acute rise > 44mmol/L) **or** Initiation of RRT	SCr 3x baseline **or** SCr > 352 mmol/L (with acute rise > 44mmol/L or 1.5x baseline) **or** Initiation of RRT
Urine output < 0.5mL/kg/hr x24h **or** Anuria x12h			
Loss	Complete loss of renal function > 4 weeks		
ESRD	Complete loss of renal function > 3 months		

Table 1. Comparison of commonly used definitions and staging of AKI. RIFLE: Risk, Injury, Failure, Loss of kidney function, and End-stage renal disease; AKIN: Acute Kidney Injury Network; KDIGO: Kidney Disease Improving Global Outcomes; SCr: serum creatinine; GFR: glomerular filtration rate; RRT: renal replacement therapy; ESRD: End-stage renal disease. Of note, the relative SCr increases are within 7 days and the absolute SCr increases are within 48h

Risk factors and risk assessment

The presence of known preoperative risk factors accounts for only 30% of the incidence of ARF after cardiac surgery. The figure below gives a general continuum to potential exposures and events that might impair or predispose to perioperative renal impairment.

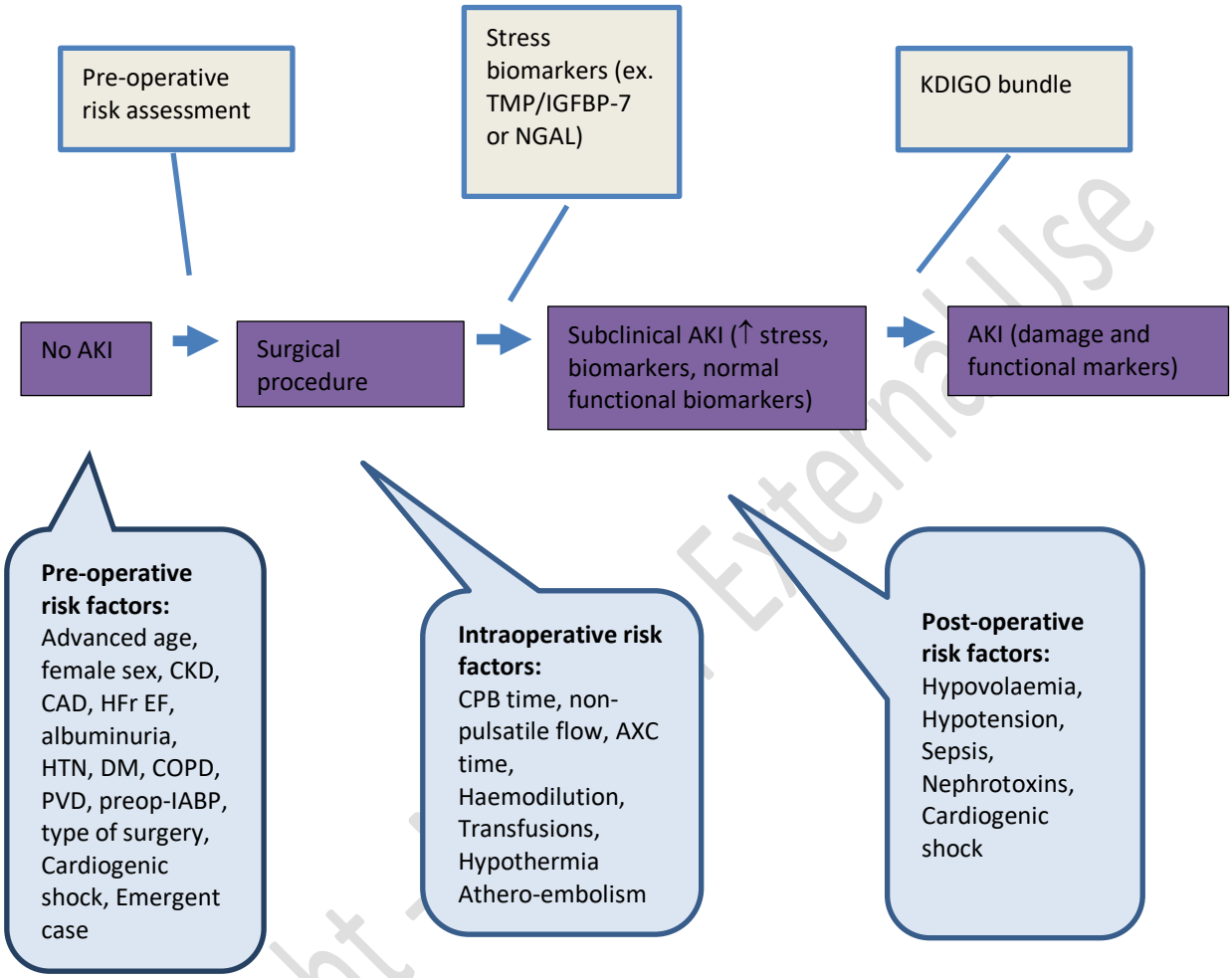

Fig. 1. A framework for the time course of risk assessment in CSA-AKI. Risk assessment should be a continual process performed repeatedly in the pre-, peri-, and early postoperative period. It should include clinical factors and biomarkers if available. HFrEF: heart failure with reduced ejection fraction; COPD: chronic obstructive pulmonary disease; CPB: cardiopulmonary bypass; AXC: aortic cross-clamp; IABP: intra-aortic balloon pump; IGFBP-7: insulin-like growth factor binding protein 7; NGAL: neutrophil gelatinase-associated lipocalin; PVD: peripheral vascular disease; TIMP2: tissue inhibitor of metalloproteinases 2.

Pathogenesis of CSA-AKI (cardiac surgery associated acute kidney injury)

Haemodynamic perturbations	**Mechanical factors**	**Inflammation/Immunity**	**Other mechanisms**
Effect of CPB circuit	Emboli	Inflammation	Neurohormonal
Blood pressure	Arterial obstruction	Oxidant stress	Vasoconstriction
Preload/volume	Venous congestion	Complement activation	Venous congestion
Anaemia/haemolysis	Perfusion pressure	Toxins	Tubular toxicity
Ischaemia/reperfusion	Abdominal hypertension	Drugs	
Venous pressure		Contrast media	

Table 2. Many common factors contributing to the development of CSA-AKI

The pathogenesis of AKI after cardiac surgery is not completely understood. It is very unlikely that only one factor could cause perioperative AKI. It is rather a consequence of multiple preoperative, intraoperative and postoperative interactions.
Two main explanations for CSA-AKI are the complex and urgent nature of the cardiac surgical procedures. Especially two consequences of CPB contribute to renal impairment: inflammation response and haemodilution. Haemodilution causes decreased oxygen carrying capacity and delivery while inflammation leads to increased oxidative stress, mitochondrial dysfunction and decreased end organ perfusion. Non-pulsatile flow of CPB also puts the kidney at danger of ischaemia. It is mainly the renal medulla, which is vulnerable since its oxygen delivery is already low. Factors influencing renal blood flow are mainly prolonged CPB (>180 minutes), prolonged AXC, low cardiac output state and ischaemia-reperfusion injury. Other contributors to renal ischaemia are the tubular insults such as toxins, metabolic derangements, neurohormonal activation, tubular oxidative stress and inflammatory response. There also seems to be an association between blood transfusion and CSA-AKI. In a study, the risk of AKI was higher when more than 2 units of red blood cells (RBC) were transfused during surgery. Apart from the economic standpoint, this is yet another reason why liberal blood transfusion should possibly be avoided.

Assessment of renal function

Urine output: increases linearly with arterial blood pressure. As such it is related to many factors including cardiac output and intravascular volume status and is therefore an insensitive marker of renal function.
Creatinine: Around 50% of the excretory function of the kidney can be lost without an increase in SCr and GFR can fall below 30 ml/min without urea retention. However, changes in SCr or creatinine clearance are still the most used markers of renal dysfunction. They have been validated against long-term outcome after cardiac surgery in large studies.
Sodium excretion: As renal perfusion decreases the kidney conserves sodium. Urinary sodium excretion tends to decrease in conditions leading to pre-renal failure and early tubular necrosis. In cases of intrinsic renal disease, the sodium resorption fails, and the urinary sodium concentration increases. Fractional excretion of sodium (FENa) is the sodium clearance divided by the creatinine clearance. A FENa < 1% indicates a pre-renal problem, while FENa > 2% hints at an intrinsic renal disease. This interpretation is only clinically validated in patients with oliguric AKI and in the absence of diuretics, ESRD, urinary tract obstruction and/or acute glomerular disease.

Therapy

The initial approach to treating the oligo-anuric patient optimizing renal oxygen delivery by correcting, if present, hypovolaemia, hypotension, low-flow state, anaemia, hypoxia, urinary obstruction. Pharmaceutical or renal replacement therapies can then be considered.

Pharmaceutical strategies

Dopamine produces a dose dependent increase in renal blood flow over the dose range 0.5-3 μg/kg/min. However, the α- and β- adrenergic effects of dopamine are still present at these doses so it is likely that there is no real "renal dose". Although dopamine does increase the urine output, there is no proof of renal protection to patients undergoing cardiac surgery. There is even some evidence that it may exacerbate renal tubular injury in the early postoperative period. Additionally, it is prolactin-inhibiting hormone and possesses some immunosuppressive effects when administered as long-term intravenous infusion (14 days).
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. It also possesses free radicals scavenging abilities. Even though mannitol produces a transient increase in urine output, its ability to prevent ARF is unclear. Mannitol is often put into the cardiopulmonary bypass prime. Care must be used in its use as it may lead to profound hypovolaemia and increases renal medullary oxygen consumption.
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.

The evidence currently does not support the use of other therapeutic interventions such as preoperative statins, acetylsalicylic acid, N-acetylcysteine, enalaprilat, fenoldopam and sodium bicarbonate.

Renal replacement therapy

When pharmacological methods have failed and urine output is inadequate to sustain metabolic balance, RRT should be considered. There is some evidence that early start of dialysis (within 24 hours after cardiac operation or 12h of the onset of oligo-anuria) improves the outcomes of the patients. RRT may be indicated for fluid volume control or for biochemical derangements such as persistent hyperkalaemia, acidosis or uraemia. The decision to commence RRT should be discussed with consultant intensivist and should include a plan for fluid balance management. Normothermic patients in the ICU will have a fluid deficit around 500ml per day in "insensible" loss from the airway, gut and perspiration. This loss increases by 100mL with every 0.5 °C > 37 °C of body temperature.

Vascular access: important determinant of the quality of the RRT. Jugular and femoral sites are equivalent in terms of infectious complications. Jugular vein insertion might be preferable in obese patients. Insertion in the left jugular vein has been associated with greater rates of catheter dysfunction compared with the right jugular or femoral veins. Additionally, deeper insertion of jugular catheters with positioning in the right atrium has an advantage in terms of filter life and overall better performance. The subclavian veins should be avoided as it can lead to subclavian vein stenosis which can jeopardize arteriovenous fistula placement for chronic dialysis or insertion of permanent pacemaker. Usually, 16cm lines will be used in the right internal jugular lines while 20cm lines will be used in the left internal jugular. For the femoral accesses, 25cm lines should be used.

Anticoagulation and mode of RRT in our ICU: Traditionally heparin would be avoided if within 12 hours after cardiac surgery, then used targeting an APTR 1.5-2.0. Alternatives to heparin include citrate, low molecular weight heparins (LMWH) with anti-Xa monitoring (0.25-0.35 U/ml) and Argatroban (HIT suspected/confirmed). The default CRRT protocol on our unit uses regional citrate anticoagulation (RCA) with a concurrent calcium infusion, the CVVHDF mode set at blood flow 150-200 ml/min and total effluent dose 25-35 ml/kg/h. Ultrafiltration is then gradually introduced as tolerated haemodynamically. Compared with systemic heparin, RCA seems to be associated with less bleeding, increased filter lifespan, and reduced transfusion rates and need for antithrombin III/platelet supplementation.

Care must be taken in the presence of liver failure as citrate accumulation may occur and is best detected by the total calcium-to-ionized calcium ratio – a ratio of more than 2.5 indicates citrate accumulation syndrome.

Conclusion

AKI after cardiac surgery is common, although most often mild. The development of any AKI remains a major predictor of adverse outcomes, including progression of CKD. Effective prevention and treatment strategies for AKI after cardiac surgery are being studied extensively. There are efforts to reduce AKI following cardiac surgery and its influence on patient morbidity, but for now they are confined to haemodynamic manipulations, close attention to intravenous resuscitation strategies including goal-directed therapy and balanced-salt fluid administration, reduced exposure to CPB, and the identification and mitigation of modifiable risk factors.

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End of Life Care

Authors: Dr Paul Diprose *Revised: November 2022*

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 based on 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. Referrals should be made via the eQuest system. For urgent advice, the palliative care team can be contacted on extension 4126 or bleep 1477.

Organ Donation

Most patients that die on CICU do so from multiple organ failure, frequently in the presence of sepsis. 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 the following number: 03000 203040

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.

Certification of Death

After death you may be asked to certify the death of the patient. This should be clearly recorded in the electronic clinical notes using the template form on Metavision. It is usually the responsibility of the surgical or medical team under whose care the patient was admitted to discuss the case with the IMEG team and (where appropriate) the coroner, and to complete both the death certificate and the cremation form. CICU 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 their 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). Bereavement care services can be contacted via extension 4587.

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.

Checklist after death

- Formal certification of death documented in the electronic clinical notes
- Appropriate documentation including tissue donation explained and leaflet given to relatives
- Relevant team informed of need to attend the next IMEG meeting

Emergency Patient Management

Hypotension after Cardiac Surgery

Author: Dr Sawal Atmosoerodjo *Revised: November 2022*

Previous authors: Drs Pierce & Wilkinson

Hypotension occurs frequently in the CICU after cardiac surgery. Due to many physiological changes after open chest and cardiac surgery, the differential diagnosis is broad.
Blood pressure is the product of cardiac output and systemic vascular resistance. Its major determinants are cardiac output (CO) and systemic vascular resistance (SVR), based on the following formula:

MAP - CVP = (CO x SVR)/80
Or
SVR = (MAP-CVP/CO) x 80

With CO = stroke volume x heart rate

Therefore, a fall in blood pressure is due to change in either HR, the SV or the SVR.

The anaesthetist from theatre will have set haemodynamic parameters based on the operation and the patient's state. These are noted in the admission notes in Metavision.
As a starting point, acceptable ranges of normal pressures after cardiac surgery are a systolic pressure of 100-140 mmHg and a MAP of 65-85 mmHg.

In the setting of hypotension, adopt a logical approach to analyze and restore the blood pressure. The following guidelines can aid in the management of hypotension. If the situation is catastrophic, attend to the patient and ask the senior nurse in CICU to contact the consultant anaesthetist.

Reduced CO		
Heart rate	**Stroke volume**	**Low SVR**
Tachycardia	Hypovolaemia	Drug induced
Bradycardia	Onset of AF	SIRS
Pacing box disconnection	Excessive PEEP/Auto-PEEP	Sepsis
	Tamponade	Anaphylactoid / Anaphylaxis
	Tension pneumothorax	
	HOCM +/- SAM	
	Acute mitral regurgitation	
	Anaemia	
	Acidaemia	
	Reduced coronary perfusion	

The table above summarizes the differential diagnosis of hypotension after cardiac surgery. A more detailed approach to hypotension is described below.

Approach

- First, exclude an artefact such as transducer error:
  - Try to aspirate and flush the arterial line
  - Check the height of the transducer
  - Check the pressure of the saline behind the transducer is >300 mmHg.

- If there were inotropes running, exclude any flow restrictions in the central venous access or occlusion errors on the syringe drivers.
- Secondly, include and examine the anaesthetic record and perfusion chart.
- Follow up with a full clinical examination including settings of the pacemaker, ventilator and syringe drivers.
- Finally, request laboratory tests and if indicated, a chest X-ray and a TOE/TTE examination.
- If there is profound refractory hypotension, avoid large doses of adrenaline to avoid the surge in blood pressure and potential for aortic disruption.

Is there a change in heart rate?

- Most patients after cardiac surgery will have temporary pacing wires. Ensure:
    - The pacing leads are connected to the box
    - The pacing box is on and has battery power
    - If the box is pacing but there is no capture, increase the output of both atrial and ventricular wire.
- Tachycardia can reduce the preload by shortening time for diastolic filling.
- Bradycardia will reduce cardiac output.
- If there are no temporary pacing wires and box, consider a 12-lead ECG to identify any arrhythmias.

Is there a fall in stroke volume?

- As stroke volume is determined by preload, afterload and contractility, issues in all three should be considered in your approach.

Reduced preload

- Are there signs of hypovolaemia? Check:
    - CVP trend
    - Chest drain production, both trend and absolute volumes
    - A good response in MAP on a leg raise test is indicative.
- Onset of new AF might reduce preload with loss of atrial kick.
- Exclude excessive PEEP or auto-PEEP. Disconnect breathing circuit if needed.
- Cardiac tamponade:
    - Tamponade is a clinical diagnosis and can be difficult to identify, as signs can be subtle. Check for:
    - A sudden fall (blocked drain) or an increase in chest drain production
    - Trends in CVP
    - Trend in inotrope requirements
    - If suspected, TOE or TTE can help in the identification.
- Tension pneumothorax:
    - Perform a clinical examination. Signs are a unilateral tympanic chest and reduced breath sounds and an increased CVP
    - There are often high peak inspiratory pressures due to the collapsed lung
    - If suspected, request an urgent chest X-ray. Look for a pneumothorax along with a mediastinal shift.

Reduced or excess afterload

- Patients with a hypertrophic obstructive cardiomyopathy (HOCM) can have outflow restrictions, with or without systolic anterior motion (SAM) of the mitral valve. TOE examination will aid the diagnosis.
- An acute mitral regurgitation (usually due to chord rupture) can be seen with TOE examination.

Reduced contractility

- Management can be difficult as reduced contractility can be a cause or an effect of hypotension. Ensuring you have optimized pre- and afterload will aid in identifying reduced contractility.
- Check the ABG if there is anaemia or acidaemia. Optimizing these can increase contractility.
- Coronary perfusion:
    - Check for adequate coronary perfusion pressure (aortic diastolic pressure – CVP or LAP)

- If occlusion of coronary grafts is suspected, perform an ECG
- TOE examination will aid in identifying regional wall motion abnormalities or systolic dysfunction.

Is the SVR abnormally low?

- The SVR can be calculated by the formula at the beginning of this chapter.
- A normal SVR is 700-1500 dynes/seconds/cm^{-5}

Causes:

- Drugs
  - Milrinone, dobutamine, Magnesium sulphate, ACE-I's, GTN or SNP can reduce the SVR. Check the syringe drivers for adequate doses.
- Pathological
  - SIRS is common after cardiac surgery, especially after longer CPB times. Check the perfusion record if large amounts of phenylephrine were required.
  - If there is suspected sepsis, take blood cultures, preferably during a fever.
  - Exclude any allergic response to recently given drugs or blood products.

Cardiac Arrest after Cardiac Surgery

Authors: Prof Charles Deakin and Dr Paul Diprose *Revised: November 2022*

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.

Although the principles of resuscitation are based on the standard advanced life support algorithm (Figure 1), there are important differences in the management of patients who arrest in the first ten days after cardiac surgery. This is because cardiac arrest in these circumstances is usually due to a different aetiology which is mostly treatable with focussed, rapid actions.

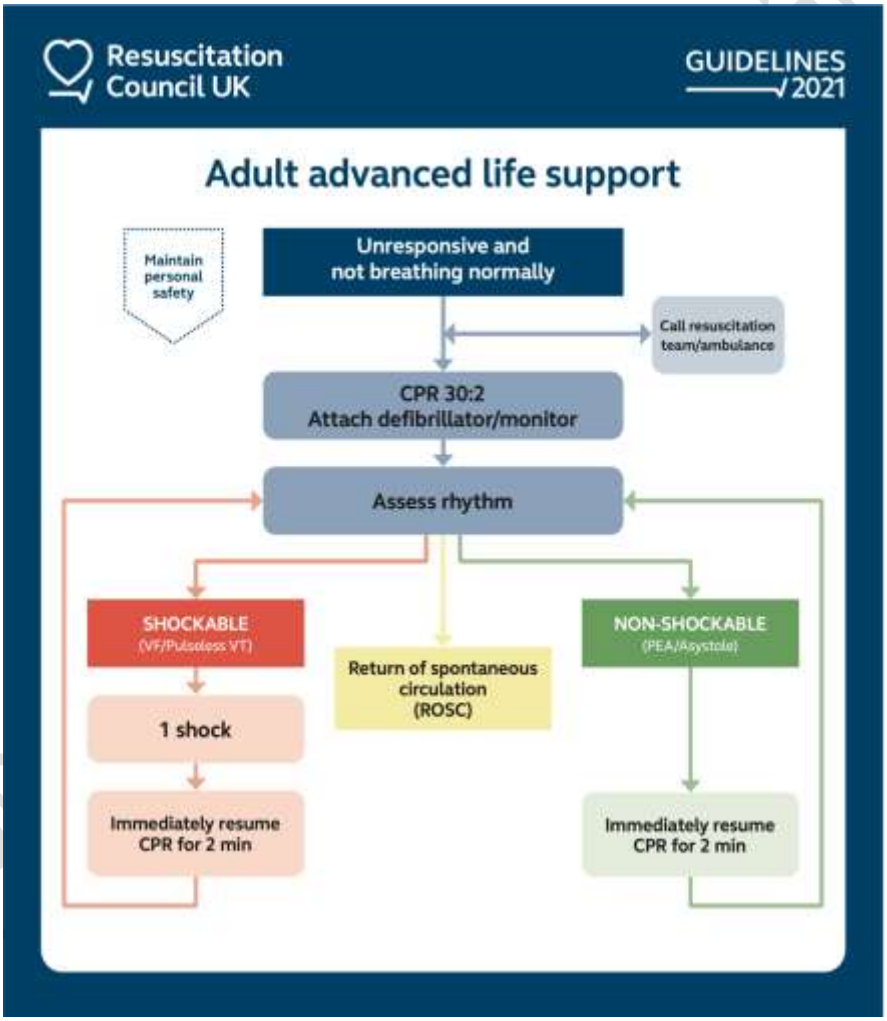

Figure 1: Adult advanced life support algorithm

To improve management and survival of these patients, this algorithm has been modified to address the specific needs of patients who suffer a cardiac arrest following 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 2 summarises the basic steps to be undertaken once an arrest has been diagnosed. The key principles are rapid identification of the rhythm and a focussed approach to identify the cause to enable appropriate treatment.

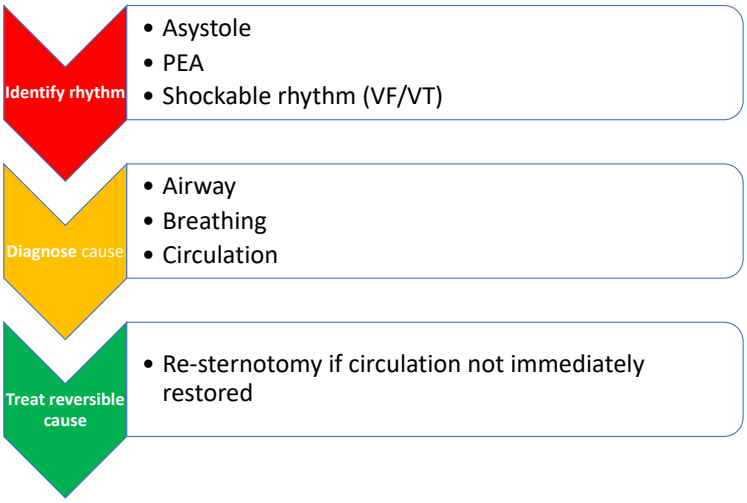

Figure 2: The basic steps to be undertaken once a cardiac arrest has been diagnosed

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.

Once a cardiac arrest has been diagnosed and a cardiac arrest call made, the following diagram summarises the steps that should be taken. **Remain focused on the identified rhythm and address the associated reversible causes**.

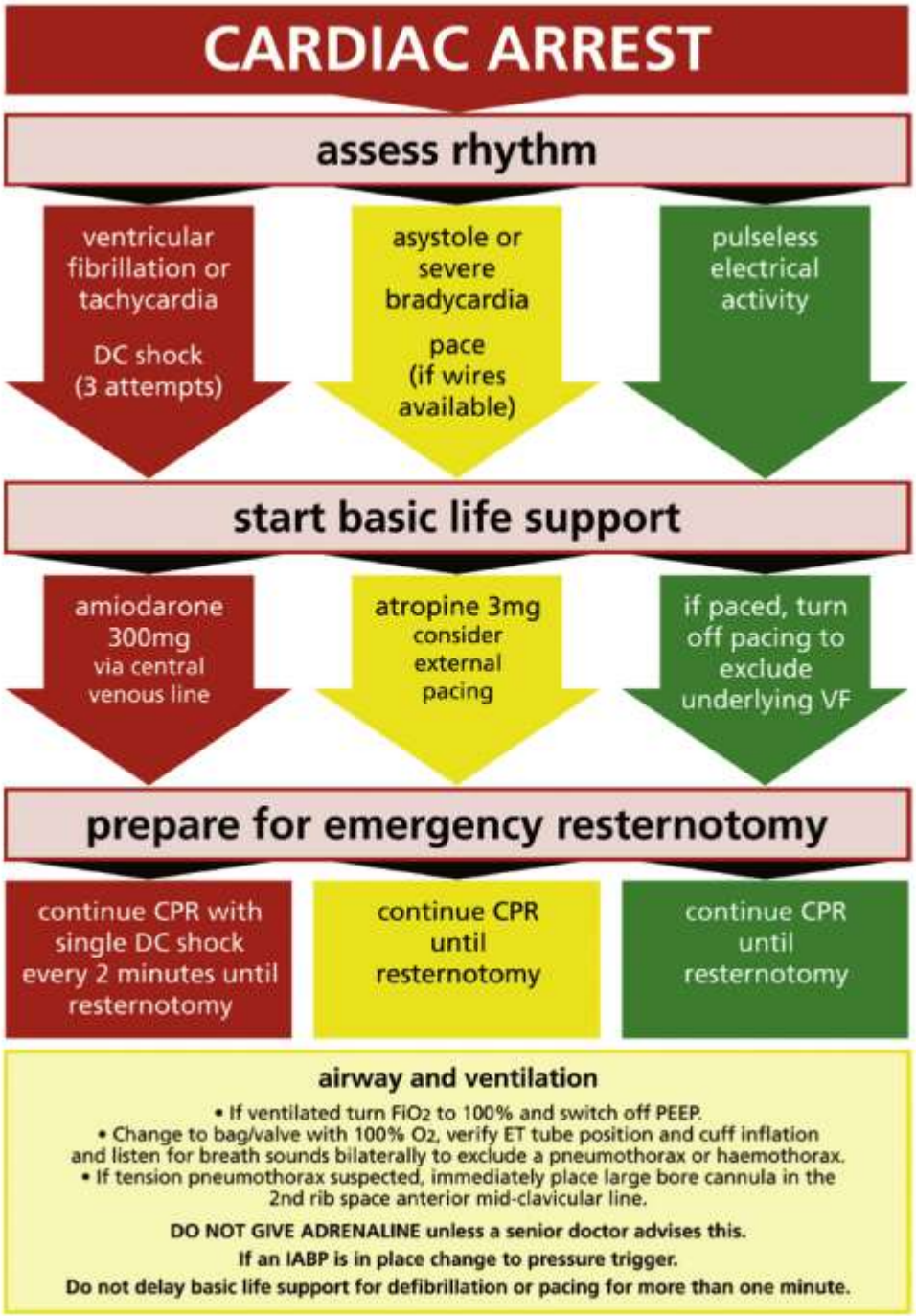

Figure 3: Pathways of management.

VF and Pulseless VT

- 3 sequential shocks (at least 150J) 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 (VF with pacing spikes may be mistaken for an organised rhythm).

Do not routinely give adrenaline during the cardiac arrest.

If these measures fail to restore return of a spontaneous rhythm, prepare for emergency resternotomy. Ensure that the chest opening team are called as early as possible in this sequence.

In addition, consider the following measures:

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.

Emergency Re-sternotomy

- See 'Chest re-opening' chapter in this manual.
- The cardiac arrest call should ensure that a member of the surgical team attends promptly. If re-sternotomy is indicated and no surgeon is present, any member of the clinical team should open the chest. This simple procedure involves cutting the skin stiches and removing the sternal wires. Opening the chest may be a lifesaving procedure in patients where the cardiac arrest is caused by a cardiac tamponade.
- A re-opening set is kept on CICU (ensure that you familiarise yourself with the location and types of equipment available).
- Internal cardiac massage is superior to external cardiac massage.

As with any cardiac arrest, appoint a team leader and ensure that all staff are aware of their roles and responsibilities. (Figure 4)

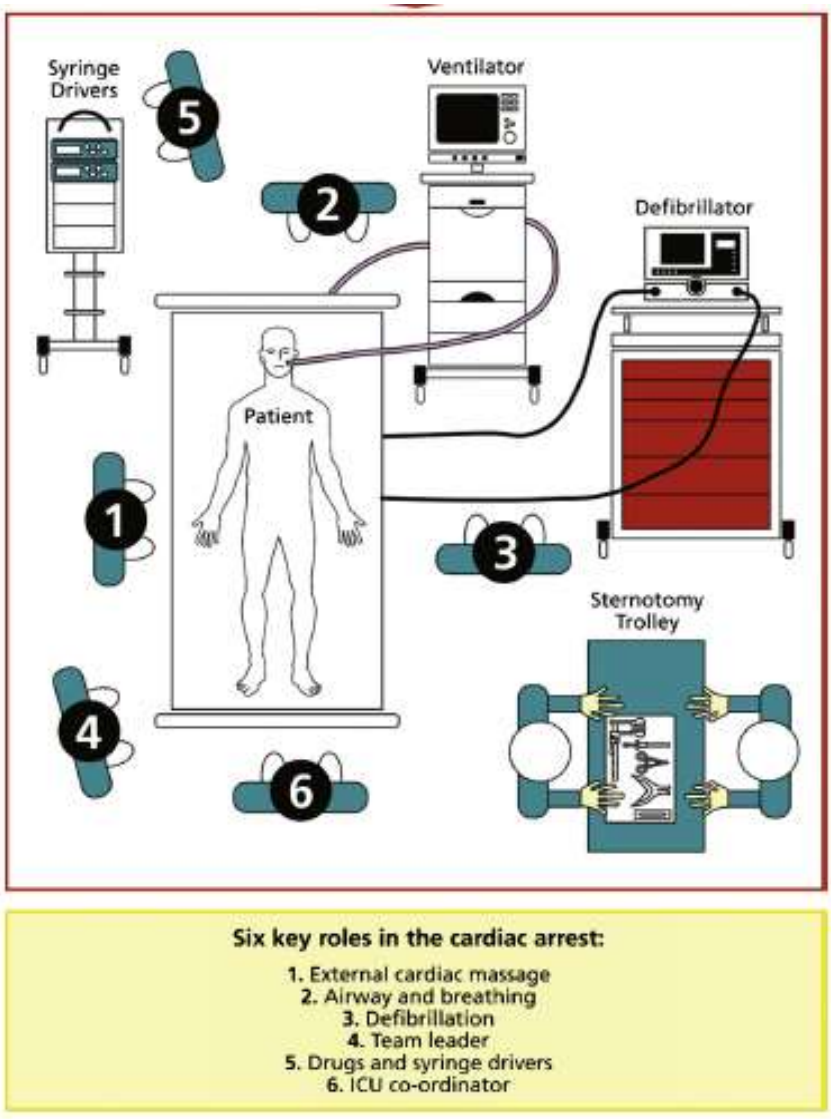

Figure 4: The six key roles for personnel during a cardiac arrest on CICU

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Emergency Chest Re-opening on CICU

Authors: Dr Tom Pierce & Dr Kirstin Wilkinson *Revised: February 2023*

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 bypass pump.

Anaesthetic and other drugs

- The patient needs to be anaesthetised.
- Sedatives, propofol or midazolam in appropriate dose for the patient and the circulation.
- Muscle 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 'overshoot' 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/crystalloid fluids
- 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

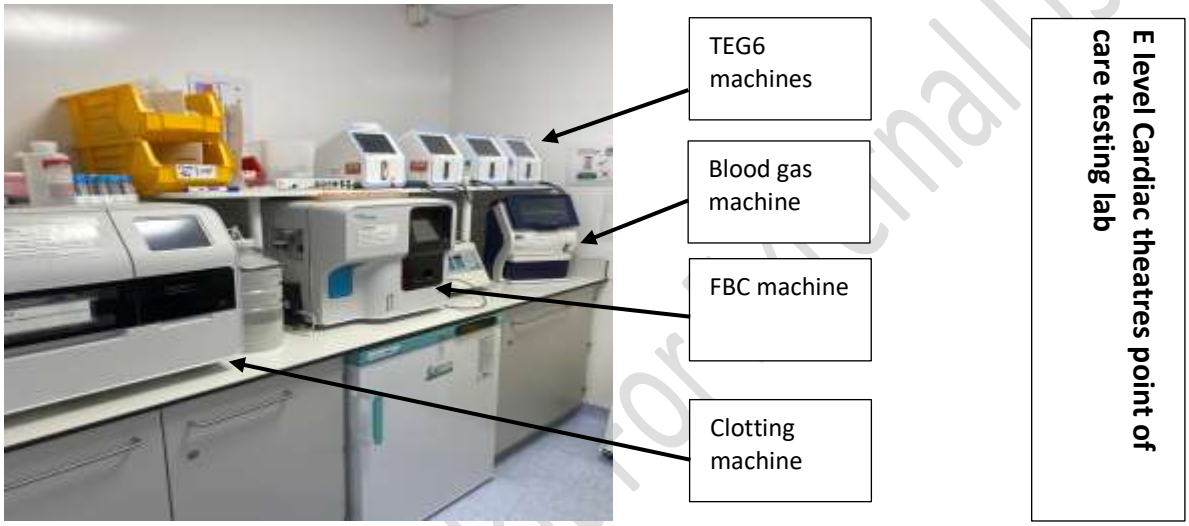
Procedures and Investigations

TEG and Near Patient Coagulation Testing

Author: Dr K Wilkinson *Revised: December 2022*

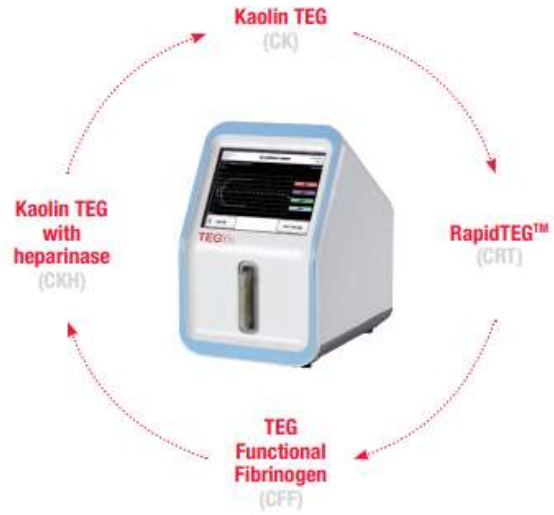
Point-of-care haematology and coagulation testing facilities on Cardiac ICU

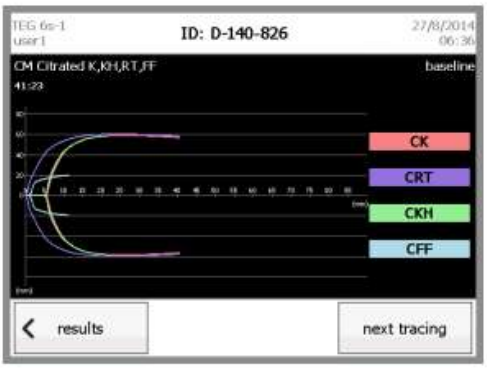
In the cardiac laboratory outside CICU and up in E level cardiac theatres (next to theatre B), we have machines to perform TEGs, as well as point-of-care testing for full blood counts and conventional coagulation screens (INR, APTR, and fibrinogen). Teaching regarding the use of these machines is provided through the perfusion department. Please do not use them unless you have been taught to do so.

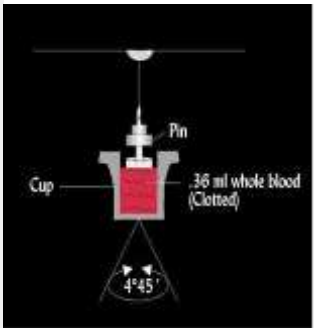


Principles of TEG

TEG measures the whole process of blood coagulation by using the viscoelastic changes of blood that are associated with fibrin polymerisation. The TEG machine used at UHS currently is TEG6s.

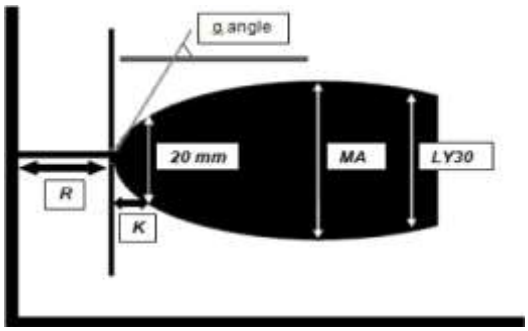






- TEG® measures the physical properties of the clot in whole blood via a pin suspended in a cup (heated to 37C) from a torsion wire connected with a mechanical-electrical transducer
- The elasticity and strength of the developing clot changes the rotation of the pin, which is converted into electrical signals that a computer uses to create graphical and numerical output
- A plot of the movement of the inner cylinder is known as a thrombelastogram.
- point of care test (quick, takes around 30min)
- can be repeated easily and compared
- requires calibration 2-3 times daily
- should be performed by trained personnel
- susceptible to technical variations
- kaolin and more recently kaolin + tissue factor (TF) (RapidTEG®) are used as activators, NATEM (TEG®using native whole blood) is slower
- other tests are available including functional fibrinogen, a measure of fibrin-based clot function, and Multiplate which evaluates platelet function

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.

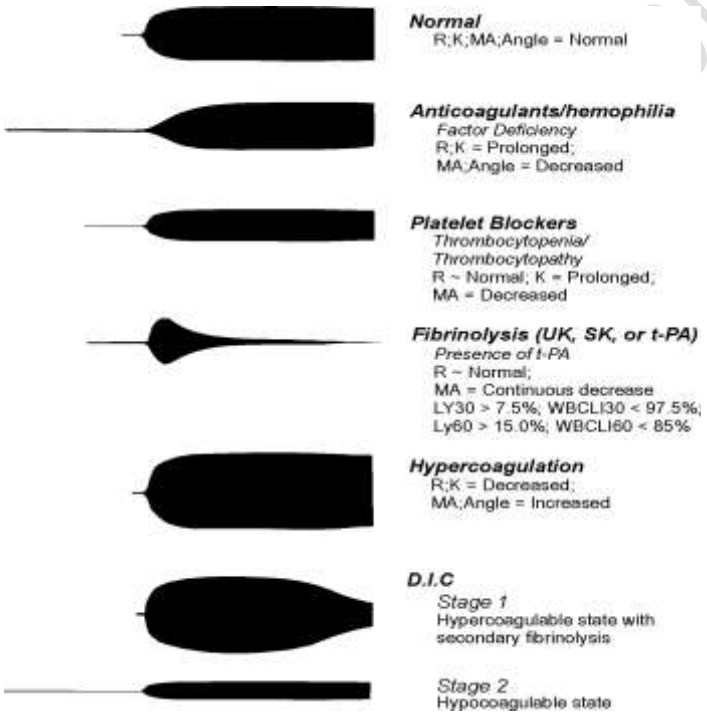


Schematic TEG waveform

What information does TEG give us?

Parameter	What it 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 Anticoagulation	Hypercoagulability syndromes

		Severe hypofibrinogenaemia Severe thrombocytopaenia	
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 Thrombocytopenia Thrombocytopathy Hypofibrinogenaemia	Hypercoagulability state
Angle (α)	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 Thrombocytopathy 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

TEG AS A GUIDE TO TREATMENT

- Increased R time => FFP
- Decreased alpha angle => cryoprecipitate
- Decreased MA => platelets (consider DDAVP)
- Fibrinolysis => tranexamic acid (or aprotinin or aminocaproic acid)

Platelet function mapping with TEG6s

- Determines the MA (clot strength) and the level of inhibition caused by antiplatelet therapy.
- Useful to perform pre-bypass if the patient has received any antiplatelet drugs prior to coming to theatre. You can assess the platelet function to guide the need for platelet transfusion.
- Full TEG Platelet Mapping assay kit provides information about platelets through four different whole blood tests.
- A Kaolin activated sample produces a strong thrombin response to maximally activate all platelets and cleave all available fibrinogen demonstrating the underlying potential for maximum clot strength (MA Thrombin).
- A second assay blocks all thrombin and uses a special activator to demonstrate the clot strength coming from fibrin (MAA).
- The 3rd and 4th assays also block all thrombin and activate platelets at either the ADP-activated receptor (that thienopyridines like clopidogrel inhibit) or Thromboxane A2 receptor (that aspirin affects), thus demonstrating the clot strength when platelets are activated only through those specific receptors (MAADP or MAAA).
- The degree of inhibition is calculated using the patient's full haemostatic potential as the baseline and contribution of platelets activated through specific receptors, yielding a personalized platelet function analysis.

All these point of care tests can be used in conjunction with the patient's clinical bleeding state to guide management of any clotting abnormalities or overt bleeding.

Cardiac Output Monitoring

Authors: Dr Imran Shahzad *Revised: November 2022*

Previous authors: Dr Andrew Richardson & Dr Kirstin Wilkinson

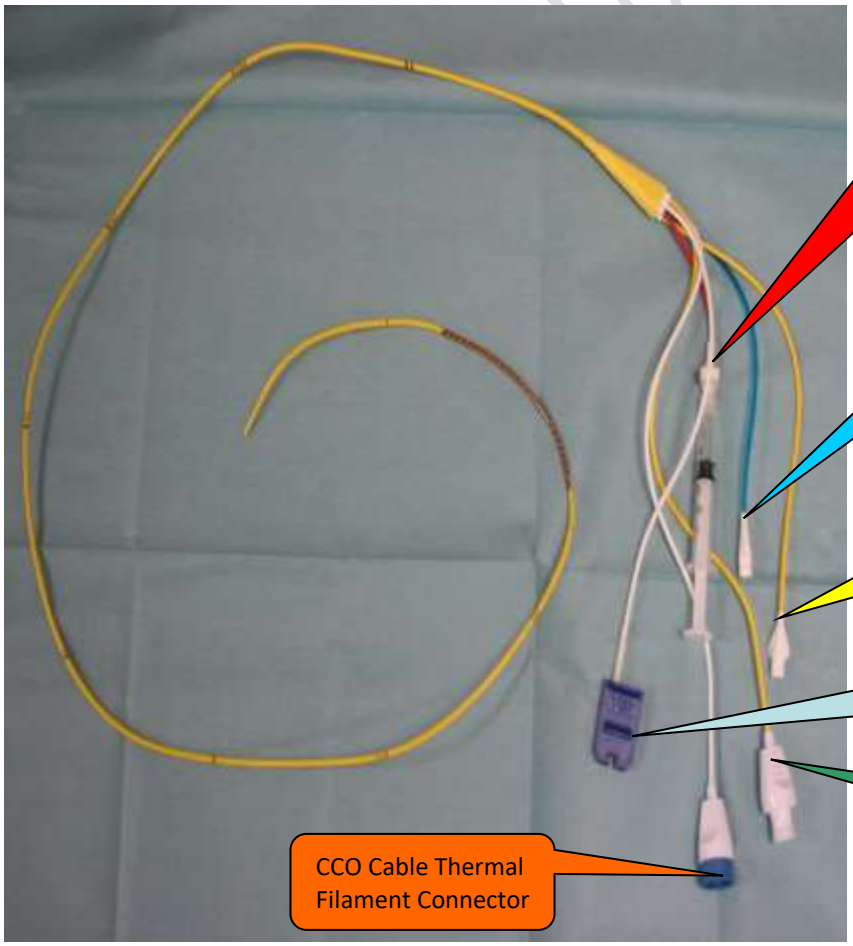
There are numerous methods of cardiac output monitoring. Some are used more frequently than others on cardiac ITU.

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 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

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

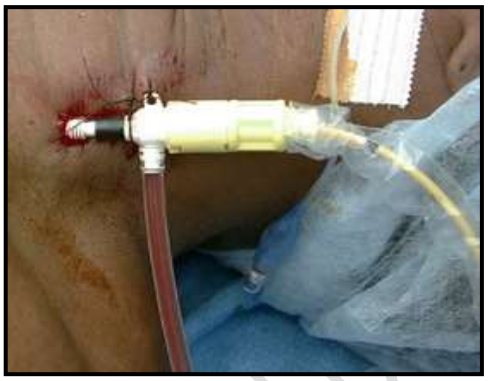
Placement

Placement of PACs is potentially dangerous: it should not be attempted without discussion with the consultant on call. You must be fully trained to insert one unsupervised. If the patient is sick enough to require a PAC, they require senior review.

You will need a properly trained nurse to assist with setting up the equipment. 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.

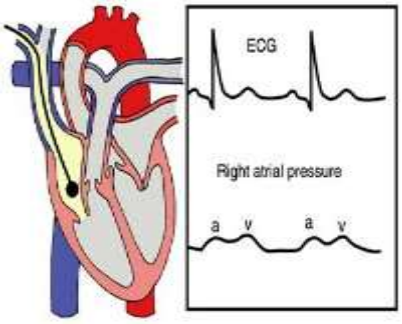
A PA introducer is inserted into a central vein with the same technique as for any central venous cannulation.

Ideally, the right internal jugular or left subclavian veins are cannulated as these allow for the easiest passage of the PAC.

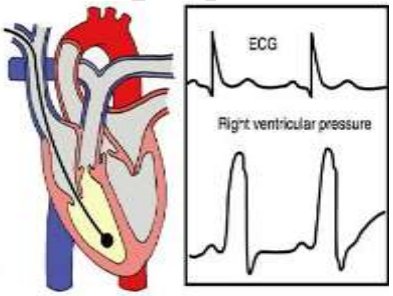


A third transducer is set up and zeroed. The PAC is removed from its packaging in a sterile fashion. Place (but do not unfold) the sterile catheter sheath over the PAC at this stage before it is forgotten. Having to remove a PAC to apply the sheath after inserting a PAC perfectly on the first attempt 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.

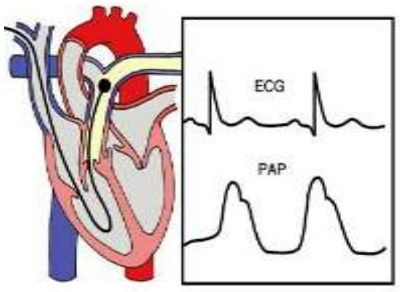
Waveforms seen during insertion



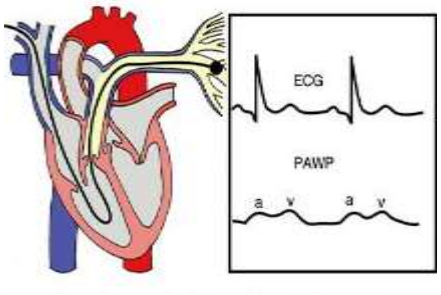
Right Atrium
Classical CVP waveform
Usually at 20 – 25 cm



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 or HemoSphere cardiac output monitoring system, and the SvO_2 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 based on a number!

Cardiac output or index (cardiac output adjusted for body surface area)
- Normal CI 2.7 – 4 l/min/m^2
- CI > 2.0 l/min/m^2 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_2
- Normal > 60%

Systemic Vascular Resistance
- Normal 770 – 1500 dyne s/cm-5

Pulmonary Vascular Resistance
- Normal 100 – 250 dyne s/cm-5

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 several factors:

- the compliance of the aorta is not a linear relationship between pressure and volume.
- the pulse pressure measured from an arterial trace is the sum of two waves – the incident pressure wave ejected from the heart, and the reflected wave from the periphery.
- 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 assumes 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 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

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

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.

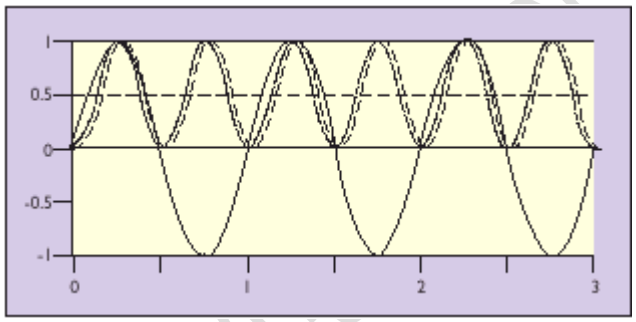


Figure 1

Estimation of the heartbeat 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 auto correlogram 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 Vmax 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 Vmax.

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.

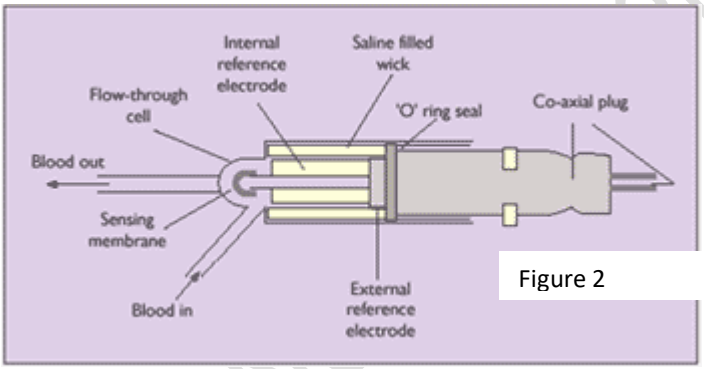
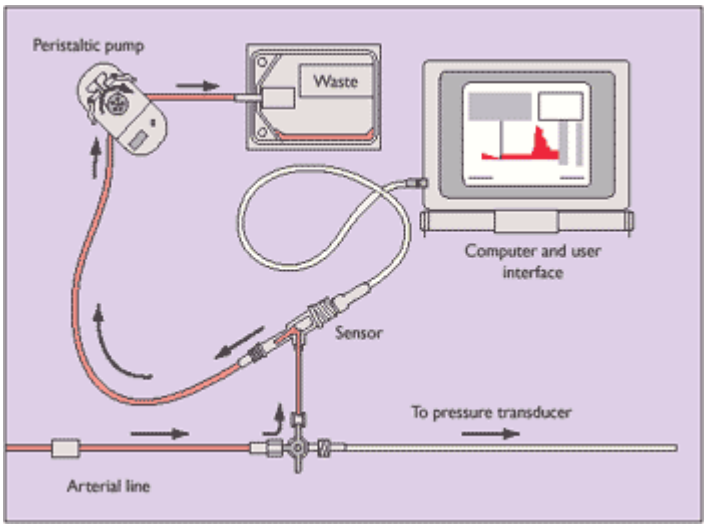


Figure 2

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.



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

Cardiac output = Lithium dose (in mmol) * 60 / Area * (1-PCV) (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 must 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.

Cardiac output calculation with echocardiography

Echocardiography has evolved into safer and simpler alternative for measuring cardiac output. Although TOE is not non-invasive (TTE is non-invasive), is used to evaluate cardiac structures and their function. The ability to measure cardiac output gives it an additional advantage.

CO measurements are based on the continuity equation.

SV = area LVOT x VTI LVOT CO= HR x SV

(SV stroke volume, LVOT left ventricular outflow tract, VTI velocity time integral)

In the absence of valvular dysfunction or intracardiac shunt blood flow is constant throughout the heart. Based on this assumption, CO is equal to the forward flow across each of the cardiac valves. Although any cardiac structure that has a measurable cross-sectional area (CSA) may be used, most commonly the left ventricular outflow track (LVOT) is used because its cross section is essentially a circle, unlike other structures (i.e. mitral valve annulus, aortic valve or tricuspid annulus, etc.). The first step in measuring CO with echocardiography is to determine stroke volume.

To understand the principle of Doppler technique, consider an example of blood vessel as a cylinder. The volume of blood that moves from one point to other will be equal to the volume in the blood vessel at that point. It can be calculated by using the following equation.

Volume = cross-sectional area of cylinder x length

$= \pi r^2 \times L$

Where r radius and L is distance.

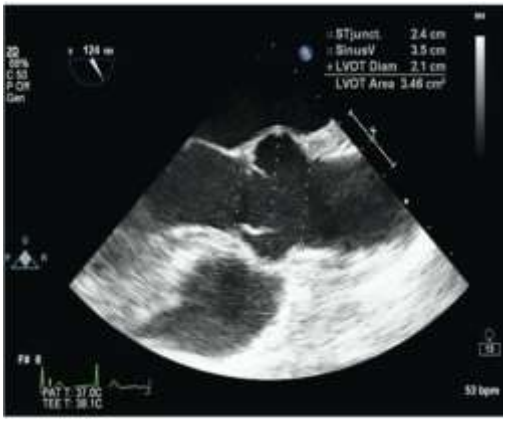
The same principle is used to measure blood flow across cardiac structures. The cross-sectional area can be measured by 2D imaging. If blood flow remains laminar and velocity is constant, then distance can be measured by the following equation.

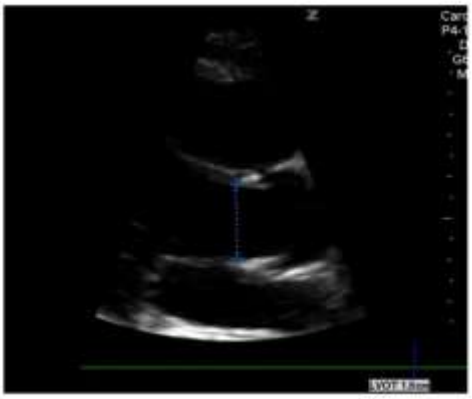
L = velocity x time

But the blood flow in the cardiovascular system is pulsatile and velocity is not constant and to measure blood flow in such conditions, mean velocity should be measured when flow through that structure occurs. This is calculated as VTI (velocity time integral) by the software and is equivalent to the product of mean velocity and duration of flow.

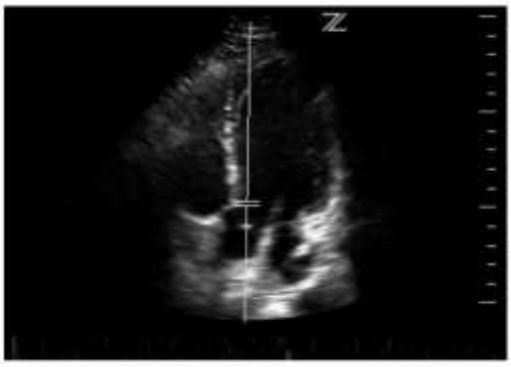
Work out the volume of the cylinder – multiply the area of the LVOT (a circle) by the length the blood travels and you get the stroke volume (i.e. volume ejected per beat) The stroke volume multiplied by the heart rate gives us the cardiac output (expressed as L/Min).

LVOT diameter is measured in ME aortic valve long axis in TOE (parasternal long axis in TTE). It is measured as the distance between hinge points of aortic valve leaflets during early systole when valve leaflets are fully open.





LVOT VTI is measured in deep trans gastric or trans gastric long axis view in TOE (apical five chamber or three chamber views in TTE) because ultrasound beam is perpendicular to the direction of blood flow. Pulsed wave sample is placed approximately 5mm proximal to aortic valve and correct position can be confirmed by closing click on spectral doppler display. VTI is derived by tracing the outer border of the signal.



LVOT being relatively circular in shape and nearly constant in size during cardiac cycle provides advantage of being more accurate in measuring cardiac output. Nonetheless, in the presence of significant aortic regurgitation, increased blood flow through LVOT may not be accurate representative of cardiac output. Similarly, increased velocity during LVOT obstruction may not represent true cardiac output.

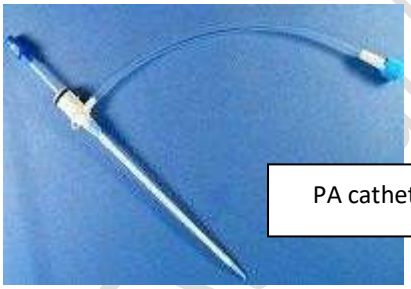
Line Insertion and Documentation

Author: Dr Vivek Koul *Revised: January 2023*

Previous authors: Dr Andrew Richardson & Dr Kirstin Wilkinson

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



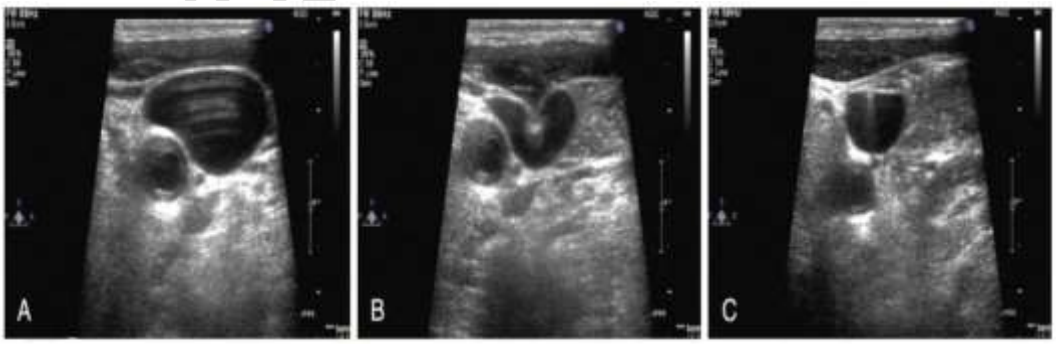
PA catheter introducer

However, like all medical interventions, there are several 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:



Transverse plane ultrasound image showing the right internal jugular vein and its typical anatomic position anterior and lateral to the right common carotid artery. **B**, Needle entering the right internal jugular vein. It is essential that the operator directly visualize the needle entering the vessel lumen, as shown here, to avoid inadvertent puncture of the posterior wall of the vein. **C**, The wire is seen as an echodense structure within the vessel lumen. Confirmation of the location of the wire should always precede the use of the vessel dilator.

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.

2. 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)

We try to avoid the subclavian route for the placement of haemodialysis catheters, as the line frequently gets pinched between the clavicle and 1st 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).

3. 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. Alternative lines are available for such cases. 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.

4. Skin should be prepared with chlorhexidine (e.g. 'Chloraprep' sticks). 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. If there is chlorhexidine allergy, please use povidone-iodine solutions.

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

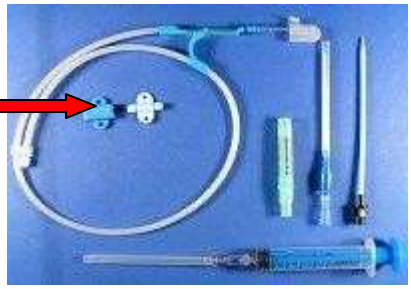
6. Awake or very lightly sedated patients should have local anaesthetic infiltration prior to starting the insertion procedure.

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

8. **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.

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

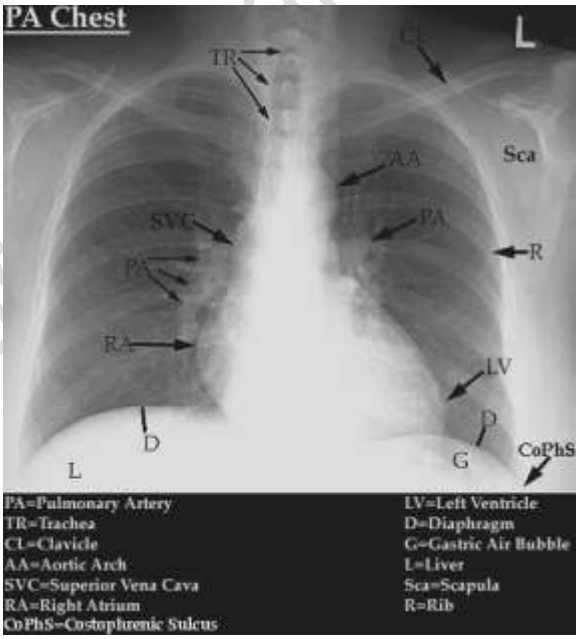
The line itself must be sutured to the skin not just the clips



10. A sterile transparent dressing should be applied that allows visualisation of the insertion site forsigns of infection.

11. A chest radiograph should be ordered for all attempted central line insertions, primarily to excludecomplications 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 (± standard deviation) from the carina to the cavo-atrial junction 40.3 mm ± 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: avoid subclavian approach where possible, limit depthof needle insertion, choose the right side rather than left where subclavian approach needed, avoid multiple attempts.
    - Management: Check post-procedurex-ray, 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 ofinotropes through a line in the carotid artery is a catastrophe)
- Bilateral Iatrogenic complications
    - Prevention: if attempted catheterization is unsuccessful, try the ipsilateral internaljugular or subclavicular approach before trying contralateral subclavian catheterization
- Catheter embolisation
    - Prevention: **never** withdraw a catheter past a needle bevel which might shear offthe 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 (increaseof 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 myocardiumof the right atrium)
    - Management: withdraw the guidewire, reposition the catheter; treat dysrhythmia according to ALS protocols.
- Air embolism
    - Prevention: maintain a Trendelenburg 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.

Chronic patients or long-term patients

- As per our CICU protocols, old CVC line should be removed only after the new CVC line position has been confirmed by waveform, proper aspiration and CXR.
- Always check for any skin colour changes or redness around CVC line area in case any sign of sepsis or rise in inflammatory counts, lines should be changed.
- Scan the site before insertion of CVC, in case of any thrombus, site might have to be changed.
- Patients on intravenous heparin infusion, need infusions to be stopped for at least 2 hoursbefore going ahead with CVC or vascath insertion.
- Consider PICC lines or tunneled catheter lines in those that will need long-term intravenous access.

Documentation in the Medical Record

The following are considered a minimum dataset 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 Metavision by the operator

Please ensure that all the above is recorded on the appropriate form in Metavision or attach the pre-printed sticker that documents the above in the patient's notes.

Percutaneous (temporary) Tracheostomy on CICU

Author: Zoe Sherlock and Dr Kirstin Wilkinson *Revised: March 2023*

Previous author: Dr Nick Goddard

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 FiO2 > 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

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 Consent 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 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 with suitable muscle relaxation. The patient should be fully monitored, especially capnography. The patient should also be on a suitable level of invasive ventilation i.e. not too high FiO2 or PEEP.

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:

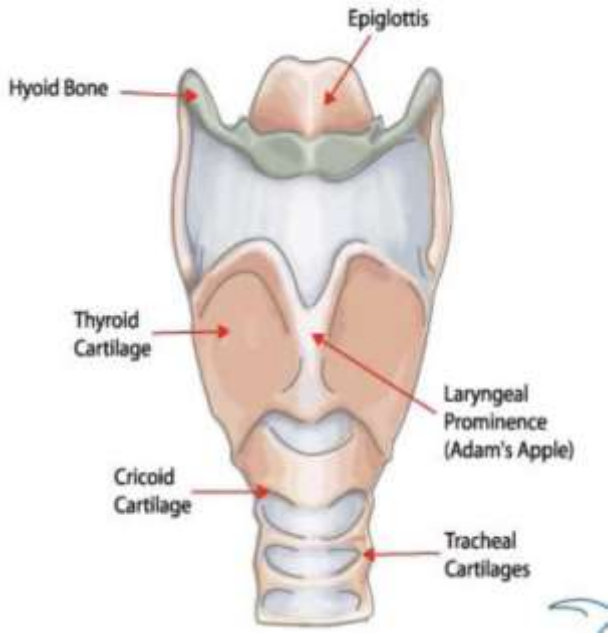
- Sterile field and 2% chlorhexidine (skin preparation)
- Lubricating jelly (for tracheostomy tube) and sterile water (for lubricating the dilator)
- Local anaesthetic with adrenaline
- Fibreoptic bronchoscope and catheter mount to accept scope – usually performed by the ICU technicians
- Replacement endotracheal tubes plus the airway trolley

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 ultrasound should be used to ensure there are no overlying major blood vessels.

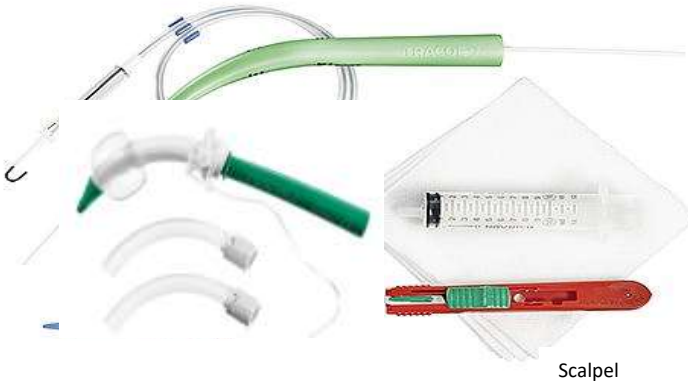
The larynx and cricoid cartilage with intervening cricothyroid membrane are identified. From the cricoid, moving caudally, the tracheal rings are identified (this may also be achieved using ultrasound). Placing the airway any higher next to the cricoid can cause tracheal erosion and long-term problems. The tracheostomy should be inserted between the 2nd and 3rd tracheal rings.



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 insertion is made into the sidewall of the trachea, life-threatening airway disruption may result. This is extremely difficult to remedy. After this, the bronchoscope will monitor dilation of the trachea.

While visualising the airway, local anaesthetic with adrenaline should 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 automatically 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)



Scalpel

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 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
11. End tidal CO2 must be seen
12. CXR to confirm position with written confirmation.

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

Tips

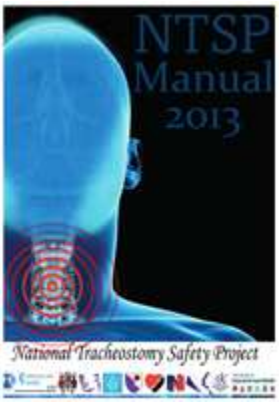
- If the dilator does not pass easily, examine the initial incision. Often, it is the skin that impedes progress, and the incision must be slightly widened with the scalpel. Keep in mind that the minimal incision necessary (sometimes no incision) for a tight-fitting tube will better avoid infection.
- 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

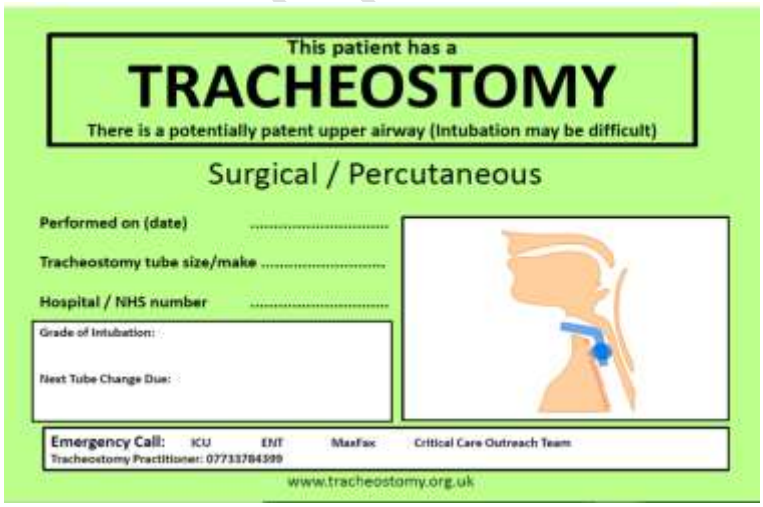
General management of tracheostomies

The most recent policy v1.0 for the management of adult patients with a tracheostomy at UHS is available on the Staffnet. This should be consulted for the safe management of all patients at UHS with tracheostomies.

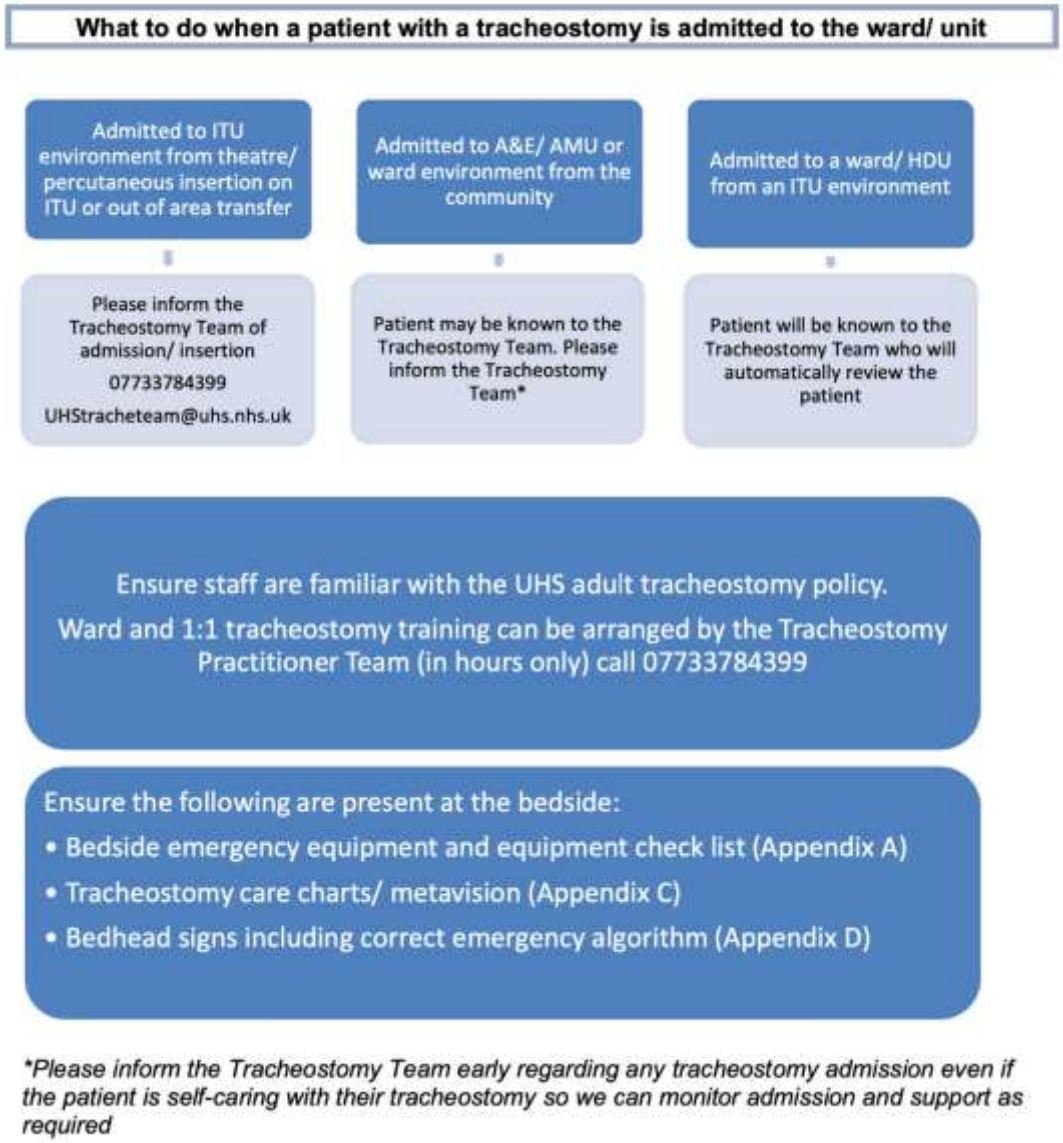
NAP4 (2011) as well as 'On the Right Trach' from NCEPOD (2014). [3, 4] have highlighted the problems associated with tracheostomies. In 2013 in the UK, the national tracheostomy safety project (NTSP) produced a national manual to improve and standardise training and practice the national tracheostomy safety project (NTSP). [8] This manual was 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 NTSP manual is the current standard of care [10].

The new UHS tracheostomy policy has comprehensive guidelines for the management of patients with tracheostomies. Much of the document's information has been incorporated into this section. However, please see document for full UHS guidelines.





4 Quick Reference Guide



**Please inform the Tracheostomy Team early regarding any tracheostomy admission even if the patient is self-caring with their tracheostomy so we can monitor admission and support as required*

Tracheostomy Emergencies

A patient with a tracheostomy is at risk of death or harm if inappropriate or inadequate care is provided. This patient population requires tracheostomies to be inserted safely, securely positioned and appropriately cared for. Failure to do so may result in a displaced or blocked with and may be fatal within minutes.

The NPSA have previously described 53 tracheostomy-related incidents from UK critical care units 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.

CICU bedside airway trays:

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

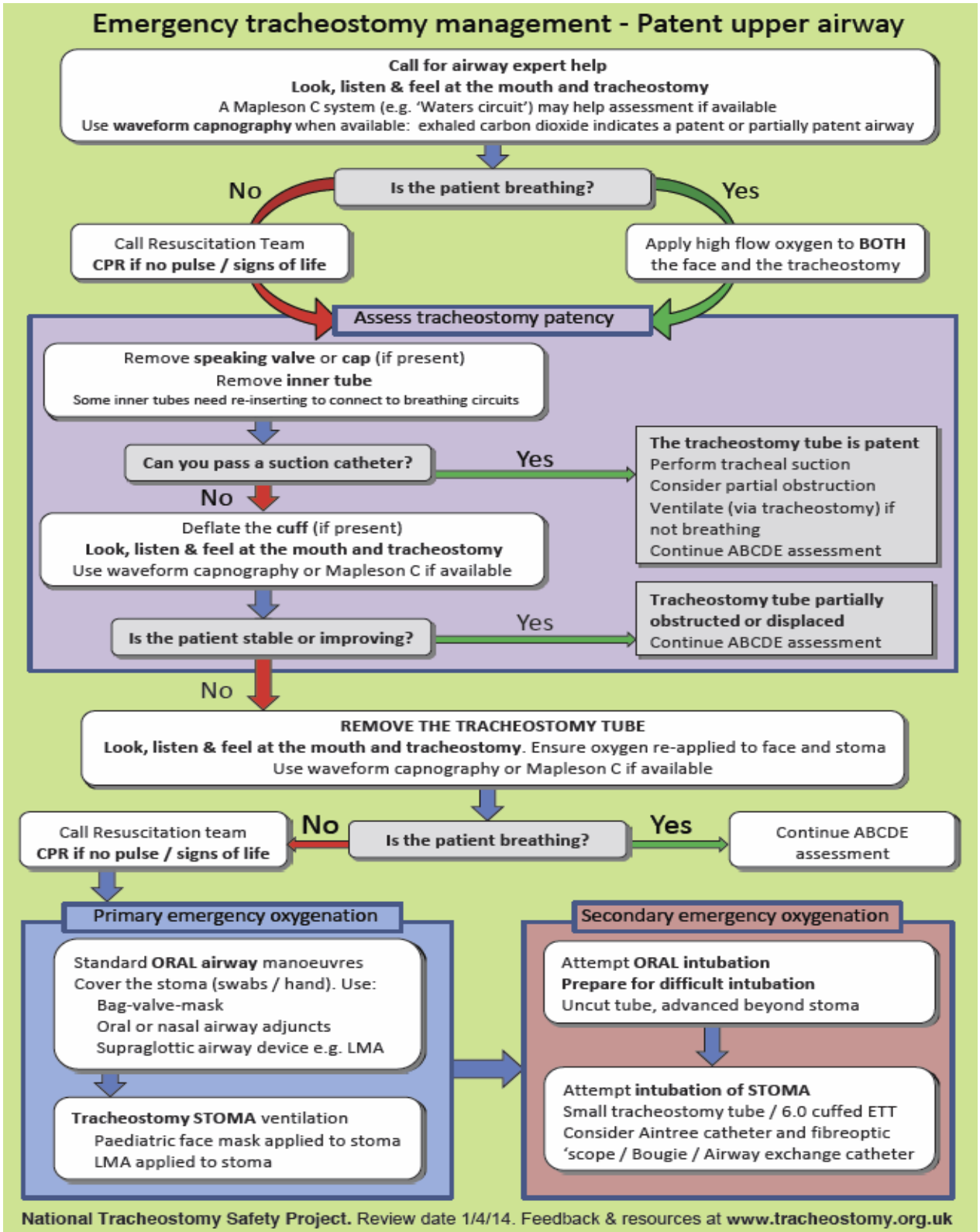
- Scissors + stitch cutter
- Lubricating jelly
- 10 mL syringe
- Spare tracheostomy tubes (same size + one smaller plus spare inner + suction tubes
- Tracheostomy dressing + tapes
- Tracheal dilators
- Size 6.0 ETT (uncut +/- armoured)
- Spare oxygen 'nipple' (to provide simultaneous 02 via face and tracheostomy)
- Occlusive dressing if tracheostomy to be removed
- Tracheostomy disconnection wedge (arguably not essential)

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 fibreoptic 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/plus):	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 fibreoptic bronchoscopy should be immediately available. Monitoring, positioning, preparation and anaesthesia are the same as for primary insertion.


Emergency tracheostomy management - Patent upper airway
Call for airway expert help
Look, listen & feel at the mouth and tracheostomy
A Mapleson C system (e.g. 'Waters circuit') may help assessment if available
Use waveform capnography when available: exhaled carbon dioxide indicates a patent or partially patent airway
Is the patient breathing?
No
Yes
Call Resuscitation Team
CPR if no pulse / signs of life
Apply high flow oxygen to BOTH the face and the tracheostomy
Assess tracheostomy patency
Remove speaking valve or cap (if present)
Remove inner tube
Some inner tubes need re-inserting to connect to breathing circuits
Can you pass a suction catheter?
Yes
The tracheostomy tube is patent
Perform tracheal suction
Consider partial obstruction
Ventilate (via tracheostomy) if not breathing
Continue ABCDE assessment
No
Deflate the cuff (if present)
Look, listen & feel at the mouth and tracheostomy
Use waveform capnography or Mapleson C if available
Is the patient stable or improving?
Yes
Tracheostomy tube partially obstructed or displaced
Continue ABCDE assessment
No
REMOVE THE TRACHEOSTOMY TUBE
Look, listen & feel at the mouth and tracheostomy. Ensure oxygen re-applied to face and stoma
Use waveform capnography or Mapleson C if available
Call Resuscitation team
CPR if no pulse / signs of life
No
Is the patient breathing?
Yes
Continue ABCDE assessment
Primary emergency oxygenation
Standard ORAL airway manoeuvres
Cover the stoma (swabs / hand). Use:
Bag-valve-mask
Oral or nasal airway adjuncts
Supraglottic airway device e.g. LMA
Tracheostomy STOMA ventilation
Paediatric face mask applied to stoma
LMA applied to stoma
Secondary emergency oxygenation
Attempt ORAL intubation
Prepare for difficult intubation
Uncut tube, advanced beyond stoma
Attempt intubation of STOMA
Small tracheostomy tube / 6.0 cuffed ETT
Consider Aintree catheter and fibreoptic 'scope / Bougie / Airway exchange catheter
National Tracheostomy Safety Project. Review date 1/4/14. Feedback & resources at www.tracheostomy.org.uk

Speaking Valves

A variety of speaking valves used within this Trust including the Aqua by Passy-Muir® https://www.passy-muir.com/valves_page/ that can be used in the ventilator circuit, and the Shiley Phonate valves or the Insight ones from Atos https://www.atos-care.co.uk/product/insight-tracheostomy-speaking-valve/

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, SpO2, accessory muscle use, increased work of breathing.

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. Weaning and decannulation should be a multi-disciplinary decision.

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, Fi02 < 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 can **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 H20 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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6. University Hospital Southampton NHS Foundation Trust Tracheostomy Guidelines for the Adult Patient (Adapted from the St. George's Healthcare NHS Trust Tracheostomy guidelines 2011). Appendix A, 2012. Approved Committee: Nursing and Midwifery Group; Patient Safety Steering Group 21st November 2014. Available on UHSFT Trust intranet.
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The National Tracheostomy Safety Project – Medical Resources

Available online at: http://www.tracheostomy.org.uk/Templates/Resources.html

Intra-Aortic Balloon Pump (IABP)

Author: Thomas Hampshire *October 2022*

Previous versions: by Drs Pierce & Wilkinson

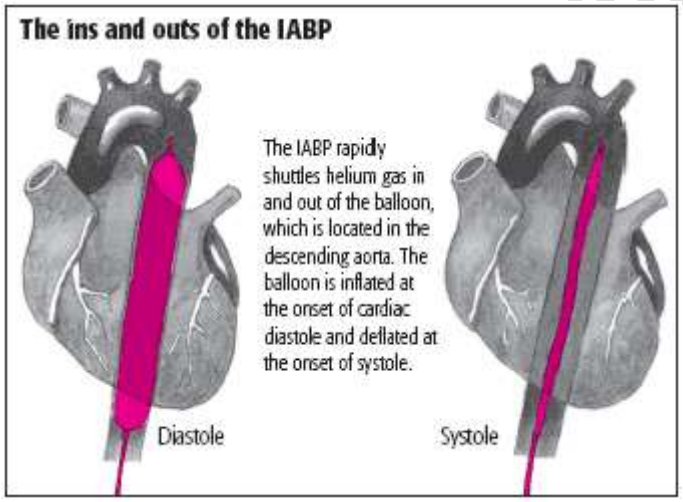
Sometimes referred to as intra-aortic balloon counter-pulsation, intra-aortic balloon pumps aim to help increase myocardial oxygen supply and reduce oxygen demand. They may be placed in patients who have resistant ischaemia before surgery or can be inserted intra or postoperatively. You will gain most 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 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. 3 sizes of balloon: 34cc for 152cm to 162cm height, 40cc or 50cc for heights 162cm or above. Driven by helium. Contain a pressure channel for aortic pressure measurement (either fibre optic or direct pressure via manometer line). Pressure is measured from the balloon tip.

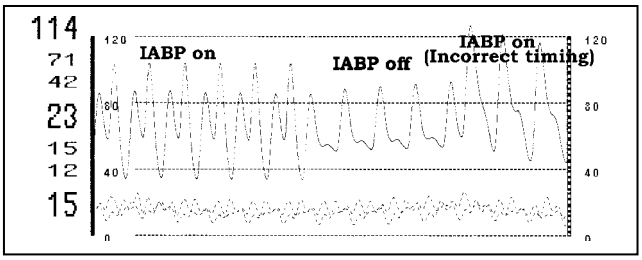
Timing

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 ⇑
- Assisted aortic end diastolic pressure ⇓
- Patient's own systolic BP⇓
- Mean arterial pressure ⇑
- Coronary perfusion pressure ⇑⇑
- 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)

More information and training regarding Intra-aortic balloon pumps is available here:

https://getinge.training/iabp-int/

Focused Ultrasound on CICU

Author: Dr Sean R Bennett *December 2022*

Previous authors: Drs P Diprose and K Wilkinson

Patient examination on the CICU should include the consideration of ultrasound/echocardiography. Just as ultrasound for central line placement shows you what lies beneath, so does ultrasound for the heart and lungs. Anything you think you detect with the stethoscope and chest X-ray is better defined with ultrasound. The use of bedside ultrasound by the intensive care doctor started in the late 1990s and with the format FATE (Focused Assessed Thoracic Echo) increased worldwide.[1] However, the anatomical echo windows used for this, and all the other protocols are the same, it is purely a matter of the question relevant to the patient and the scope of the examination required. For nearly a decade FICE (Focused Intensive Care Echo) has been proposed in the UK as a slimmed down version of FATE. The ICS website has modules for the use of ultrasound on heart, haemodynamics, TOE and lung under the banner of FUSIC.[2]

Here is presented a practical guide to using ultrasound and then consider what modality is best for the patient. You will always have a question to answer and always include the information from other monitors. Always document your findings on the clinical information system.

Low blood pressure. Haemodynamic instability. Unexpected increase or use of vasoactive support.

No CICU patient should have a SBP <90mmHg. There is a simple paradigm of, poor contractility, hypovolaemia and vasodilation which can be resolved with echo.[3]

A- What is the left and right ventricular (LV and RV) function?

B- What is the size of the chambers of the heart? Left atrium should be < 5cm, Left Ventricular End Diastolic Diameter (LVEDD) should be <5cm. The RV should be smaller than the LV and the RV free wall should be moving towards the septum with an objective measure using Tricuspid Annular Plane Systolic Excursion (TAPSE).

C- Pericardial Effusion. Even a good LV can succumb to the pressure of an effusion. The size is less relevant than the pressure generated. Therefore, a rapid effusion of 1.5 cm will be more significant than a 7cm chronic effusion. The purpose of echo is to refine the diagnosis. You are trying to manage an effusion before it becomes a tamponade situation. This was the primary reason for echo on CICU in the 1980s.

D- Volume status is difficult unless it is extreme and along with CVP requires examination after a trial of treatment. Assess the IVC along with the chamber size. The IVC will appear dilated in most ventilated patients but should have some respiratory variation. Easier to assess in the non-ventilated patient. Leg-raise with assessment before and after is considered as accurate as measuring pulse wave variation. Fluid overload is accompanied by B-lines on Lung Ultrasound (LUS) and often pleural effusions.

E- In the first 24hrs post-op, low BP is bleeding until proved otherwise and the above plus other observations will provide the answer.

F- Occasionally low BP can be valvular. Check the procedure. At the bedside you can rule in/rule out severe mitral regurgitation (MR), tricuspid regurgitation (TR) and aortic regurgitation (AR).

G- In the pre-operative patient with a history of chest pain always consider the complications of aortic stenosis, myocardial infarction and ascending aneurysm/dissection. Specifically look for VSD and ruptured papillary muscle causing severe MR. Look for dilated aorta with AR, left pleural effusion and dissection flap.

Infection. Suspected endocarditis.

Sepsis in addition to vasodilation will cause ventricular dysfunction. Endocarditis causing structural damage will cause regurgitation. Any abnormal valve is likely to become infected and in drug abusers TR occurs in normal valves.

1. Placement of lines for ECMO and weaning ECMO

2. The lungs: The most practical application of LUS is again rule in/rule out:

A- Pneumothorax- look for lung sliding, stratosphere sign, seashore sign, lung point, A-lines.

B- Effusions- measure extent and nature in the patient context.
C- Fluid- B-lines are a sensitive measure of fluid overload in the appropriate areas.
D- Consolidation. Difficult with chest X-ray. With ultrasound it is obvious.

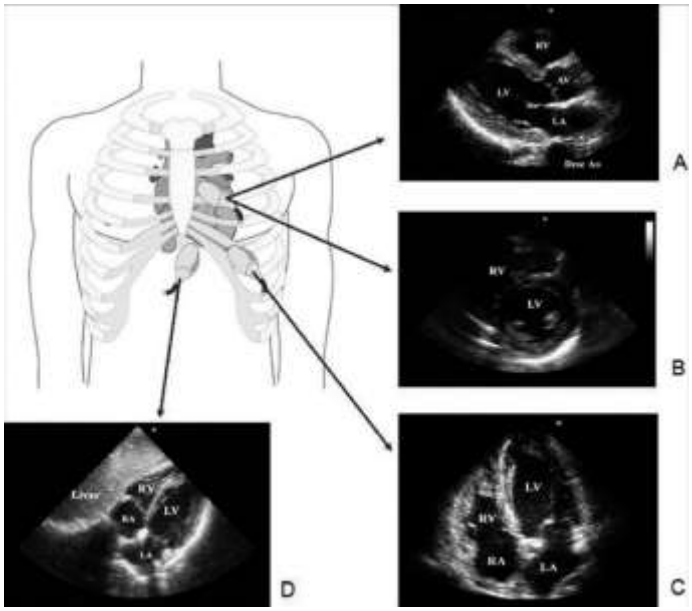
Should I use TOE or TTE?

Since the collaboration of the Association of Cardiac Anaesthesia and Critical Care (ACTACC) with the British Society of Echo (BSE) more than 20 years ago the concept of combining Trans-oesophageal Echocardiography (TOE) and Trans-thoracic Echocardiography (TTE) in the perioperative period has always existed. As a rule, start non-invasively and work-up if you fail to answer your question. TOE is less readily available, requires at least sedation and there are risks; displacement of NG tubes, localised damage/bleeding to the oropharynx and the oesophagus. There is a risk of serious complications with TOE (including major bleeds and oesophageal perforation) in around 1 in 1300 studies, almost half of these will be fatal.
TTE is rapid and non-invasive. It causes no harm. Studies have shown you only rarely get all the views, but you don't need all the views and can answer your question in about 90% of cases. If you only echo intubated patients, then a lot of patients are not benefiting from echo information. Plus, TTE is easier in non-ventilated patients. It will also confirm when TOE is required and therefore justified. There are some specific areas in which you should progress to TOE:

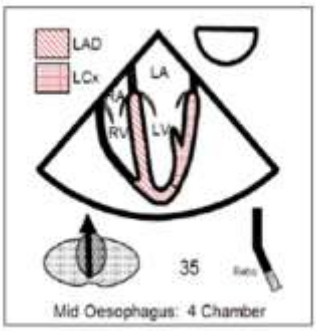
1. Incomplete pericardial effusions. The pressure effect on the Right Atrium can cause hypotension and arrhythmia not seen well with TTE.
2. Suspected endocarditis. Exclusion of endocarditis requires TOE. Confirmed endocarditis requires full inspection of all valves.
3. Mitral regurgitation should have the cause investigated for which TOE is indicated eg. Ruptured papillary muscle, acute myocardial ischaemia.
4. Ascending aortic dissection. Dissection flap is clear on TOE along with inspection of descending aorta.
5. Exclusion of the presence of left atrial thrombus.
6. Placement of IABP. Placement of lines for ECMO can be done with TTE but requires a non-standard lateral approach from the patient's right thorax.

What are the TTE views?

TTE using the FICE protocol (which does not include aortic valve).
A= Parasternal long axis (PLAX).
B= Parasternal short axis (PSAX).
C= Apical 4 chamber long axis (A4chLAX).
D= 4 chamber sub-costal view.



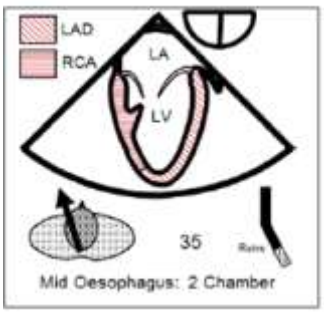
What are the TOE views?[4]



Views 1 + 2 (Mid-Oesophageal 4- and 2-chamber):

1. LV+RV visual assessment. Note apical foreshortening
Regional wall motion abnormality (RWMA)- septal and lateral walls. Pacing prevents interpretation of RWMA.
Shaded area is coded for vascular territory.
Mitral and Tricuspid Valve pathology, effusions and other pathology e.g. VSD

1 and 2. Size of LA >5cm? cause; MR, MS, diastolic dysfunction.
End-diastolic (EDD) and End-systolic diameter/area (ESD) can be measured for EF% although the transgastric view more commonly used for diameter.



2. LV with no apical foreshortening. Inferior wall and anterior wall assessment. Apical thrombus.

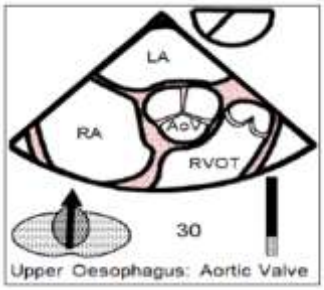
TIP: Ejection fraction is calculated in these views.

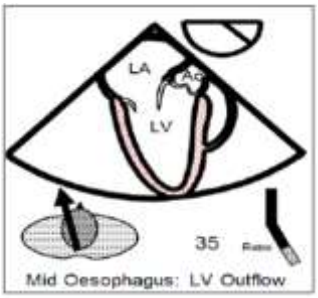
Views 3 AV SAX + 4 AV LAX (Mid-oesophageal):

3. AV SAX all three leaflets, mobility and coaptation. CFD to measure location and quantification of regurgitation vena contracta - severe AR if vena contracta (max jet width) is > 0.6cm.
This view usually includes the tricuspid valve for assessment of TR.

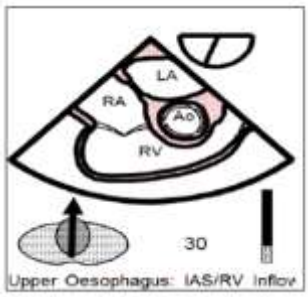
4. AV LAX using CFD in M-mode to quantify AR. Severe AR is ≥ 65% of LVOT diameter, where the LVOT dimension is measured in mid-systole ~ 0.5cm from the aortic annulus.
LV inferolateral and anteroseptal walls.

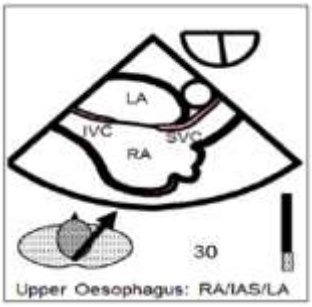
Note AV gradients cannot be measured in these views.





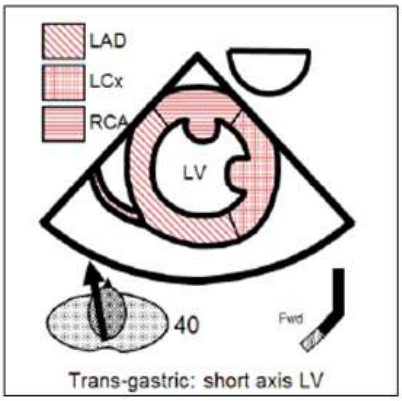
Views 5 RV inflow outflow view (very close to No3) + 6 Bicaval view 90° with probe turned Right:





No 5 RV free wall movement and TAPSE if good alignment
Massive PE may present with acute Cor pulmonale, and signs of RV pressure overload seen as acute dilation.
5,6 Pericardial effusions may be seen to compress RV and RA > 1.5cm may be clinically significant.
No 6. Atrial septal defects, PFO.
Move from 5 to 6 looking at LAA. thrombus.

View 7 Transgastric SAX at the Mid-Papillary level.

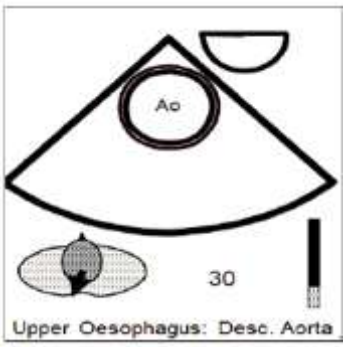


LV function can be assessed by fractional shortening (FS%) +/- use of M-mode or Fractional area change (FAC%) + filling status using LVEDD. In health less than 5.0 cm.
Posterior effusions and circumferential LV collections which may have less effect on CVP.

120° rotation gives the trans gastric long axis view which is used to assess AV gradients. (Considered advanced).

View 8 Descending Aorta SAX and LAX.

Descending aorta dissection. Note distance from incisors. Note atheroma which may be contraindication for IABP.
Monitor insertion of IABP- tip should not advance beyond the subclavian artery. Or should lie just distal to the arch.
Also, placement of ECMO lines.
Any aortic line should have the wire position confirmed in this view when not using X-ray before dilating and insertion of any catheter.



LUS can be widely applied across the thorax with some specific areas according to the question being asked. Specifically, **always locate effusions before chest drain insertion.**

In summary

Ultrasound and echocardiography are essential tools for optimal patient care on CICU. Practise is essential. Both TTE and LUS give the opportunity to learn ultrasound skills without harm. TOE may be extremely valuable but is less often required and requires more training. Interpretation of any echo and documentation should be done by competent practitioners but this should not stop trainees using and becoming competent. If you order a departmental TTE try and do your own assessment first and always attend and watch the echo technician when they do the echo.

Be able to identify cases of hypo/hypervolaemia, LV or RV systolic and diastolic failure, new RWMAs, cardiac effusions, endocarditis, severe AS, MS, MR, TR aortic dissection/aneurysm. Other pathologies include PE, intracardiac masses, pleural effusions, pneumothorax and consolidation. Once the examination is completed, the findings must be documented and made known as part of the patient assessment, either on the CIS or using the form reproduced overleaf.

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Extra-Corporeal Assist Devices & Extra-Corporeal Membrane Oxygenation (ECMO)

Author: Gemma Youdle *Updated: October 2022*

Previous authors: Drs Richardson & Wilkinson

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). VADs can be used to augment perfusion and relieve congestion; potentially reversing the damaging effects of severe heart failure.

VADs are mechanical blood pumps that can provide either left, right or biventricular support. In general, a left ventricular assist device (LVAD) withdraws oxygenated blood from the left atrium or left ventricle and returns it to the aorta. A right ventricular device (RVAD) draws blood from the right atrium or right ventricle and returns it to the pulmonary artery. The output of an LVAD is dependent on sufficient right ventricular function to deliver blood across the lungs into the left heart chambers for the LVAD to pump. Similarly, an RVAD can only provide clinical benefit if the native left ventricle can generate adequate stroke work to cope with the pulmonary blood flow produced by the RVAD.

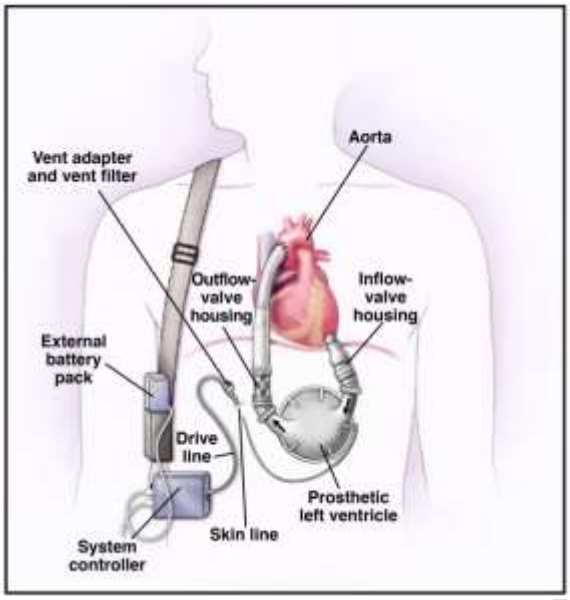
Currently, there is a spectrum of VAD systems available for clinical use including: pulsatile devices, non-pulsatile devices, short term and long-term systems.

Short Term VADs

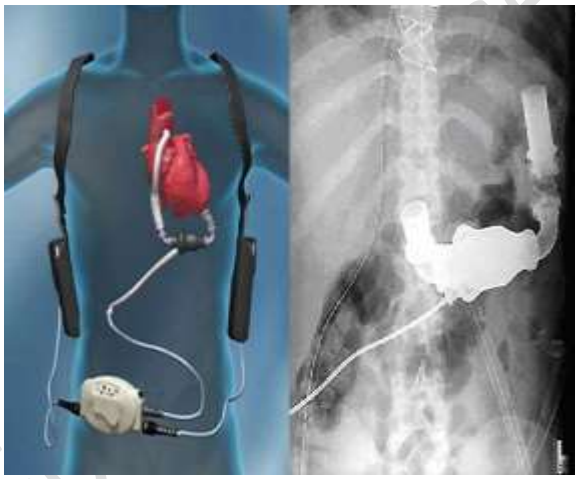
The Levitronix CentriMag is a continuous flow system intended for short term support for up to 30 days. The circuit is comprised of the following components:

Long term VADs

Long term VADs are designed to provide circulatory support for months or years. Continuous flow LVADs such as HeartWare and HeartMate II are implanted within the pericardium. The pump inlet is implanted into the LV apex and the outflow graft is anastomosed to the ascending aorta. These LVADs are driven electrically via a percutaneous driveline, which is connected to a small controller and external energy source. The pumps generate up to 10 L/min of flow, and as flow is continuous most patients have an undetectable peripheral pulse. The Berlin Heart is the only LVAD currently available for long term use in infants.



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. Most patients only require support with an LV assist device; some require RVAD or even BiVAD therapy.

Categories of patients considered for VAD treatment:

- <u>Cardiogenic shock following cardiac surgery</u>
  Cardiogenic shock may occur post-cardiac surgery (although rare <1%), because 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^2, 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 must be placed in the left ventricular apex rather than the left atrium, as poor flow across the mitral valve leads to thrombosis and potential embolization.

Intra-cardiac Shunts

Any intra-cardiac connection (PFO, ASD, VSD, etc) that allows a right-to-left shunt 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 in the setting of intensive care units. ECMO provides a means of supporting blood gas exchange because of the incorporation of a membrane oxygenator, which acts as an artificial alveolar pulmonary capillary system. Cardiac support is achieved by the action of a centrifugal pump in an ECMO circuit, which generates circulatory flow, similar to a VAD.

ECMO has been widely used in the context of severe ARDS. Research data from the CESAR trial suggests ECMO increases survival among adult patients with severe, but potentially reversible respiratory failure; compared with conventional ventilatory support. However, its use for either purpose is generally restricted to certain specialist centres, although is on occasion used at Southampton Hospital.

Indications for ECMO

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

The criteria for cardiac failure are the same as for VAD therapy. However, an LVAD is preferable to ECMO for patients with left ventricular heart failure. This is due to the reduced circuit surface area, which contributes to immune activation. ECMO may be 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 – veno-venous (VV) and veno-arterial (VA).

VV ECMO is generally used for respiratory failure with preserved cardiac function (even if dependant on inotropic support). It is a lung protection ventilator strategy to rest the lungs and provide optimal conditions for recovery of lung function. Blood drains from systemic veins where it passes through the oxygenator and is then returned to the right side of the heart. Blood oxygenated by the ECMO circuit through the native lungs and left side of the heart as normal. Not all of the patients' venous return will pass through the oxygenator; thus, arterial oxygenation may be lower than normal.

VA ECMO is used for severe cardiac failure. It involves the passage of blood from systemic veins, which is then pump through the oxygenator into a systemic artery. VA ECMO support can bypass the native heart and lungs completely, allowing normal blood gases and haemodynamics to be obtained.

There are, however, several potential disadvantages:

1. Complications of surgical cut down and arterial cannulation:
    - Risk of bleeding when heparinised
    - Distal limb ischaemia
    - Risk of gas embolism
2. Retrograde flow from the arterial cannula towards the heart may impose a high afterload, which compromises ventricular function and possibly recovery.
3. Pulmonary perfusion is significantly reduced, potentially impairing recovery from acute lung injury.
4. 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.
5. 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 (drainage) and jugular (return) veins
    - For VA ECMO, peripheral cannulation usually via the jugular vein (venous) and femoral, axillary or carotid arteries (arterial); central cannulation directly via the RA and aorta
- 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_2; determinants of pO_2 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 extracorporeal 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. FiO_2 0.3 – 0.5, PEEP 10 – 15 cm H_2O, 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 $PaCO_2$ 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 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 to isolate the patient 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 should be monitored. Larger clots require a change of pump head, oxygenator or the entire circuit.

Air embolism

- May occur due to excessive negative venous line pressure, partial venous cannula withdrawal exposing a side hole or open central venous catheter ports.
- Small volumes of air 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 in VV ECMO when oxygenated blood from the return cannula is promptly siphoned off by the venous cannula, resulting in shunt and hypoxaemia.
- Revealed by the venous SaO_2 being greater than the patient's 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, change to VV ECMO if cardiac recovery is sufficient, convert to central VA ECMO, or add an additional return cannula to the IJV.

Bleeding

- Minor bleeding is managed by keeping the platelet count > 100,000 mm^3
- Major bleeding requires a higher platelet count (> 150,000 mm^3), 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

Cardio-respiratory 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 ECMO circuit is needed to maintain oxygenation targets, it suggests that the lungs are improving. FiO_2 is gradually weaned down to 21%, and then in combination with invasive ventilatory settings the ECMO 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

6 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 should be reviewed by regular TOE, and haemodynamic parameters assessed for evidence of adequate tissue perfusion. If cardiac function has not recovered in 5 to 7 days, then recovery is unlikely.

Specific Patient Groups on CICU

Medical Patients Following Cardiac Arrest

Author: Prof Charles Deakin *Date: December 2022*

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_2 in the range of 8.0 - 15.0 kPa and $PaCO_2$ 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). In patients refractory to amiodarone, consider the addition of lignocaine (10ml 1% bolus).
- 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 37.5°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 > 37.5 °C) for the first 72 hours post-arrest.
- Measure and record core temperature at least hourly.
- Ensure core temperature is recorded from a suitable core site (NOT axilla).
- If antipyretics are ineffective, use active cooling (tepid sponging, water blankets, cold IV fluids etc.).

Therapeutic Hypothermia

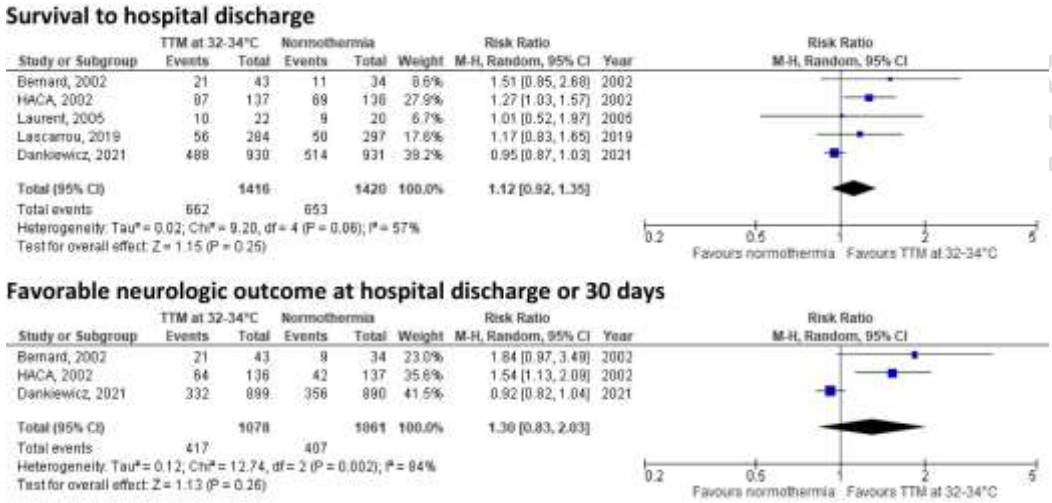
Scientific Evidence

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). Intraoperative therapeutic hypothermia is used routine in cardiac surgery and undoubtedly has neuro and cardioprotective effects. This is of course in the setting where cooling is instigated prior to reducing oxygen delivery to the tissues; the opposite temporal sequence to cardiac arrest.

Targeted temperature management at the wide range between 32 °C and 36 °C has been one of the main therapeutic strategies to improve neurological outcome in post-resuscitation care for the past two decades. This recommendation has been mainly based on 2 small, randomized trials that were published 20 years ago. These showed 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) study included a small subset of patients with in-hospital cardiac arrest. One study documented improved metabolic end points (lactate and O_2 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).

More recently, data derived from the TTM2 (Targeted Hypothermia Versus Targeted Normothermia After Out-of-Hospital Cardiac Arrest) trial, which included 1861 patients, challenge this strategy as it showed no benefit of targeted hypothermia at 33 °C over normothermia at 36 °C to 37.5 °C with fever prevention. The positive effects of temperature management (Core < 37.5 °C) on neuroprotection are possibly mainly achieved by preventing hyperthermia

The meta-analysis below summarises evidence fr0m these combined studies (Granfeldt, A et al. 2021)



Treatment Recommendations

Although there is no conclusive evidence that therapeutic hypothermia is of benefit, it is important to remember that the inflammatory response after cardiac arrest often results in a low-grade pyrexia above 37.5 °C and in these cases, active cooling is warranted.

Current treatment recommendations are being revised, but at present, there is no indication to actively cool patients below 37.0 °C.

- Ensure that unconscious adult patients with spontaneous circulation after cardiac arrest do not have a core temperature that exceeds 37.5 °C. In these patients, DO NOT DELAY INSTIGATION OF COOLING. COMMENCING COOLING IS OF THE UTMOST URGENCY.
- Consider neuromuscular blockers if the patient is shivering.
- Correct hypophosphataemia and hypomagnesaemia.
- Be ready to treat hyperglycaemia as per CTICU protocols.

Methods of cooling

Active cooling can be achieved by several different methods, including simple interventions such as application of ice packs, cooling blankets or gel-adhesive pads with feedback mechanisms, or automated endovascular devices.

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.

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.

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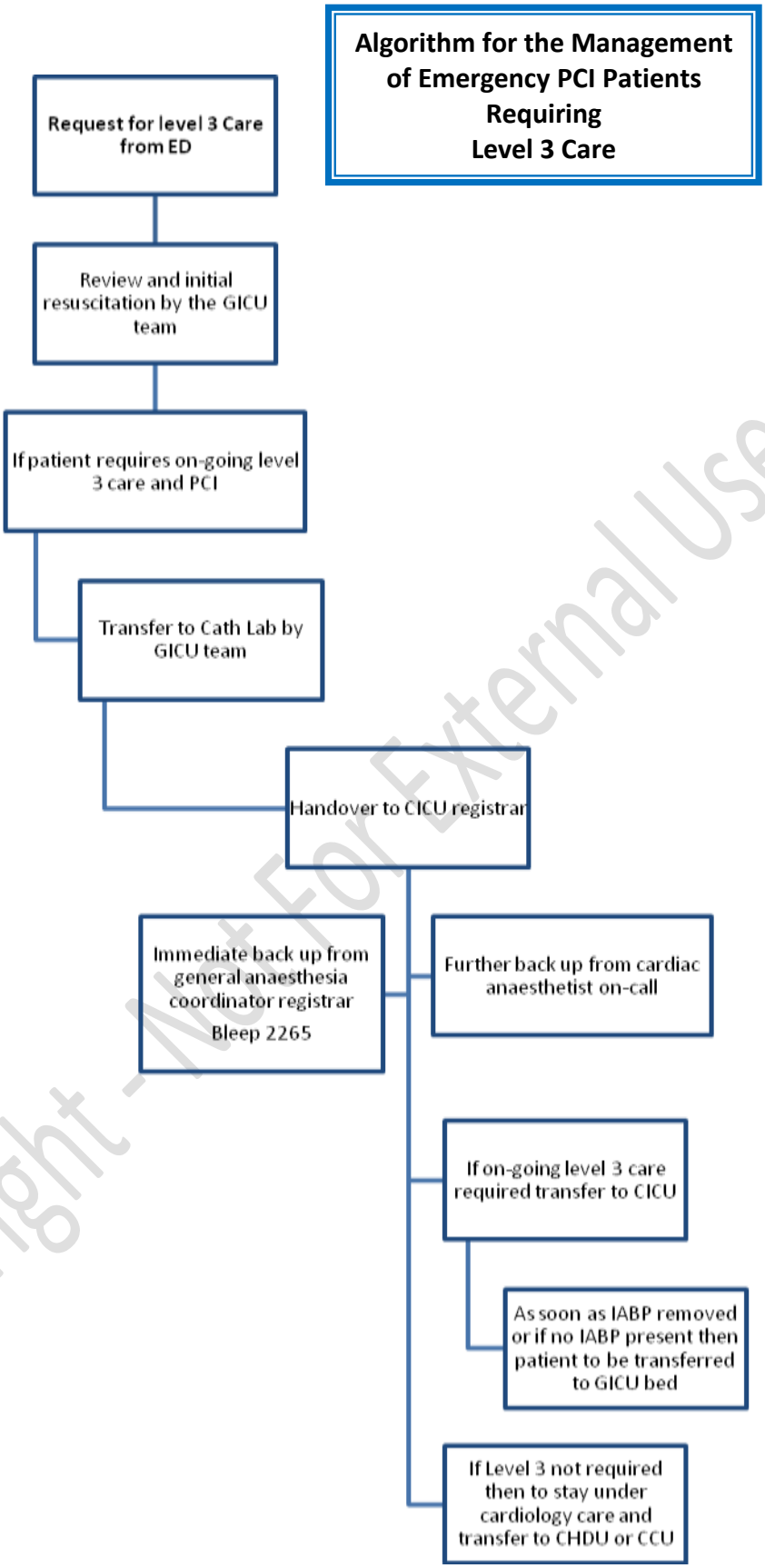
Management of Cardiology Patients requiring Emergency PCI

Author: Dr Paul Diprose *Revised: February 2023*

Patients presenting in cardiac arrest because of an acute coronary syndrome are managed in the Emergency Department (ED) by the ED and General Intensive Care Unit (GICU) teams. If successfully resuscitated, they frequently require emergency percutaneous coronary intervention (PCI). 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/fellow on duty who will take over care after a handover from the GICU team.
    - An ODP will be made available (the cath lab team will contact the theatre coordinator to facilitate this).
    - 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 consultant 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.

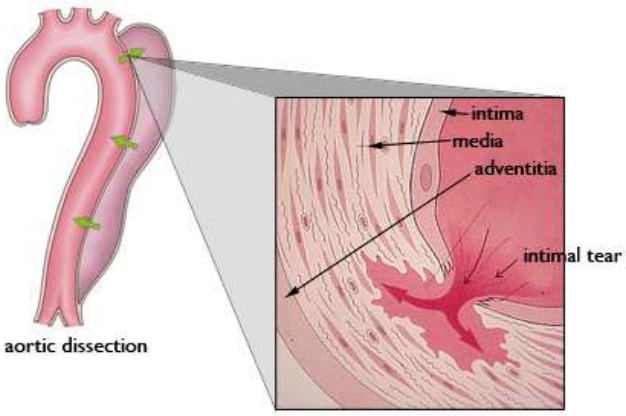


Aortic Dissection

Author: Dr Nick Goddard *Revised: October 2020*

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 comon cardiac surgical emergencies. In contrast Type B dissection is usually managed conservatively.



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)

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).

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

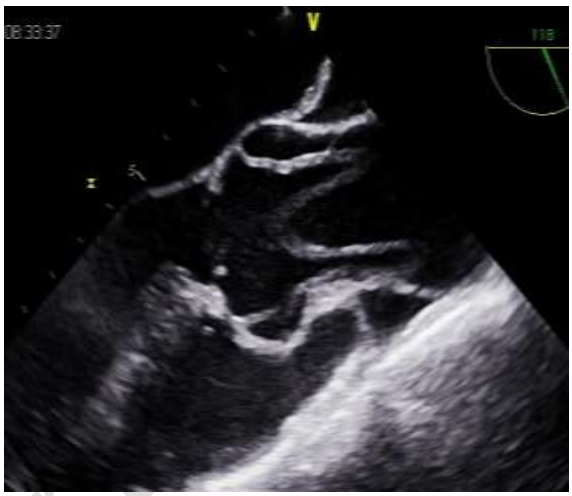
The typical story for acute Type A (< 2 weeks from onset) is central chest pain with or without haemodynamic instability. Instability usually arises following proximal rupture of the adventitia into the pericardium (causing tamponade) or distal rupture into the mediastinum (causing haemorrhagic shock). 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.

Question:	**Role:**
Correct diagnosis?	Alternate diagnoses possible: leaking aneurysm? or intramural haematoma? – *type and timing of surgery will vary*
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 ideally (as right subclavian may be involved in dissection). Label the proximal line red and the distal line white (and/or label on anaesthetic and perfusion monitors CLEARLY)
- PA sheath + CVC
- NIRS monitoring + TOE
- If full stomach – modified RSI (i.e. controlled haemodynamic transition with cricoid)

Consider all line placements awake 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 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) +/- full arch replacement if tear extends distally
- If composite graft used, coronaries would also need to be re-implanted

If cannot get above tear with cross clamp, hypothermic circulatory arrest will be required (remember ice and steroids, although weak/non-existent evidence base for the latter). I target an isoelectric EEG waveform.

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] true 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 to avoid free rupture (not consistent with survival) and any treatment should consider other injuries and their respective management.

For aortic syndrome affecting the descending aorta (and trauma 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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Descending Thoracic and Thoracoabdominal Aneurysm Surgery

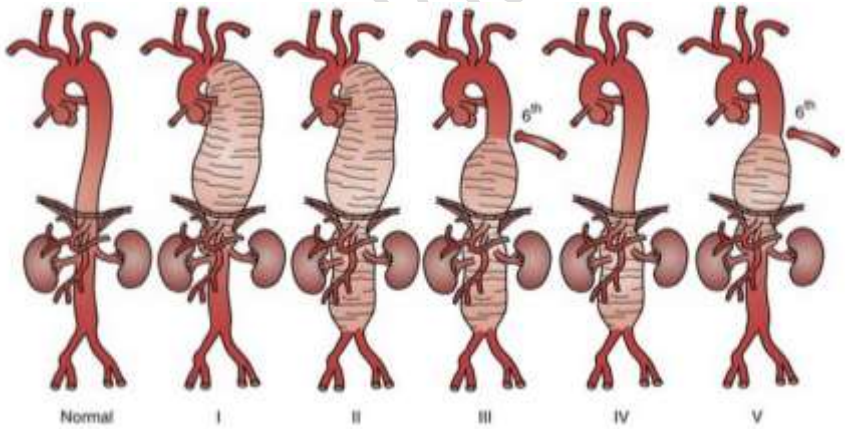
Author: Dr Nick Goddard *Date: Jan 2023*

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 more often managed by cardiothoracic surgeons where access to the chest is required and where cardiopulmonary bypass (CPB) or deep hypothermic circulatory arrest (DHCA) may be needed. As stent technology has improved significantly there is now considerable overlap in care. Patients requiring complex aortic surgery of the descending thoracic aorta may present electively or urgently (with increasing symptoms). They may have had previous aortic root, arch, or thoracic aortic surgery (including type A dissection), and they frequently have concurrent connective tissue syndromes, such as Loeys-Dietz or Marfan syndrome. Patients with connective tissue disease usually are younger, require earlier intervention and surgery is usually more appropriate than stenting. Currently all patients at UHS with thoracic aortic disease are seen in a joint vascular, radiology and cardiothoracic surgical MDT prior to deciding choice and timing of intervention.

Principles of Intra-operative 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 aneuryms distal to the left subclavian: [1]



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

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

Surgical Technique

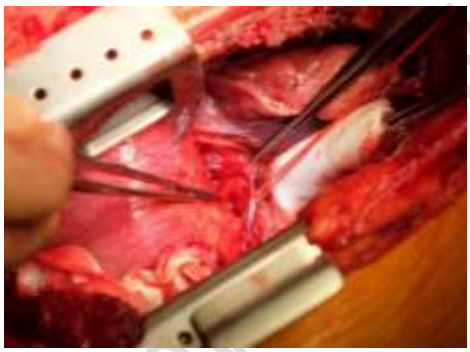
The aim of surgery is to remove the diseased section of aorta and replace it with a synthetic (usually Dacron) graft. Access depends on the location of aneurysm, but a full thoracoabdominal incision is usually necessary for Crawford types II, III, IV. The left lung must be deflated to expose the thoracic 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.

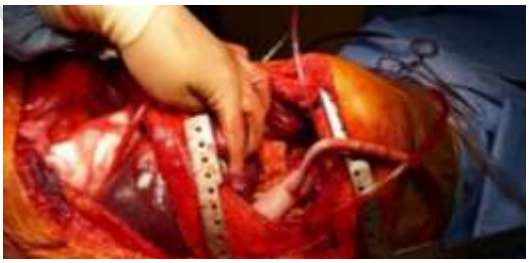
A cross-clamp may then be applied to the aorta just proximal to the aneurysm. However, this will create unacceptably high obstruction (afterload) to left ventricular ejection, leading to cardiac distension, ischaemia and failure. Instead, the heart can be offloaded using 'partial' or 'left heart bypass' (LHB). 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 is maintained at 32-34˚C using a heat exchanger (lower would increase the risk of ventricular arrhythmias). When the aorta is open, return flow pipes can also be split such that oxygenated blood can be provided to visceral organs during surgery. This technique requires a careful team approach as significant amounts of blood (50 - 100L) may need to be returned to the patient by the anaesthetic team alone. The advantage of LHB overall, is in avoiding DHCA.

DHCA (deep hypothermic circulatory arrest) is an alternative strategy to arrest the overall circulation, particularly to sew the proximal anastomosis where this is close to the left subclavian/arch. Hypothermia is the main protection Full CPB is used to cool the patient to 18 - 22˚C (usually via 2-stage femoral venous line and subclavian artery return) 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.

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





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. Also note the extent of incision – posterolateral thoracotomy through 6th intercostal space extending across the midline as far as the umbilicus.

Following completion of the proximal anastomosis, major abdominal vessels are sequentially anastomosed – either as a patch (e.g. including the coeliac, superior mesenteric artery [SMA] and right renal) or using a preformed graft. Intercostals may be tied off or re-implanted. Motor end plate potentials (MEPs) are used (N.B. not possible during deep hypothermia) to assist in determining which intercostals to re-implant, especially in the lower thoracic/ upper lumbar section which represents a 'watershed area' and highest risk for spinal ischaemia/ infarction. 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 - in particular the spinal cord. As well, there is as a significant risk of thromboembolic embolism to organ vessel beds from a diseased aorta. '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

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

The technique/s chosen will depend on the type of aneurysm and all the above may be appropriate for Crawford Types I and II aneurysms.

Outcomes

Historically, outcomes relating to thoracic aneurysm surgery were very poor (up to 50% paraplegia for Type II repairs) but have improved dramatically in modern times.This is due to a combination of improved surgical technique, better organ protection and advances in perioperative and ICU care [2]. The best current figures for published mortality in a high volume centre (by the Coselli Group in the USA between 1986 - 2016), show renal failure, risk of permanent paraplegia (also referred to as spinal cord injury, or SCI) and mortality risk all in the region of 5%. However, in type 2 aneurysms specifically, these figures approach 10% [3]. For the rest of the world, published risk of permanent SCI ranges 5% to even 22% in type 2 aneurysms [4,5]. If it does occur, paraplegia is a devastating complication for the patient when it occurs and everything possible should be done to prevent it both during surgery and post-operatively.

Post-operative (CICU) Care

The specific priorities for CICU care are:

1. Maintain a stable cardiovascular system (i.e. organ perfusion)
2. To detect spinal cord ischaemia + trigger corrective protocols if suspected
3. To be aware of the implications of above for potential bleeding

Any organ protection strategy/plan commenced in theatre needs to continue into the post-operative period on CICU. Any targets set for MAP, CI, CVC, and Hb at handover should be closely followed.

A **minimum MAP of 80 mmHg** must be followed to achieve an SCPP of 70 mmHg

In general (and especially in the case of type II aneurysms), post-operative blood pressure should **not** be artificially lowered at the expense of adequate perfusion of the spinal cord. This applies even in the context of bleeding (unless completely unmanageable). Patients will typically arrive on CICU with a spinal (CSF) drain which may be of 2 types – either a passive drainage system ('Medtronic® Becker Extraventricular Monitor and Drainage system') or an actively managed electronic system (LiquoGuard® 7). Either system should typically be initially set to achieve a CSF pressure of 10mmHg. Intraoperative and planned spinal catheter pressure/drainage rates will also be handed over. Typical would be to allow a drainage rate of up to 10mL/hour.

The other most salient organs in need of protecting are:

- Kidneys
- Liver
- GI tract

The latter is also important as the IMA is routinely sacrificed during surgery and flow is dependent on collateral supply from other preserved territories. Strategies to improve organ perfusion in general include low dose dopamine (or dopexamine) infusion +/- PA Catheter monitoring, and other augmented therapies may include a furosemide infusion or N-acetylcysteine (NAC) for renal protection. The latter will not usually be running by the time of arrival to CICU.

Finally, it is important to state that most of these patients have not had DHCA and if there is cardiovascular stability, with stable or improving lactate, moderate supports only and no evidence of bleeding or ischaemia; then these patients should be woken up and extubated in a standardised way. In fact it is desirable to observe early motor responses as evidence of spinal cord function.

Avoiding spinal cord ischaemia

Blood flow to the spinal cord, and hence neurological function of the lower limbs is dependent on spinal cord perfusion pressure (SCPP). Assuming the spinal drain is initially set at 10 mmHg (with normal CSF pressure approximately 8–15 mmHg) and assuming adequate SCPP to be 70 mmHg:

> SCPP (70 mmHg) = MAP (80 mmHg) – CSF pressure (10 mmHg)

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). Postural changes can also affect CSF pressure. CSF pressure is artificially controlled in TAAA surgery by use of lumbar spinal drains. Spinal drains have been reported to reduce the incidence of neurological injury by 80%. Monitoring and drainage are usually maintained for up to 72 hrs post-surgery but may be kept in situ up to 7 days in the event of ongoing spinal cord ischaemia.

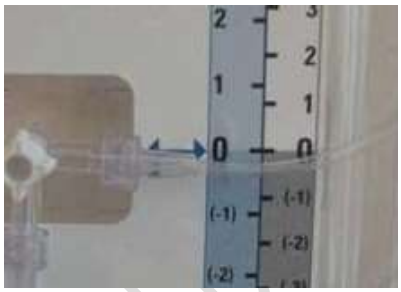
At UHS we currently use the 'Medtronic® Becker Extraventricular Monitor and Drainage system' (left) as well as the LiquoGuard® 7 monitor. Both systems can be confusing to those unfamiliar with them and if zero points are not strictly maintained when patients are moved, **unnoticed over-drainage can quickly and easily lead to subdural haemorrhage**. Whatever the desired CSF pressure, drainage volume should be > 5 mL/ hour and never > 20 mL/hour. Initial CSF pressure should be set to 10-15 mmHg. Where this is not possible, MAP should be further augmented > 80 mmHg with vasopressors.

Medtronic® Becker Spinal Drainage System

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.

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 - see left) relative to a zero point (see lower right). The height of the collection chamber is manually adjusted so that it is always initially 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_20 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. 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 or mid-thoracic spine. 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 unless the patient is moved, and the drain should be left on free drainage. Patency of the drain may also be assessed by periodic lowering of the drain to floor level and if flow is observed, the drain is patent.



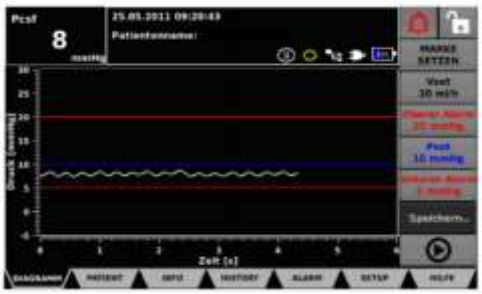
Medtronic Becker spinal drainage system – Zero point

'Pressure line' double arrow set (prescribed) to 10 mmHg by adjusting height of the collection chamber

Automated CSF drainage

The LiquoGuard® 7 (Möller Medical) is an electronic CSF monitor with a pressure transducer that allows the desired pressure of CSF to be set exactly. The LiquoGuard monitor then actively drains CSF to achieve/ maintain the constant pressure set using a peristaltic roller pump. To avoid inadvertent excessive drainage of CSF volume, the minimum and maximum hourly drainage can be set by the user. An additional pressure trace waveform is displayed, giving further reassurance of correct placement and patent drain.

The drainage system will already be connected to the spinal catheter in theatre and should be clearly marked. The transducer itself comes with a sticky dot and must be either applied to the invasive lines plate or to the patient directly as there is still a risk of inadvertent excessive drainage on patient movement.



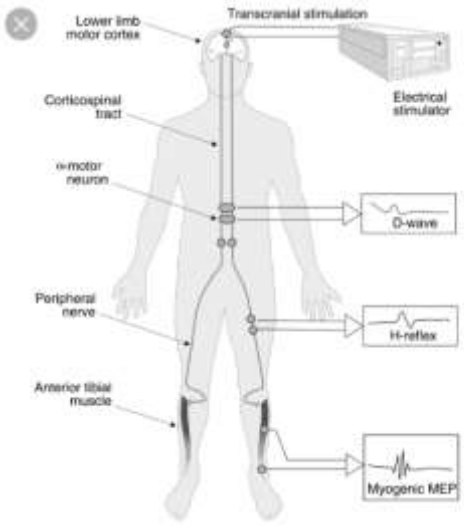
LiquoGuard 7® unit (above) and Visual display for LiquoGuard 7® (below) showing pressure waveform as well as pressure (Pset) at 10 mmHg..

Detecting spinal cord ischaemia (in the unconscious/sedated patient):

At least hourly neurological assessments are required (including motor power score and limb sensation assessments) on day 0 - 3. If the CSF drain is in situ, a minimum of hourly CSF pressure and drainage volumes are also required. It is important that the patient is 'lightened' as early as possible after arrival from theatre so that a full neurological assessment can be completed. However, while the patient remains unconscious, motor end plate potentials (MEPs) are available to detect potential cord ischaemia until the patient regains consciousness. However, this service is usually only reserved for the first post-operative night.

MEPs:

These are setup in theatre by neuro-electrophysiology technicians who are contracted from an external company to provide both theatre and CICU overnight monitoring. Stimulation of the motor cortex is via subdermal electrodes in the scalp. Recordings of muscle contractions are recorded peripherally:



MEPs are affected by increasing depth of inhalational anaesthesia as well as muscle relaxants and hypothermia. In the absence of excessive confounding influences, a reported loss in signal amplitude > 50% or absence of a signal suggests ischaemia in the anterior descending motor tracts and requires urgent action to investigate and rectify the cause. MEPs should be recorded at least hourly where there is actual or has been suspicion of ongoing ischaemia. They are no longer required when the patient is awake, moving all limbs and/or able to self-report neurological symptoms.

Management of spinal ischaemia ('COPS' protocol):

Cord compromise is an emergency and may be immediate or delayed. Vigilance in early detection is key to preventing permanent paraplegia. 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 any doubt/ suspected ischaemia the following actions should be **immediately** considered and the patient meticulously reassessed:

- Lie the patient flat (improve venous drainage + reduce CSF pressure)
- Check function of drain/ re-site if necessary
- Ensure O2 delivery (SATS > 96%), cardiac index (> 3L/min/m^2), Hb > 110 g/dL
- Ensure/increase MAP > 90 mmHg (SCPP > 80 mmHg) in 5 mmHg increments

OR:

1. C for CSF drainage
2. O for oxygen delivery
3. P for pressure management
4. S for status of patient

In addition to above, 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**
- **Stop draining CSF (place liquoGuard 7 to 'pause')**
- **Correct coagulopathy**
- **Consider urgent CT scan head (if altered mental status or focal deficit)**
- **Consider urgent CT scan spine (check for haematoma)**

A CT scan of the spine will exclude cord compression due to an epidural haematoma and a CT brain will exclude intracranial haemorrhage.

If it is not possible to maintain a CSF pressure < 15 mmHg, acetazolamide 500mg 8hrly may also be considered to reduce CSF production.

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 has been corrected, platelet count is > 100 and S/C heparin has been discontinued for > 8 hours [6 - 8].

Management of issues with spinal drains:

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.

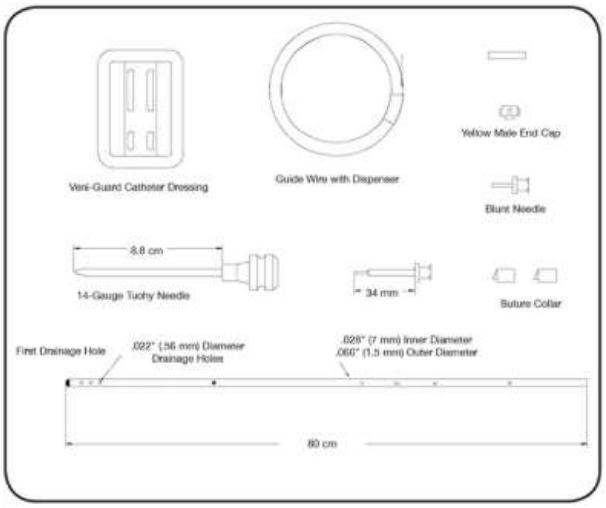
Drain sites should be inspected a minimum of 6 hourly. If the LiquoGuard drainage bag is full, press 'pause', change and then press 'start' again. CSF may be cloudy (infection), blood stained (subdural haematoma) or leaking (increased risk of infection). Leaking CSF may be improved simply by placement of a purse string suture or may require re-siting of the drain.

Post-removal, a transparent occlusive dressing is applied, the patient is placed in a 30-degree semi-recumbent position for 6 hrs to reduce the risk of headache from leakage of CSF. The insertion site is inspected every 4 hrs for signs of leak increasing in frequency to every 2 hrs if the patient complains of headache. Any external CSF leak will require a purse string suture. Heparin can be administered 8 hrs post spinal drain removal unless contraindicated.

Re-siting of Drain:

If a re-site of a spinal drain is necessary, care should be taken to familiarise with the insertion set prior to re-insertion. Coagulopathy should be corrected. The correct catheter to use is the integra spinal drainage catheter, located in the cardiac storeroom, E-level theatres. This is a soft tipped, multi-port silastic catheter, licensed for intrathecal use. The Tuohy introducer is 14G and may be inserted directly preferably at lumbar level L4/5 until CSF is reached. Be careful to not leave the introducer uncovered while preparing the spinal catheter.

The catheter itself should be pre-loaded onto the introducer wire and both should be bathed in saline prior to loading and insertion for lubrication. Approx 5 – 10cm may be inserted into the intrathecal space and any excess catheter should be looped at the skin x 3, sutured in place x 3 and then connected to a sterile manometer line and taped. The proximal end of the silastic tubing must be pushed on to a luer connector prior to attaching the manometer line. This also requires a suture tie to prevent accidental disconnections with further clear adhesive tape wrapped over the connection as well. [9]



Integra spinal catheter sets. 14 G Tuohy, 8.8cm length needle, 80cm tubing with markings at 10cm, 20cm, 30cm.

Analgesia

The options for analgesia are as follows. Some or all may be used concurrently:

1. ESP catheters (Erector spinae plane) or equivalent fascial plane continuous blockade
2. Cryo-ablation of thoracic nerves by surgeon [10]
3. Lidocaine patches (5%), up to x 3 with 12 hr life span.
4. Paracetamol, IV fentanyl, remifentanil, or other opiates (but taking careful note of renal function)
5. Thoracic Epidural

N.B. - Intrathecal diamorphine has a speed of onset < 10 minutes and lasts for 4 – 6 hours. While theoretically possible to give intrathecal diamorphine (500 mcg to 750 mcg, 6 – 24 hourly via the spinal catheter/drain with a 10mL flush of saline), this has the potential for significant side effects, and requires temporary clamping of the drain. Side effects may occur including:

- Respiratory depression (may be delayed up to 12 hours > administration)
- pruritis
- Nausea and Vomiting
- Urinary retention (leave catheter in situ)
- Infection (full aseptic technique for administration + filter on three-way stopcock)
- Inadvertent administration of other medication (all access ports/ bungs covered with labelled stickers/ gauze)
- **Arachnoiditis**

At the time of writing, and largely on account of the risk of arachnoiditis, the use of intrathecal analgesia/ access of any form is strongly discouraged.

****Only a consultant should ever administer intrathecal diamorphine.****

Further ICU care:

An NGT will usually be sited in theatre, so a postoperative CXR is necessary to confirm the position. In the event of accidental extubation, laryngeal oedema following a double lumen tube may make reintubation more challenging. After elective extubation, CPAP or Optiflow must be applied immediately. 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.

CICU instructions/ parameters should be clear and re-iterated daily. The daily review should include written instructions for:

1. MAP (i.e. > 80,85 or 90 mmHg)
2. ICP (LiquoGuard: PSet at 8 – 15 mmHg)
3. Max CSF volume loss/ rate set (20 mls/hour)
4. O2 Sats (> 95%)
5. Hb (> 100g/L)
6. Patient position allowed (usually > 60 degrees + can be turned as normal)
7. DVT prophylaxis (S/C heparin 5,000 units BD, first dose day 1 eve > R/W bloods)

Remember though, in the absence of any significant problem, the default pathway is to lighten and extubate if appropriate!

References + further reading

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Adult congenital heart disease patients in CICU

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Previous authors: Dr Richard Cope & Dr Kirstin Wilkinson

Congenital heart disease (CHD) is common, occurring in just under 1% of all live births. With the advancement in all treatment modalities of CHD, both in the cardiac operating room and the cardiac catheterisation laboratories, the survival of these patients has improved significantly. However, this is with varying degrees of physiological impairment and/ or sequelae. The adult congenital heart disease (ACHD) population is growing at a rate of about 5% per year.

Patients present to cardiac intensive care for several reasons,

1. Intensive care medical management of organ dysfunction
2. Post-operative care after cardiac surgery
3. Post-operative care after non-cardiac surgery e.g. general surgery, obstetrics

These patients have a wide spectrum of pathology ranging from simple lesions through to complex lesions with multi-organ involvement. The patient may have had corrective, palliative or no cardiac surgery depending on the nature of their cardiac lesion. It is important to understand their anatomy and physiology as that will impact on the management strategy on CICU.

All patients will have an adult congenital heart disease cardiologist caring for them along with the team of liaison nurses who manage multiple aspects of their care both within and outside the hospital setting. This team should be notified of any admission to the CICU.

Basic concepts

CHD can be classified in several ways and each system is imperfect but acts to help identify the pathology and physiology involved. 2 major systems are,

1. Segmental approach – analysis of lesions to give a framework for the analysis of the path of blood flow through the heart. The 3 major segments are the atria, ventricles and great arterial trunks. Firstly, determine the position of the heart in the thorax, the direction of the cardiac apex and the situs of the thoracic and abdominal organs.
2. Physiological approach – classification of lesions according to presence of shunts, obstruction or combinations of both.

Shunting: anatomic or physiologic.

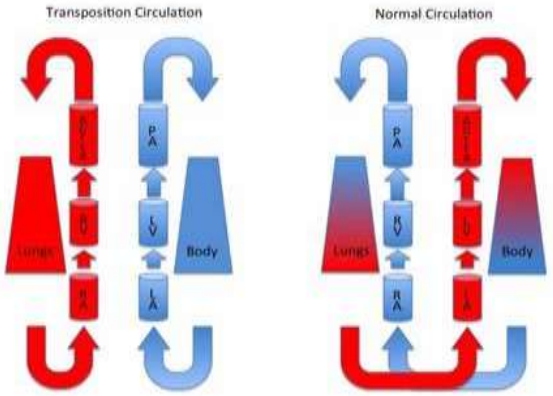
- Anatomic: communications between 2 circulations at either the atrial, ventricular or great arterial level. May be simple or complex.
  In simple shunts, blood flow is determined by the size of the shunt orifice. For large or non-restrictive orifices (dependent shunt), the size and direction of the shunt is determined by outflow resistances or the ratio between the pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR). This ratio is the Q_p: Q_s. Haemodynamic and ventilation changes on these resistances can have large impacts on the size and directions of the shunts.
  For complex shunts, an obstruction exists along the shunt. The degree of shunting is determined by the degree of obstruction along with the PVR or SVR.
- Physiologic: venous return from one circulation recirculating through the arterial outflow of the same circulatory system. Physiologic shunts are often due to anatomic shunts, but they can occur in the absence of an anatomic shunt e.g. transposition of the great arteries. Obstructions may be fixed or dynamic or both e.g. tetralogy of Fallot.

Effective blood flow: quantity of venous blood from one circulation that reaches the arterial system of the other circulation. Effective pulmonary blood flow is the quantity of systemic venous return that reaches the pulmonary arterial system. Effective pulmonary blood flow and effective systemic blood flow are always equal. This is the flow necessary to maintain life.

Total blood flow: sum of both effective blood flow to a circulation and recirculated blood flow. Total systemic blood flow and total pulmonary blood flow are not equal, even in normal patients, as there is always a small degree of physiological shunting. Recirculated or physiological shunt flow is the extra non-effective blood flow added to the effective blood flow = total blood flow to the circulation.

Obstruction(s): may affect either systemic or pulmonary blood flow at one or multiple levels.

Series or parallel circulations: the normal systemic and pulmonary circulations are in series where blood travels through each circulation without any mixing of oxygenated and de-oxygenated blood (except bronchial veins). An example of a parallel circulation is unrepaired d-TGA where blood only flow into the pulmonary or systemic circulation. Mixing at one level (atrial, ventricular or great arteries e.g. patent ductus arteriosus) is essential for survival. Single ventricles are also examples of parallel circulations with both circulations dependent on one pump. Balancing the circulations occurs when the performance of each circuit is optimised through manipulating the resistance or flow in each circuit.



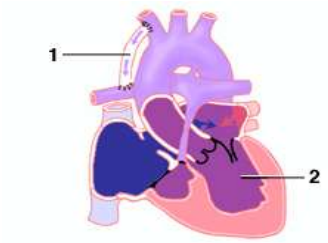
Revision Fontan patients in CICU

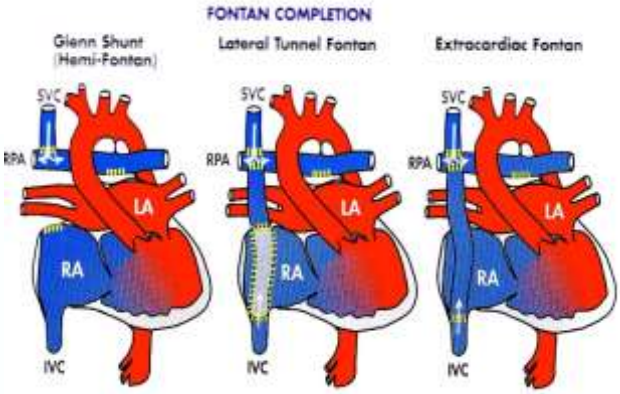
Introduction

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. These patients demonstrate many of the physiological/ anatomical concepts described above and will be discussed in further detail to show how the management strategies in CICU can impact the patients.

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,

BT Shunt





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 (the patient may have an actual RV with an absent LV but this single ventricle assumes the role of the systemic ventricle) 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 ended up with very larger right atriums that formed 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 can 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 because 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).

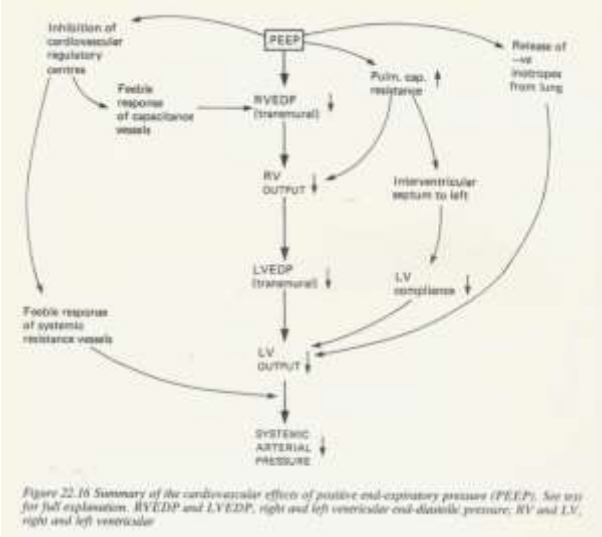


Figure 22.16 Summary of the cardiovascular effects of positive end-expiratory pressure (PEEP). See text for full explanation. RVEDP and LVEDP, right and left ventricular end-diastolic pressure; RV and LV, right and left ventricular

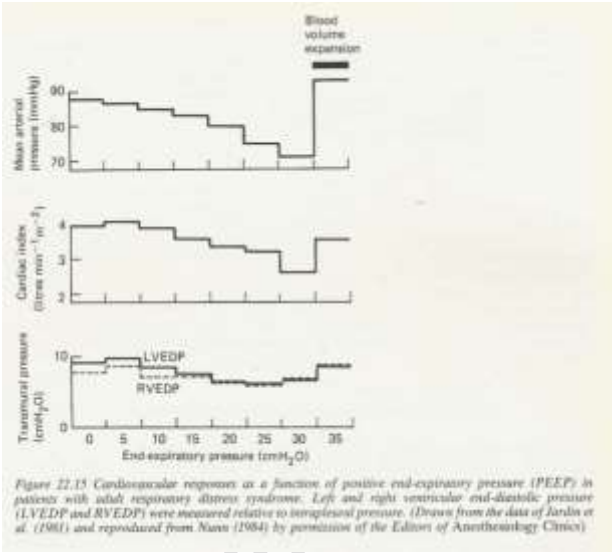


Figure 22.15 Cardiovascular responses as a function of positive end-expiratory pressure (PEEP) in patients with adult respiratory distress syndrome. Left and right ventricular end-diastolic pressure (LVEDP and RVEDP) were measured relative to intrapleural pressure. (Drawn from the data of Jardin et al. (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 afterload. 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 must 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.

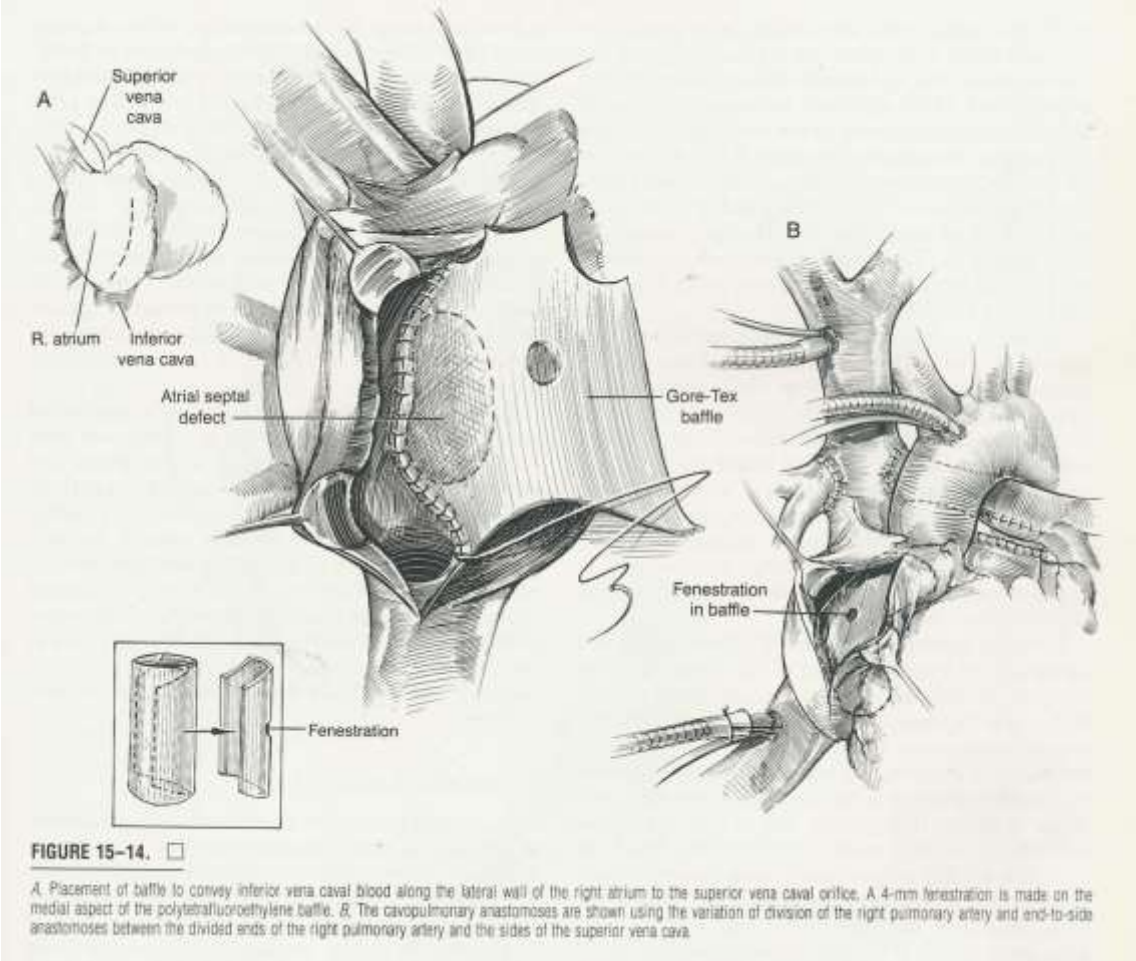


FIGURE 15–14. □

A, Placement of baffle to convey inferior vena caval blood along the lateral wall of the right atrium to the superior vena caval orifice. A 4-mm fenestration is made on the medial aspect of the polytetrafluoroethylene baffle. *B*, The cavopulmonary anastomoses are shown using the variation of division of the right pulmonary artery and end-to-side anastomoses between the divided ends of the right pulmonary artery and the sides of the superior vena cava.

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 within 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. There is no right or wrong answer here and every case must be judged on its merits. On balance 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. Vasopressin may be better in the context of a low afterload.
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 yet.

Renal Failure

There is a relatively high risk of this because:

- Some of these patients have poor renal function preoperatively
- 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 because 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 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 2023*

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 the groups of patients that are deemed suitable for TAVI are rapidly expanding.

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. All the procedures take place in the catheter or hybrid lab. Post-operatively, the trans-apical cases are managed on CICU, all 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 CHDU
- Patients should have standard cardiac monitoring
- Patients can eat and drink as normal

Trans-apical cases

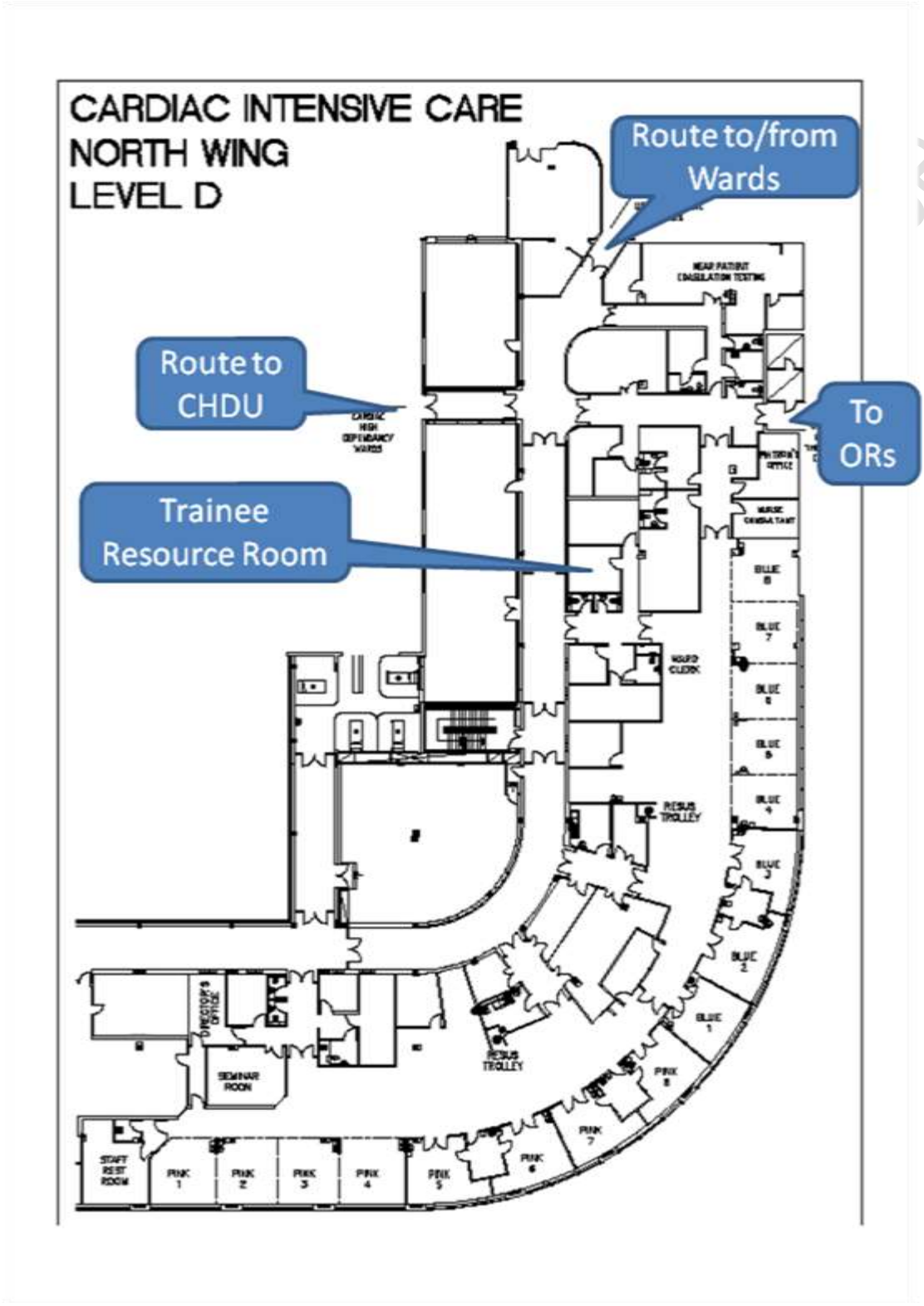
- Will have an extra-pleural catheter sited by the surgeon
- 10ml 05% bupivacaine bolus is given before leaving lab
- Extra-pleural infusion of 0,.125% or 0.25% bupivacaine
    - Start infusion at 8ml/hr
- They are transferred to CICU on propofol IV infusion for sedation
- Should be extubated as soon as possible 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
  - 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
    - Time is critically important since some patients may be suitable for mechanical thrombectomy
- Para-valvular leaks
  - This is 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 & clinical fellows 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.

	Use/ might use	Never use	Competent through previous use	Had training	Needs training
Ventilator					
Hamilton C6					
Oxylog transfer					
Hamilton transfer					
Syringe drivers					
CTITU					
Defibrillators					
Theatre lifts					
Zoll (Int/Ext)					
Monitoring					
Vigilance CO monitor					
LIDCO CO monitor					
CTITU					
Transfer					
Miscellaneous					
Metavision					
IABP					
Pacing box					
Fibreoptic stack					
Difficult intubation trolley					
Chest opening kit					

Sharps and Contamination Incidents

Author: Dr Paul Diprose *Revised February 2023*

Best Practice with Sharps

Sharps, such as syringe needles, scalpel blades and many other sharp devices, are routinely used in healthcare. Every year, numerous people sustain injuries from contaminated sharps. These injuries pose a significant risk to the physical and mental health of the injured person, cost healthcare organisations time and resources and have the potential to result in costly litigation.

Sharps must always be handled carefully, in accordance with the following principles:

- always get help when using sharps with a confused or agitated patient
- never pass sharps from person to person by hand - use a receptacle or 'clear field' to place them in.
- never walk around with sharps in your hand
- never leave sharps lying around - dispose of them yourself
- dispose of sharps at the point of use - take a sharps bin with you
- dispose of syringes and needles as a single unit - do not remove the needle first
- needles or other sharps must not be bent
- when transporting a blood gas syringe, remove the needle using a removal device and attach a blind hub prior to transport
- safer needle devices, such as safety cannula and needleless systems, **must** be used where available
- do not re-sheath used needles
- in exceptional circumstances, if re-sheathing **cannot** be avoided, use a specific needle re-sheathing / removing device.

Questions and Answers

Q. How do I report a needle stick / contamination injury (NSI)?
A. Ring the **NSI Hotline on 6353** at any time and follow the instructions

Q. Why do I need to visit Occupational health after a needle stick/ contamination injury?
A. We offer appropriate advice and treatment following risk assessment of the injury. We will help to collect information about the source of contamination and will document the incident.
Even if you have had a full course of Hepatitis B and know that you are protected, you must still be seen so that we can check your immunity and advice about booster doses.

Q. What information do I need to bring with me to Occupational Health or A&E?
A. If the source is known, the patient's name, date of birth and ward. Any other information about the patient and/or equipment involved to assist in the assessment of risk is helpful.

Q. What blood tests will be taken?
A. From you: At the time of injury, blood will be taken to test for Hepatitis B antibodies. The sample will also be stored. We recommend that more blood is taken 3 months after the injury, again for storage. Blood is stored indefinitely to help you prove the origin of infection, should you become infected with any blood borne viruses from the injury.
From the patient: With the patient's consent, blood will be taken to see if they are infected with Hepatitis B or C.

Q. What happens if the patient involved is infected with HIV?
A. In the event of high-risk exposure to HIV infected blood or body fluids; three anti-HIV drugs may be prescribed by an expert (as listed in the Policy for Management of Sharps and Contamination Incidents). These drugs should ideally be started within 1 hour of injury. The drugs are available in Occupational Health or A&E.

Q. Who do I need to inform when I have a needle stick injury?
A. At the time of injury you need to inform your consultant and ring the **NSI Hotline on 6353**. If you are seen in A&E out of hours, the Occupational Health Dept. need to be informed about the injury, so that we can check you have had the appropriate follow-up and document the injury in your record.

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

Bleed it:
Encourage bleeding – but do not massage the site **(DO NOT squeeze to make it bleed)**

Wash it:
Wash injury with soap and hot running water

Cover it:

Apply a waterproof dressing

Report it:
Inform your manager and
Contact the Needlestick Hotline 6353
(out of hours leave a message)

And attend the
Occupational Health Department UHS Service

08.00 – 16.00 Mon – Fri

Or
Out-of-hours
Needlestick Hotline x6353
And then the Emergency Department
SGH, 023 8120 4994

Complete an electronic Incident form

More detailed guidance can be found in the following policy:
Management of Sharps and Contamination Incident

Cardiac anaesthetic and ITU weekly teaching

Weekly teaching programme:

	Topic	Time	Location	Presenter	Cons Chair
Monday	M+M 1st Monday of month	07:30	CICU seminar room or Teams	Consultant	
Tuesday	Cardiac anaesthetic/ ITU teaching	15:00	CICU Seminar Room	Consultant	
Wednesday	TOE teaching	07:30	CICU Seminar Room or Teams	Consultant	
Friday	Cardiac anaesthetic meeting	07:30	Anaesthetic dept. seminar room or Teams	Clinical fellow/ trainee/ consultant/ outside speaker	

This is the usual teaching programme. There will be a weekly schedule emailed out and everyone is invited to attend. Please ask a consultant or fellow trainee/ clinical fellow if you need more information or to be put on the notification list.