Centrifugal Clinical Guidelines

Paediatric Intensive Care & Neonatal Intensive Care
Royal Hospital for Sick Children, Glasgow

The Extracorporeal Life Support (ECLS) service was established at the Royal Hospital for Sick Children (RHSC), Glasgow in 1992 and was designated as a National Service, funded by National Services Division (NSD), from April 2001. The service provides temporary life support to paediatric patients with cardiac and/or pulmonary failure and is one of four such services in the UK, and the only one in Scotland. The service plays a key role in supporting the national Paediatric Cardiac Surgery Programme.

The RHSC ECLS service is a member of the European chapter (EuroELSO) of the Extracorporeal Life Support Organization (ELSO).RHSC received the ELSO Award for Excellence in Life Supportfor 2009-11 and again for2013-15.A designated Center of Excellence has demonstrated extraordinary achievement in the following three categories:

- Excellence in promoting the mission, activities, and vision of ELSO
- Excellence in patient care by using the highest quality measures, processes, and structures based upon evidence; and
- Excellence in training, education, collaboration, and communication supporting ELSO guidelines that contributes to a healing environment for families, patients and staff.

The purpose of these guidelines is to provide training to individuals in the theory and practice of prolonged ECLS using the Levitronix centrifugal pump system. RHSC specialists will, after appropriate didactic, practical and clinical experience, be competent to independently conduct and manage extracorporeal circulation for pulmonary or cardiac support under the guidance and supervision of an ECLS physician.Prerequisites for the training will include a sound knowledge of basic anatomy, physiology, pathophysiology, critical care nursing, and critical respiratory care.

The new manual reflects the introduction of new equipment, new procedures, a more diverse case mix than we could have imagined in 1992 and, not least, the change of emphasis from roller pump to centrifugal pump ECLS.Developments in the design and function of third generation centrifugal pumps have seen them increasingly used for , both in the UK and internationally. This, together with the move from Silicone membrane oxygenators (with high resistance to blood flow) to very low resistance Polymethylpentene hollow fibre oxygenators has allowed much miniaturised circuits with smaller priming volumes, quicker and easier deployment and reduced Heparin requirements. However, unlike roller pumps, centrifugal sysyems are not occlusive and are therefore pre and after-load sensitive andare more sensitive to patient physiological fluctuations.

This maunual includes several updates including the a redesign of the circuit, updated CVVHF guidelines, a Pertussis leucodepletion guide and fibrinogen concentrate guide. Older versions of the manual should be discarded.The aim of the guidelines is to act as an "aide memoire" and to give structure to the introduction of this technology. It is not set out as a bible on how and when to utilise centrifugal technology. We would like to thank all those who have contributed to these guidelines.

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Date of Publication of Version 3:August 2013
Date of Review:August 2015

Contents

YORKHILL ECLS PROGRAMME

The ECLS Programme at Yorkhill celebrated its 20th Birthday in 2012. Its success to date has been based on teamwork and a multi-disciplinary input to management of these patients. We are indebted to all those who have given so much time and effort to make the programme work. Since 2001, the ECLS service at Yorkhill has been funded by the NHS National Services Scotland. We work closely with the other UK ECLS Programmes at Leicester, London (Great Ormond Street) and Newcastle to provide a service for the whole of the UK. Since starting the ECLS programme at Yorkhill in 1992, we have undertaken almost 500 ECLS runs. Initially, the service was for neonatal respiratory conditions but has since expanded to include paediatric respiratory conditions and later cardiac ECLS. We have also supported a couple of older children and adults in exceptional circumstances. To date we have supported over 480 patients.

Results are encouraging and are comparable with other major centres providing ECLS.Overall neonatal respiratory survival to discharge is 80%, paediatric respiratory is 72% and cardiac survival is 53%.Our case mix reflects modern ECLS with a larger percentage of cardiac support than is seen in the ELSO registry.

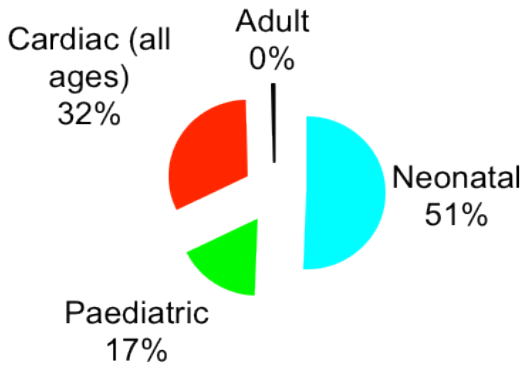


Figure 1 ECLS Categories & Caseload in RHSC

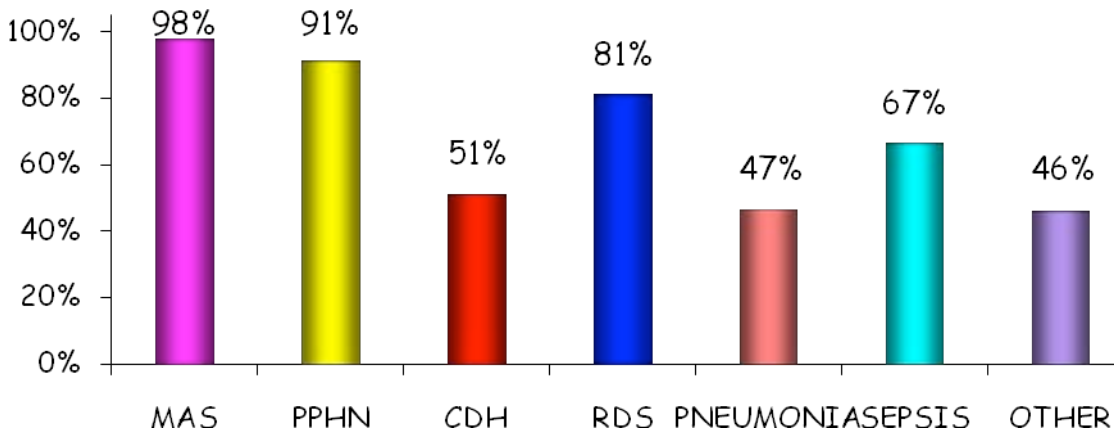


Figure 2 Neonatal Respiratory Outcomes by Diagnostic Category

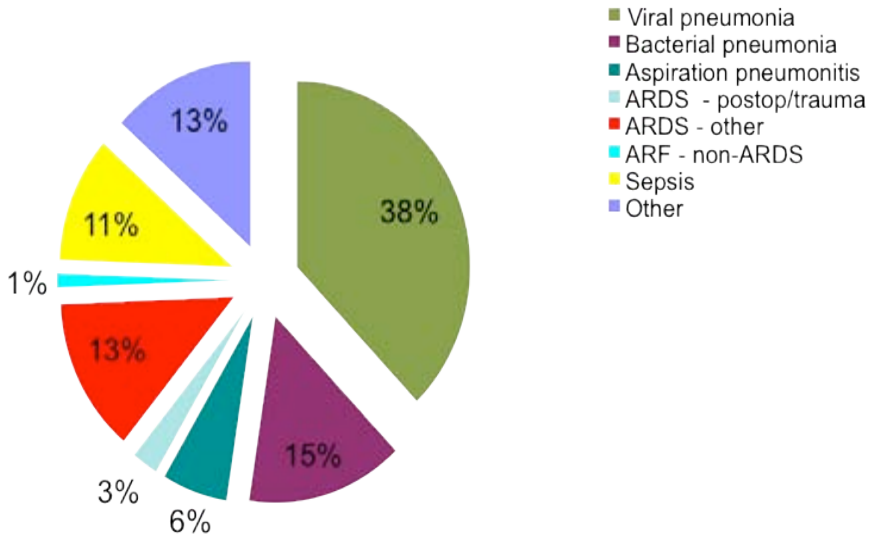


Figure 3 Paediatric Respiratory Caseload by Diagnostic Category

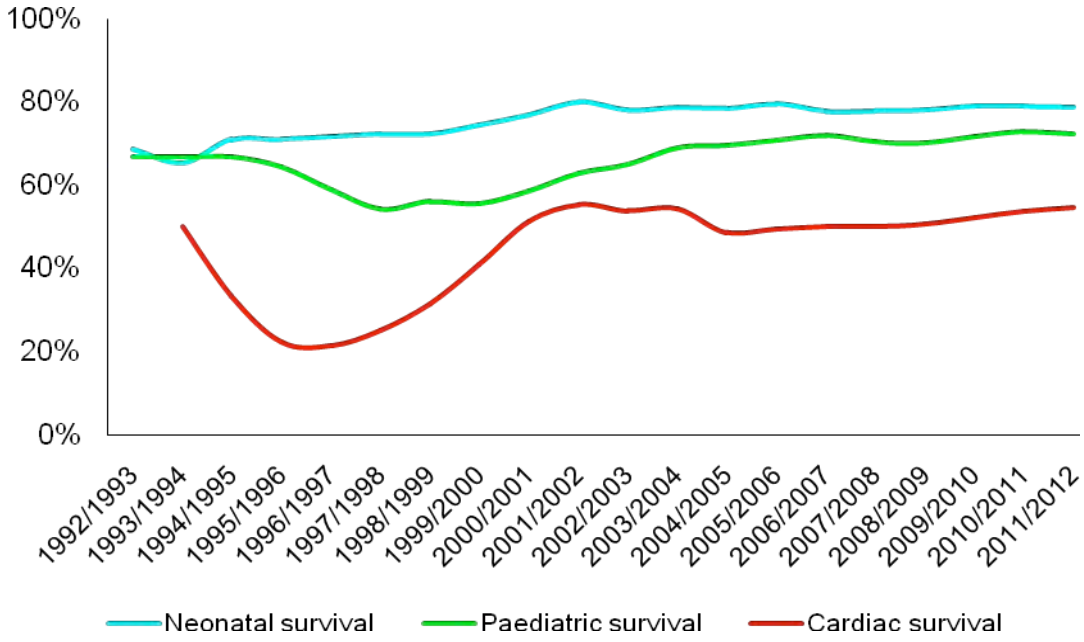


Figure 4 Overall Cumulative ECLS Survival by Category

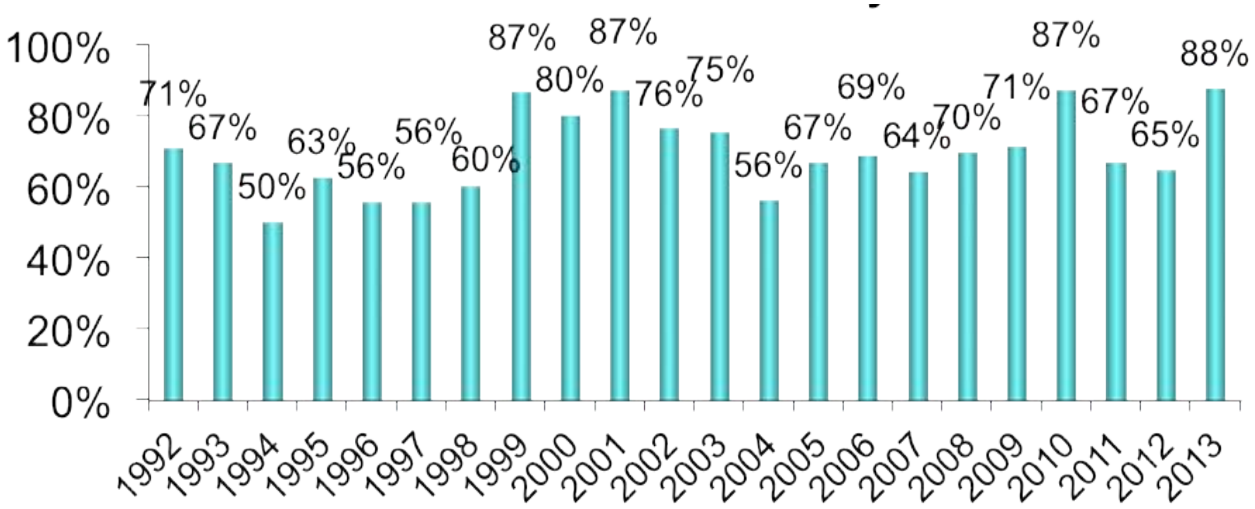


Figure 5 RHSC Overall Survival by Year

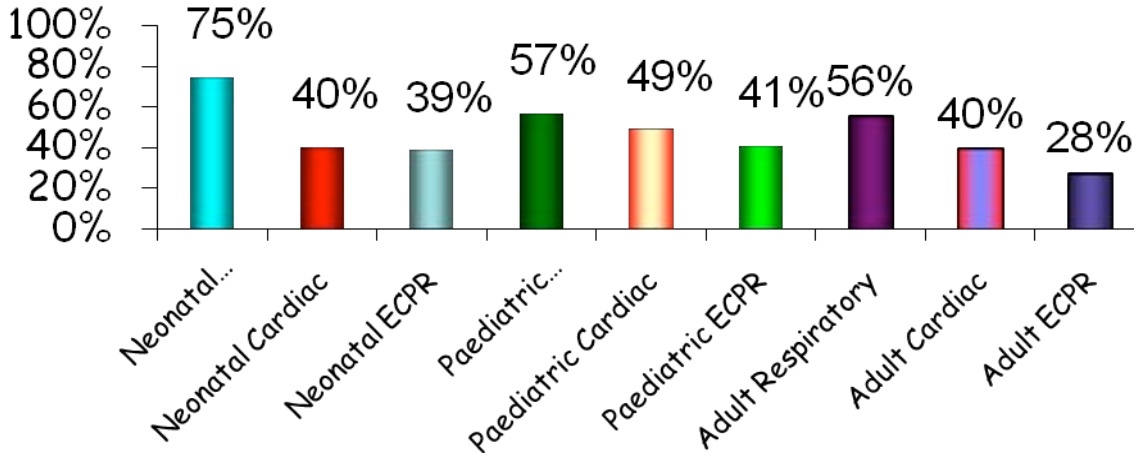


Figure 6 ELSO Survival by Category

Education

The Extracorporeal Life Support Organisation (ELSO) the professional advisory body for ECLS lays down minimal standards for the ongoing training of ECLS specialists in centres providing an ECLS service. RHSC has a well developed ECLS educational programme to meet these standards which has a requirement for staff to attend as a minimum:

- 6 monthly water drills (for VAD and)
- 1 ECLS meeting per annum
- 1 clinical simulation scenario or update day (alternating)
- Annual Exam

These training requirements must be considered **mandatory** in order to secure the continuing safe provision of the ECLS service and they form a key component in ensuring appropriate clinical governance of the ECLS service.

Audit and Research

A pro-active research and audit programme exists at Yorkhill with a focus currently on ECLS related infections, nutritional support of the patient and the coagulation management on ECLS.

We have presented regularly at national and international meetings.

Staff are given the opportunity to attend these meetings regularly.

Regular updates on on-going research projects are placed on the ECLS website on the GGC intranet under Paediatrics / Critical care.

Centrifugal pump overview

Centrifugal pumps have 4 major parts: the pump head, motor drive, pump console, flow probe. The heat exchanger is not required and has been removed from the system

The **pump head** comes in 2 sizes the **Pedivas** with a priming volume of 14ml and the **Centrimag** with a priming volume of 31ml. Within the pump head (see figure 1) is an impeller or rotor which, when the motor drive is switched on, is magnetically levitated within the pump head so there is no contact between the impeller and the casing. This means there is minimal heat generation and therefore blood trauma unlike in the roller pumps or older centrifugal technology. The maximum rated flow of the Pedivas is 1.7L/min and the Centrimag 10L/min. This equates roughly to children less than 10kg requiring the Pedivas pump head and those greater than 10kg requiring the Centrimag pump head.

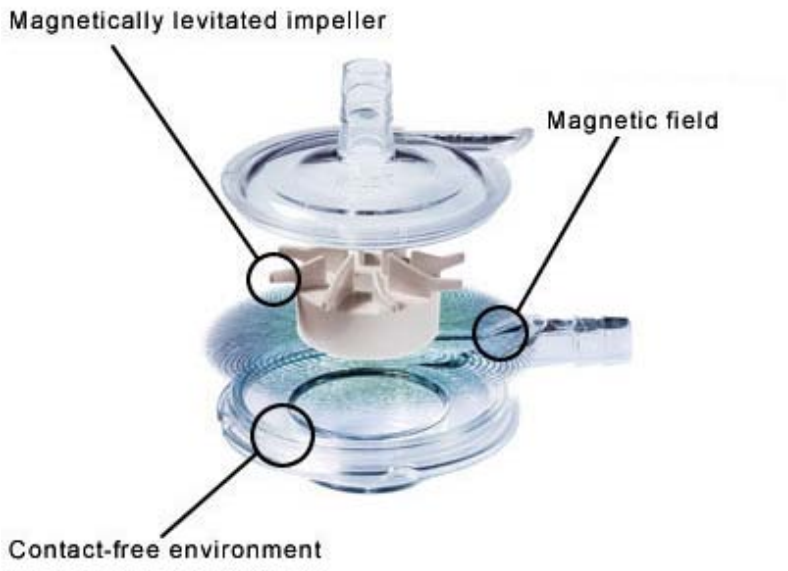


Figure 1. Pump head

The pump head sits in the **motor drive** and is held in place with a retainer grub screw. The motor drive contains the magnetic that holds the impeller in place when in use. The motor drive rotates the impeller via magnetic force to impart kinetic energy and therefore blood flow.

The **motor drive** is connected to the pump console by the drive line which is 2m long and can be positioned anywhere relative to the patient. The pump console is the control panel and lets us set the revolutions per minute (rpm) of the impeller. This dictates the blood flow that is measured by the **flow probe** which is shown on the pump console. The rpm are set and constant and will not change dependant on the pre-load and afterload to the pump. The blood flow as measured by the flow probe will be the factor that will change. This is different to the roller pump which will keep ejecting blood even if the after load is extremely high. The bladder in the roller pump allows some forgiveness to the degree of pre-load to the pump but pre-load in the roller pump is limited by gravity drainage (ie. height of the bed) and cannula position.

Centrifugal pumps work by the impeller spinning and thereby imparting kinetic energy to the blood in the pump head (figure 2). It pulls blood into the pump head and is therefore not reliant on gravity drainage as the roller pump is. This means the patient can be nursed in a normal bed and appropriately positioned at 30° "head up" to optimise lung care.

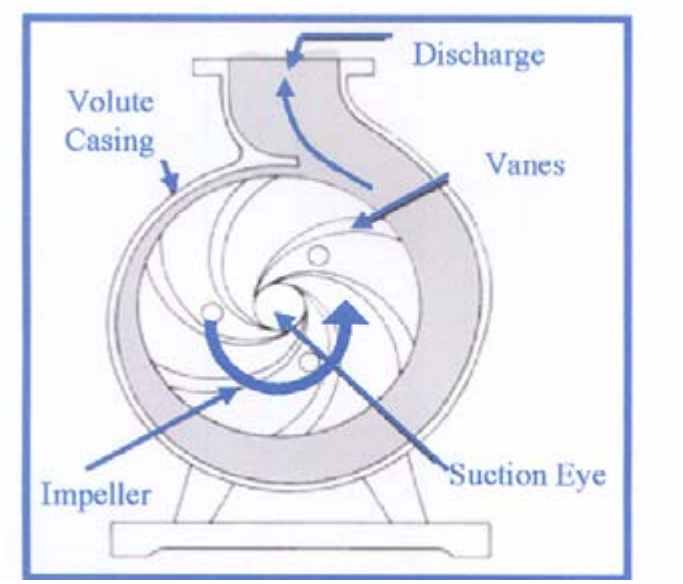


Figure 2. Blood flow through pump head

Centrifugal pumps are not occlusive unlike roller pumps and are therefore pre-load and after-load sensitive. This means that if the patient's blood pressure goes up there is more resistance against the blood exiting the centrifugal pump and therefore there is less blood flow exiting the pump despite the rpm staying the same. This is monitored using the flow probe. Appropriate pre-load to the pump is important. With a low pre-load or filling pressure the heart will provide sub-optimal effective cardiac output and the same is the case with the centrifugal pump. If the pre-load to the centrifugal pump falls the blood flow will fall despite constant rpm. Remember the rpm will stay static. This can happen for example if the patient is actively bleeding and blood loss is not replaced appropriately. In a severe situation the venous cannula will judder indicating this and if the atrium is nearly empty the atrial wall can be pulled onto the venous cannula causing local trauma.

Pressure monitoring

Pre and post-oxygenator pressure monitoring is measured on the pump console and is not servo regulated. The inlet pressure to the pump is important as if it gets too negative it can create trauma at the venous cannula site or increase the risk of air entrainment. The inlet pressure will be dependant on the cannula type, cannulation site, targeted flows and patients filling status. Itshould be monitored but the trend should be considered to be most important factor. It is difficult to give target values but it will usually range somewhere between +5 and -50mmHg. Larger patients with the Avalon cannula may have inlet pressures as low as – 99. It is important that any sudden changes to pressure and any juddering of the tubing are urgently investigated and managed.

Duration of centrifugal

The Levitronix centrifugal pump has an EU kite mark approving use up to 30 days though many centres have used them for more than three times this duration safely.

Summary of advantages of centrifugal pumps versus roller pumps

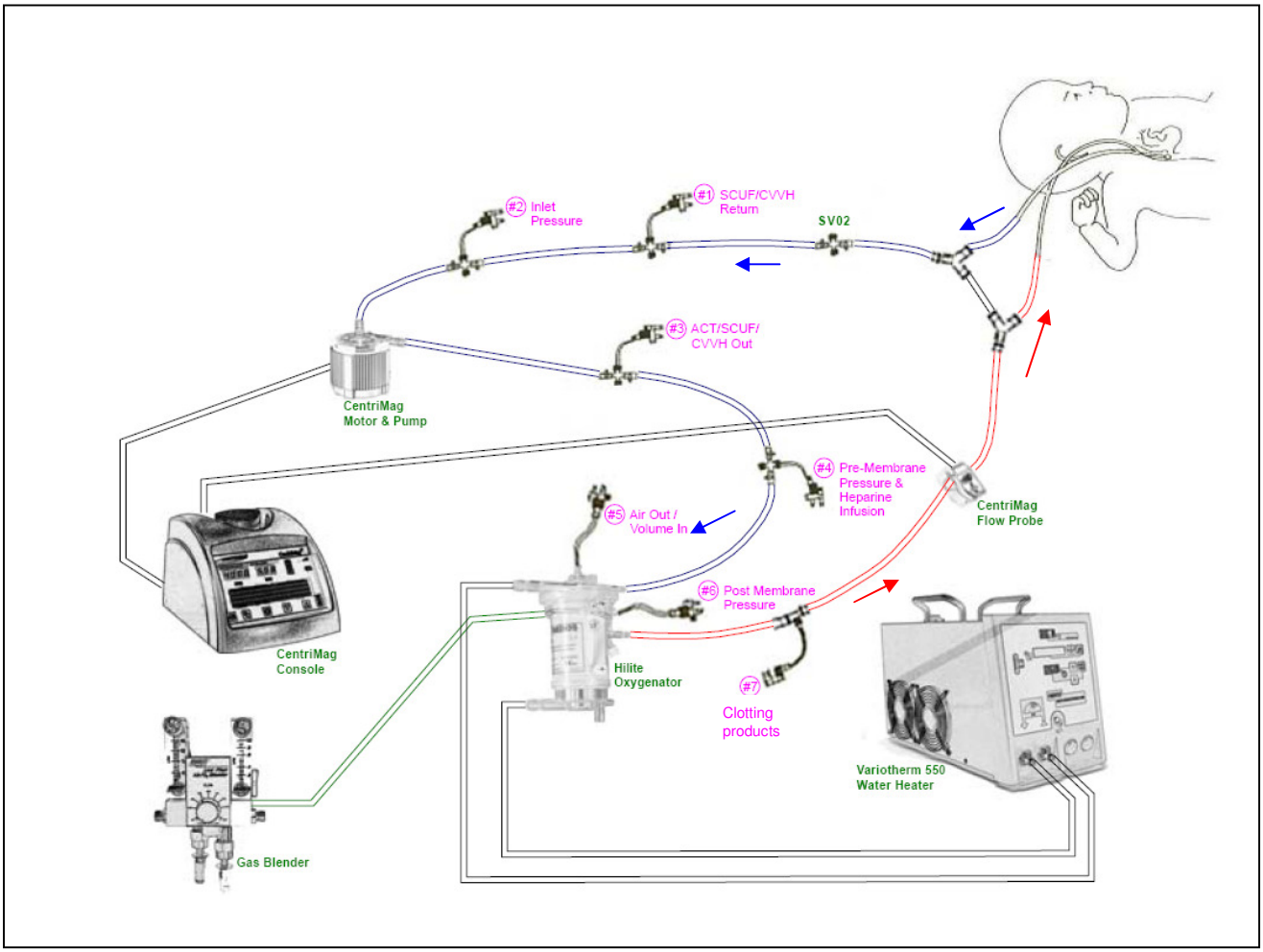
- Patients no longer need to be elevated and can be nursed in normal positions as the pump is not reliant on gravity drainage of the blood.
- The priming volume of the circuit is much smaller allowing rapid and easy non-blood primes in an emergency and less exposure to plasticisers
- Blood flow is not interrupted by roller occlusion with less haemolysis
- Lower anti-coagulation required and therefore less bleeding and blood products required
- Relatively easily transported inter and intra-hospital
- Patient management more akin to normal patient's physiological management.
- Nursing and medical staff experience of it being "easier to run"

Summary of disadvantages of centrifugal pumps versus roller pumps

- Need to remember that rpm does not equal blood flow.
- This is an open system therefore blood which usually flows forward can also flow backwards – retrograde flow.
- Potential for air entrainment to circuit at cannulation site as pulls blood into pump and tubing
- Unit cannot be hand-cranked hence a spare console, flow probe and motor drive is always available

The nuances of the centrifugal pump will be fully covered in this manual, during the teaching programme and by follow-up water drills, education sessions and scenario training.

Centrifugal ECMO circuit equipment



The circuit consists of (from patient):

- Venous saturation probe (SvO_2)
- Pigtail #1 used for SCUF/CVVH return
- Pigtail #2 used for inlet pressure monitoring/CVVH access
- Centrimag motor and pump connected to centrimag console
- Pigtail #3 used for ACT measurement / SCUF out
- Pigtail #4 used for pre membrane pressure and heparin infusion
- Membrane oxygenator connected to Gas Blender and Water Heater
- Pigtail #5 used for air removal and ac[illegible]ion of volume
- Pigtail #6 used for post membrane pressure
- Pigtail #7 used for clotting products (FFP, Cryo, Platelets) administration
- Centrimag flow probe (should be sited after bridge on arterial side)

The (Hollow Fibre) Oxygenator

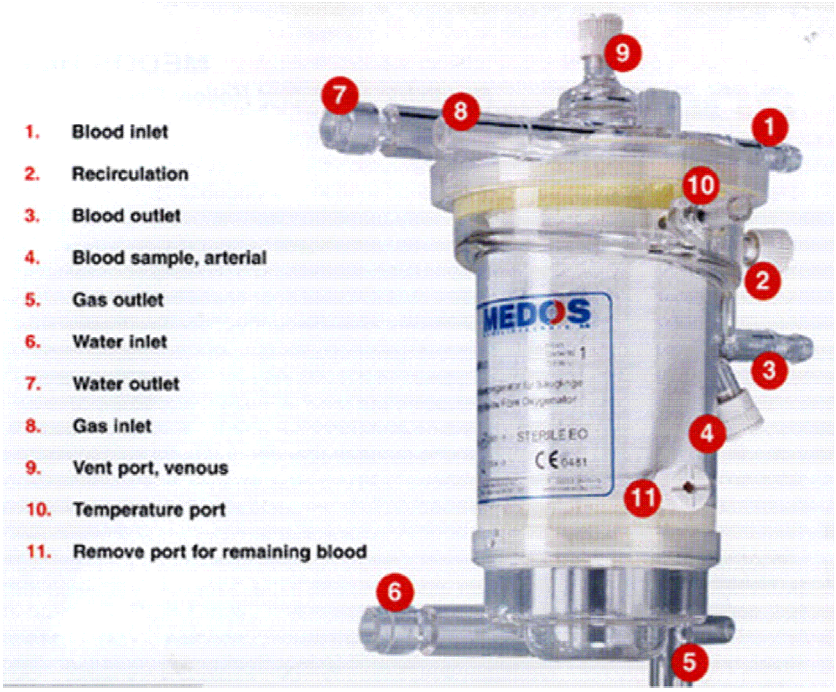


Figure 1. Medos hollow fibre HILITE Oxygenator LT

Description

The oxygenating part of the Medos HILITE 0800 LT, HILITE 2400 LT and HILITE 7000 LT family of oxygenators is made of plasma-tight **hollow fibre** membranes. (Our previous oxygenators were sheets wound around a spindle - **membrane oxygenators**). In hollow fibre oxygenators the gas flow takes place through the lumen of the fibres. Blood is in contact with the outer side of the fibres, so that oxygen can diffuse into the venous blood while carbon dioxide is eliminated from the blood. The effectiveness of the device is due to the material (polymethylpentene) being non-porous thus presenting the patient's blood with a heparin-coated, biologically inert surface through which gas transfer occurs. As there are no pores, the membrane does not "wet out" in use. As the material is both thin-walled and in capillary form, the device can produce high gas transfer with a very small priming volume and very little platelet activation.

Gas transfer

Oxygen transfer capacity of the Medos membrane oxygenator is related to the blood flow, the FiO_2 of the sweep gas and the degree of oxyhaemoglobin desaturation of the blood entering the membrane. The 0800 oxygenator will transfer approximately 55-70 mlO_2/min at the rated blood flow (maximum capacity) of the membrane.

MEDOS Oxygenators

These come in 3 sizes:

- 0800 neonatal <4.5kg
- 2400 paediatric 4.5kg - 18kg
- 7000 adult >18kg

They are quick and easy to prime and have a small priming volume.

Gas exchange is very efficient with minimal sweep gas requirements.

Typical sweep gas values for neonatal VV ECLS would be:

0.3L/min (1ml of sweep gas for every 1ml of pump flow e.g. 350ml/min pump flow would require 350ml/min sweep gas flow)

Blender 100% for VV ECLS. (It is now policy to set the blender at 60% for VA ECLS as this seems to reduce the incidence of "stun" – see manual)

These oxygenators clear CO_2 very efficiently. If the patient CO_2 is low, reduce the sweep gas but to no less than 0.2l/min. If the CO_2 is high, the sweep gas flow should be increased to reduce it.

Model Number	Priming Volume (ml)	Surface area (m^2)	O_2 and CO_2 Transfer (ml/min)	Max blood flow (l/min)	Max Gas flow (l/min)	Min Gas Flow (l/min)
0800 LT	55	0.32	55-70	0.8	1.6	0.2
2400 LT	95	0.65	55-70	2.4	4.8	0.2
7000 LT	275	1.9	40	7.0	14	0.2

Table 1: Nominal Medos Oxygenator specifications

Pressure Characteristics of the Medos Oxygenator

As the blood is pumped through the plasma tight hollow fibre oxygenator, a pressure gradient is produced between the blood inlet and outlet ports. The pre-membrane pressure is usually between 45-65 mmHg (normally 55 mmHg) and should always be slightly greater than the post-membrane pressure which is usually 40-60 mmHg (normally 50 mmHg). **Pressures are related to blood flow rate and arterial catheter size.** (See Physiology of ECLS). The pressure gradient should be approximately 5-20 mmHg when measured by pressure transducers. If there are signs of oxygenator failure, the pre and post membrane pressures must be checked. A clot or obstruction will cause a marked increase in the pre membrane pressure and a widening Trans membrane pressure gradient as the membrane starts to fail.

Oxygenator Failure is characterised by:

- An **increasing** pump and patient $PaCO_2$ and a **decreasing** pump and patient PO_2 (>13 KPa)
- An **increasing** trans membrane pressure gradient

Oxygenator failure can be caused by:

- Water/moisture

Note: "Wet oxygenator" is usually characterised by an **increasing** pump and patient $PaCO_2$ and **a stable** pump and patient PaO_2

- Fibrin/clot formation

Note: Characterised by **increased** $PaCO_2$ and **decreased** PaO_2
Subjecting the oxygenator to high outflow pressures (greater than 450 mmHg)

What to do in the event of a "wet" oxygenator (see guideline 19)

- Increase sweep gas to maximum for size of oxygenator for 2-3 minutes with blender at 100%
  - Medos 0800 Max sweep gas flow 2.0L/min
  - Medos 2400 5.0L/min
  - Medos 7000 10.0L/min
- Repeat blood gas after 2-3 minutes at this gas flow
- Observe circuit closely for any bubbles
- If no improvement call Perfusionist and ECLS Physician

Important facts

It is essential that the blood compartment pressure be greater than that of the gas compartment during the priming and operating phase. Over-pressurisation caused by blockage of the gas exhaust port will force gas into the blood compartment resulting in air emboli to the patient. The two relative pressures (blood and gas) can be maintained by proper positioning of the device and restricting the gas flow.

Supersaturation

To prevent excessive oxygen saturation of the blood during recirculation off bypass, stop all perfusion gas to the oxygenator or set the air/O_2 blender at 0.21 FiO_2. Failure to comply with this manoeuvre could cause foaming in the blood compartment and oxygen emboli to the patient.

Be sure to turn the gas on after going back onto ECMO or increase the blender back to the previous level.

During idling on VA ECLS (usually 60-80 ml/min) supersaturation may occur if sweep gas is kept at an FiO_2 of 1.0. Blended oxygen should be used at this time (FiO_2 0.3-0.4 max).

If supersaturation of blood occurs during idling:

- Take the patient off ECMO
- Turn the blender to 21% O_2 (air)
- Turn the pump flow off
- Aspirate the bubbles from the high pressure pigtail
- Check the top of the heat exchanger for bubbles
- If bubbles continue to come out of solution, an exchange transfusion with deoxygenated blood (PRBCs) may need to be done to decrease the saturation below 100% (use 100 ml PRBC's)

- Turn the pump on and check all bubble trap areas for gas
- Turn the blender back to 60% (for 70 ml/min)
- Place patient back on ECMO

The Centrimag Version 1 Console

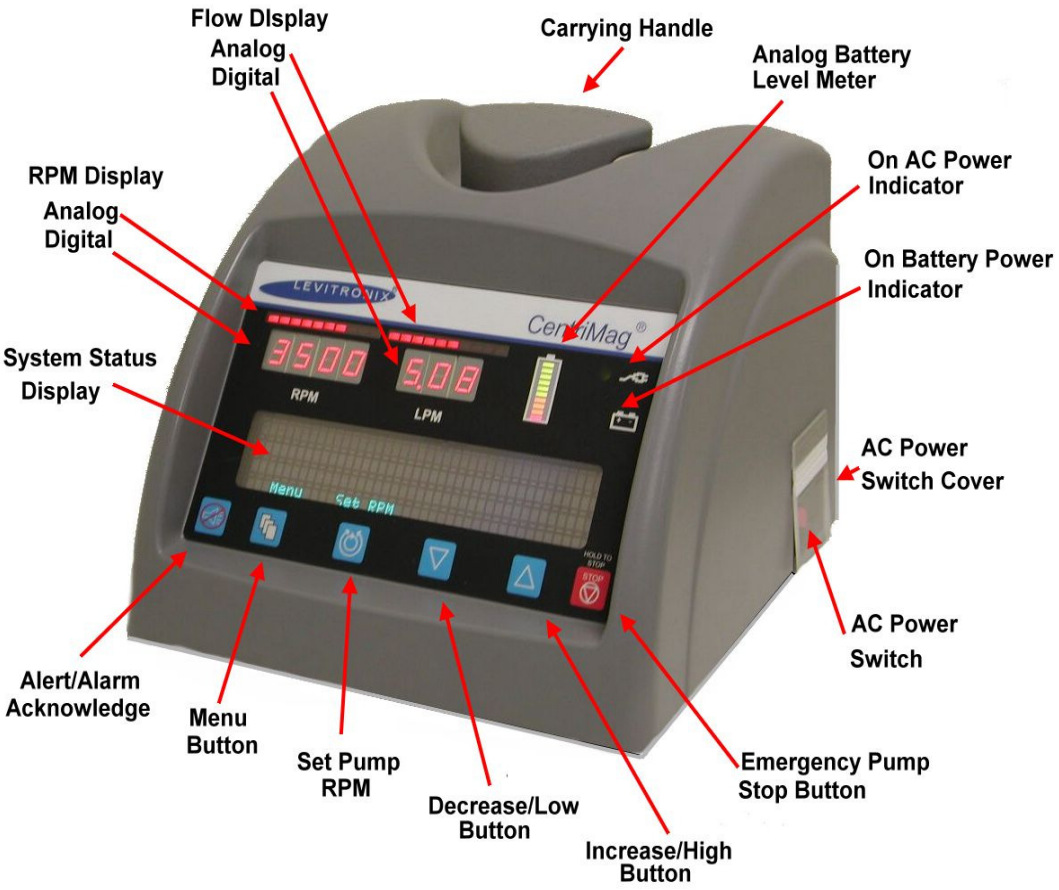


Fig 1

CentriMag® Control Console **Fig 1**

The Levitronix CentriMag® Console (Figure 1) is a microprocessor-based device. An alphanumeric screen is used to display monitored data, system options, and menus. Operator settable alarms and parameters are accessible via the system menus.

Console Back Panel

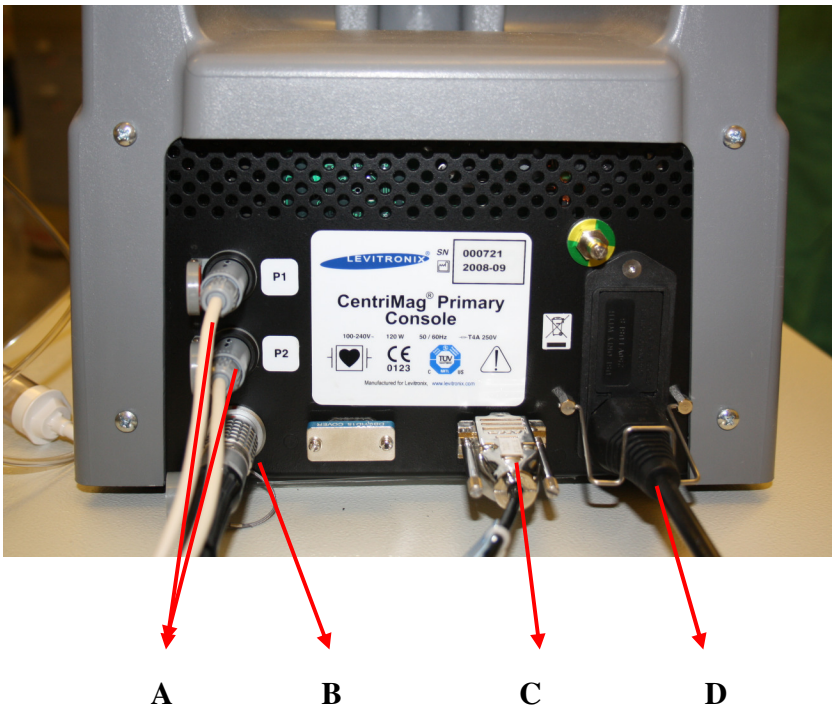


Figure 2. Console Back Panel

Console Back Panel: (Fig 2)

From Left:

Pre and Post membrane pressure cables (P1 & P2) connected	**A**
Motor cable connected	**B**
Middle: flow probe connected	**C**
Right: mains cable connected	**D**

The mains console on/off (switch **E)** is located on the right hand side and is protected by a cover

Figure 3. Console Right Hand Side:

CentriMag® Operator Control Panel

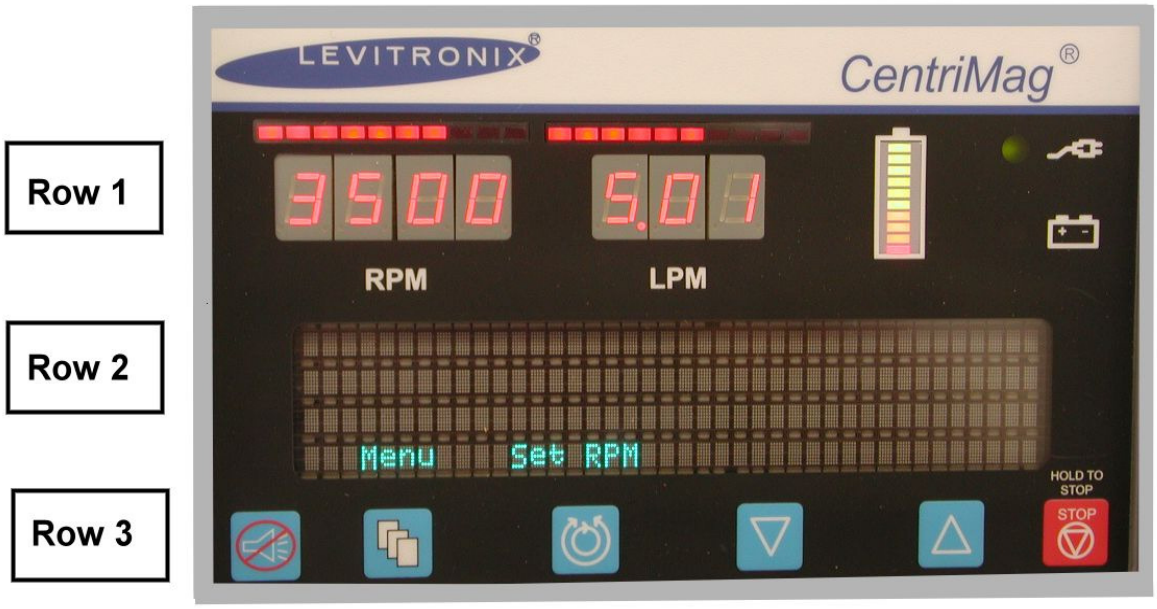


Figure 4

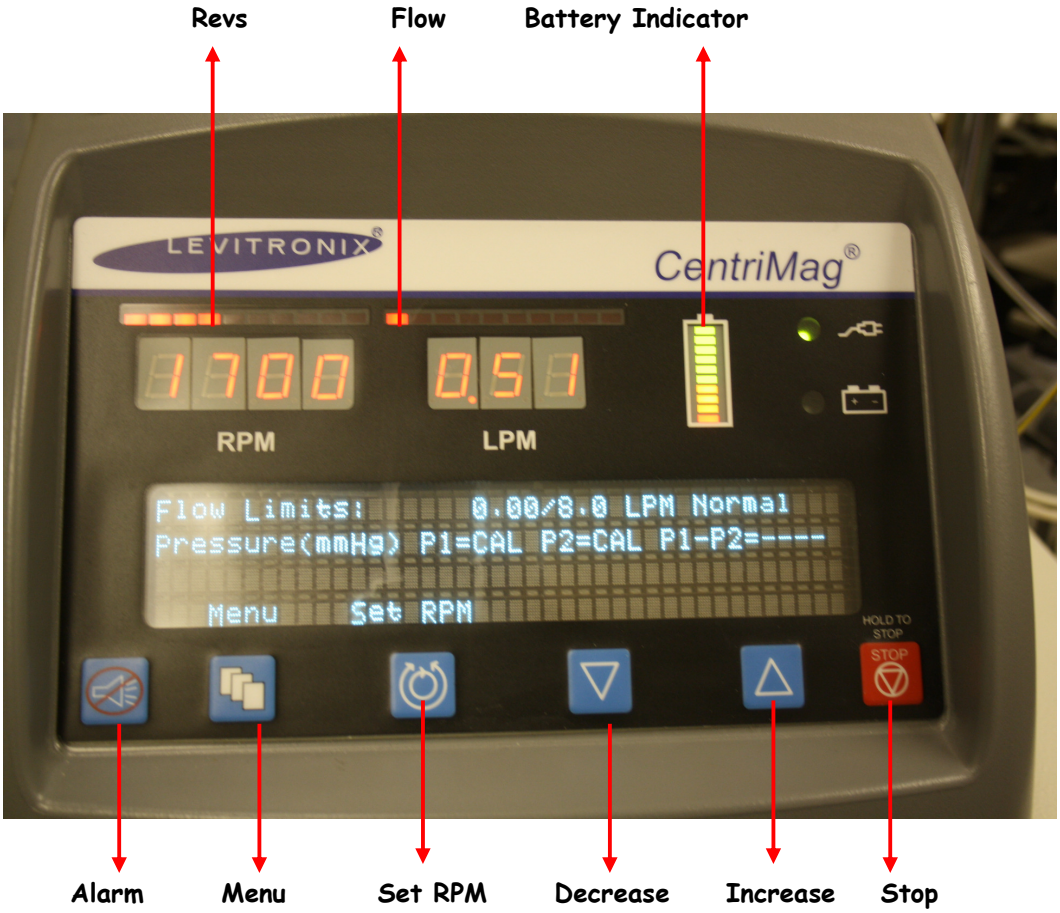
The CentriMag® Console Operator Control Panel (Figure 4) contains three rows of displays:

Row 1 includes indicators (bars and LED's) for the Blood Pump's speed (RPM), flow rate (LPM), and power source (battery gauge and battery icon for battery power and plug icon for AC power).

Row 2 consists of a four-line alphanumeric vacuum fluorescent display. The top two lines on the display are used to display system status. The third line is used to display the selected system parameter, and the bottom line displays the four soft keypad descriptions for the active screen.

Row 3 consists of six keypads. The first keypad (furthest to the left) silences the alarm audio, and the last on the right stops the Blood Pump. The other four keypads from left to right are: menu options (**MENU**) Blood Pump speed adjustment (**SET RPM**) and menu item adjustment (**DECREASE) (INCREASE)**.

Console Front Panel



Start the blood pump by depressing the SET RPM keypad. Depress the INCREASE arrow until the flow rate is at the required level.

The flow is adjusted by depressing the SET RPM keypad and then using the INC/DEC arrows to increase or decrease flow.

Flow Limits and Pressures

Both the lower and upper flow alarm limits are displayed on the 1st line of the main screen

Pre-membrane pressure (P1) and **Post membrane pressure** (P2) are displayed on the 2nd line of the main screen. This line also displays the difference in P1 and P2 (P1-P2). P1 & P2 will display a pressure down to -99mmHg. If the pressures measured are less than this then the following will be seen on the readout "**---**". This will only happen if the inlet pressure is being measured on either P1 or P2.

These will fall as flow is weaned and less blood passes through oxygenator. Under normal circumstances there will be a relatively small drop in pressure across the oxygenator. The pressures will rise and fall with the pump flow rate.

Pressure 3 (Inlet Venous pressure) is displayed on the patient monitor. It records the negative pressure generated by the pump and should be viewed as an important trend indicator and is dependant on the cannula type, cannulation site, targeted flows and patients filling status. Ideally it should not be allowed to drop below -60mmHg (up to -99 for adult patients). It will usually sit between +5 and -50mmHg.

Battery Operation

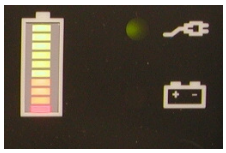
The Console is designed for operation on AC power; however, it also contains an internal rechargeable battery and charger. If a power failure causes loss of AC power or patient transport is necessary, a new fully charged internal battery will operate the Console and Pump for a minimum of 60 minutes at 5500 rpm, 3 Lpm. The switch from AC power to battery power is automatic and is accomplished without interruption of patient support.

THE LEVITRONIX CONSOLE MUST BE CONNECTED TO THE MAINS AT ALL TIMES, INCLUDING WHILST STORED FOLLOWING USE.

If a LOW BATTERY alert message is displayed, AC power should be restored as quickly as possible. Each bar on the battery life equates to approximately 5minutes of battery time.

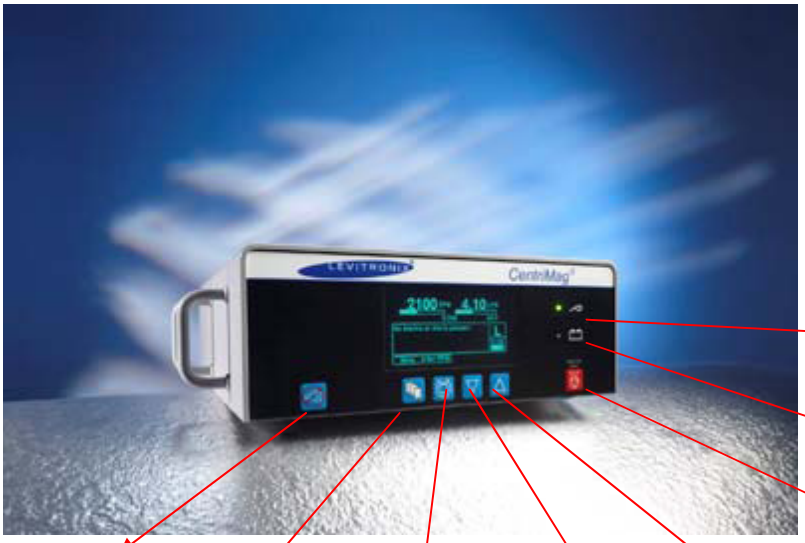
The symbols used on the Console and their meanings are listed below.

SYMBOL	NAME	*DESCRIPTION*
Controls – Front Panel		
	Alarm Acknowledge	Depressing this keypad signals the Console that the user is aware of an alarm/alert condition(s). The Console will silence the audio alarm/alert indicator for a fixed period depending upon the nature of the Alert/Alarm.
	Menu	Depressing this keypad will allow the user to select system settings to view or modify (e.g., minimum flow alarm, audio volume, language).
	Set Pump RPM	If **SET RPM** is displayed above this keypad, on the alphanumeric display screen, depressing this keypad will allow adjustment of the Blood Pump speed. If **EXIT** is displayed above this keypad on the alphanumeric display, depressing this keypad will disable the ability to adjust Blood Pump speed and maintain the Blood Pump speed at the displayed rate.
	Decrease/Low	This keypad is used to select/modify the value for the displayed item to be adjusted.
SYMBOL	**NAME**	***DESCRIPTION***
	Increase/High	This keypad is used to select/modify the value for the displayed item to be adjusted.
	Emergency Pump Stop	When depressed for at least 2 seconds this keypad will cause the Pump RPM to immediately be set a zero causing the Blood Pump to stop.

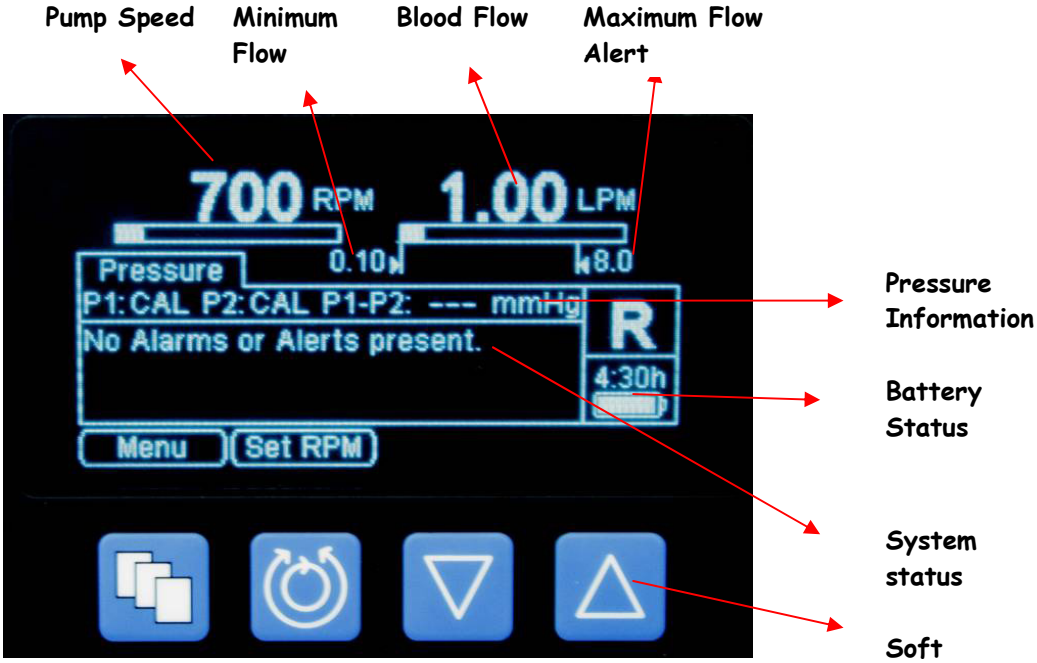
	ON/OFF Switch	Power switch located on the lower the right side panel of the Console. The power switch is recessed, and covered with hinged plastic cover to prevent inadvertent actuation. Switching to off disables all functions and displays except for battery charging.
Indicators – Front Panel		
	Pump Speed	Blood Pump speed (**RPM**) Display: The Top portion of the Blood Pump Speed display is an analog representation of the Blood Pump Speed (shown as dashes); one LED (one dash) is equivalent to 550 RPM. The bottom portion of the Blood Pump Speed display is a 4-digit display that provides a digital representation of the Blood Pump speed in RPM.
	Flow Rate	Flow rate (**LPM**): The Top portion of the Flow Rate Display is an analog representation of the Blood Pump Flow (shown as dashes); one LED (one dash) is equivalent to 1.0 LPM. The bottom portion of the Flow Display is a 3-digit display that provides a digital representation of the Blood Pump Flow in LPM.
	Power Source	AC plug icon indicator (GREEN DOT) illuminates when operating under AC power. Battery icon (GREEN DOT) illuminates when operating under battery power. Battery charge LED indicates charge remaining in the battery. Full battery charge is indicated until nominal battery charge falls below 80%.

The Version II Centrimag Console

Front Panel – Primary Console



Digital Display – Primary Console



Levitronix Monitor

Alarm/Alert Acknowledge

Menu Button

Set Pump RPM

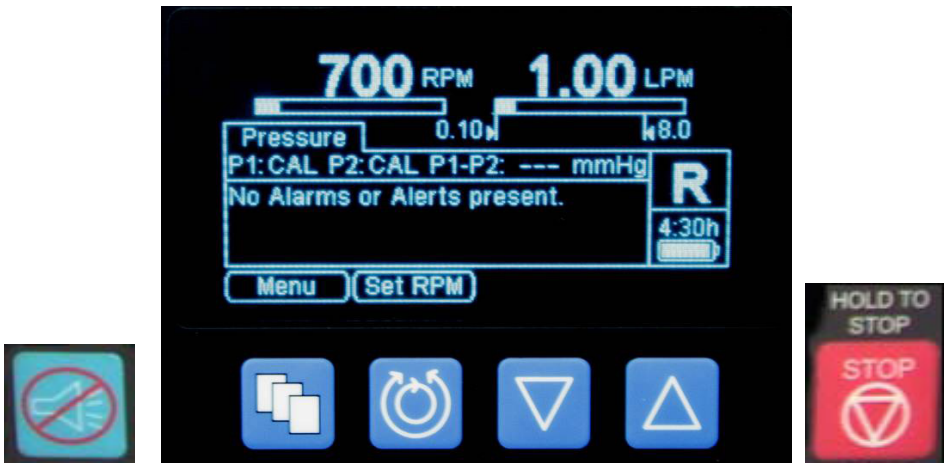
Increase/High Button

Decrease/Low Button

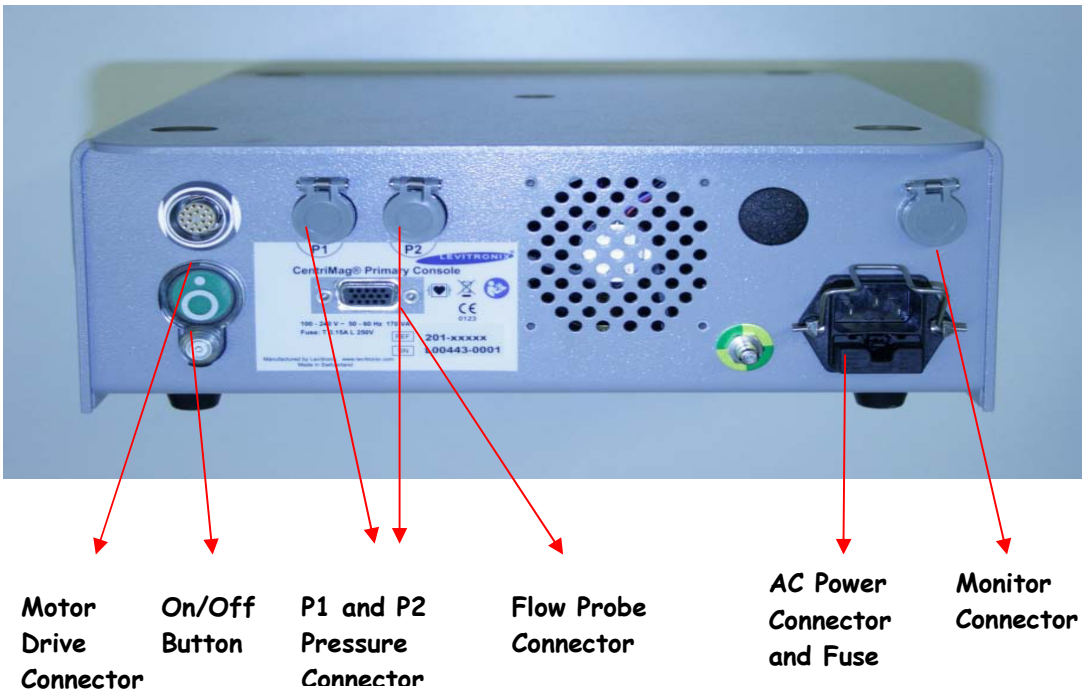
Emergency Pump Stop Button

Controls on the Primary Console

The Primary Console Control Panel contains three rows of displays. **Row 1** includes indicators (bars and digital) for the Blood Pump's speed (RPM), flow rate (LPM), flow limits (LPM), and pressure measurements (mmHg). The top two lines of **Row 2** on the display are used to display system status. The bottom line displays the four soft keypad descriptions for the active screen. The remaining Battery Time is also provided in Row 2 with both digital and bar indicators. **Row 3** consists of six keypads. The first keypad (furthest to the left) silences the alarm audio and also serves as the keypad to be depressed to acknowledge the alarm condition, and the last on the right stops the Blood Pump. The other four keypads from left to right are: menu options (**MENU**) Blood Pump speed adjustment (**SET RPM**) and menu item adjustment (**DECREASE**) (**INCREASE**).



Console Back Panel



Battery Operation

The Console is designed for operation on AC power; however, it also contains an internal rechargeable battery and charger. If a power failure causes loss of AC power or patient transport is necessary, a new fully charged internal battery will operate the Console and Pump for a minimum of 120 minutes at 3500 rpm, 5.5 Lpm for the CentriMag Blood Pump and approximately 180 minutes at 1.0 LPM and 3,000 RPM when used with the PediVAS Blood Pump. The remaining battery life is displayed on the console. The switch from AC power to battery power is automatic and is accomplished without interruption of patient support. Whilst on battery the alert will sound every 16mins.

THE LEVITRONIX CONSOLE MUST BE CONNECTED TO THE MAINS AT ALL TIMES, INCLUDING WHILST STORED FOLLOWING USE.

If a LOW BATTERY alert message is displayed, AC power should be restored as quickly as possible or the patient transferred to another primary or backup unit as the blood pump may stop at anytime.

Flow Probes

Flow Probes in two sizes are available for use with the 2nd Generation Primary Console. Each Flow Probe is a reusable, non-patient contacting ultrasonic Flow Probe which is optimized to detect flows from 0-10.0 LPM or 0 – 3.0 LPM depending on probe size. The flow probes provided with the 1st Generation Primary Console are **not interchangeable** with and will not work when connected to the 2nd Generation Primary Console. The flow probes can detect retrograde flow. Retrograde flow of up to 2.0 LPM is displayed as a negative number such as "-0.65 LPM". Retrograde flow greater than 2.0 LPM, is displayed as downward arrows "vvvv LPM". A disconnected or malfunctioning probe will display dashes "--.--". If the probe detects forward flow of more than 10 LPM then it will display as "^^^^ LPM".

Version II Flow Probe

Alarms

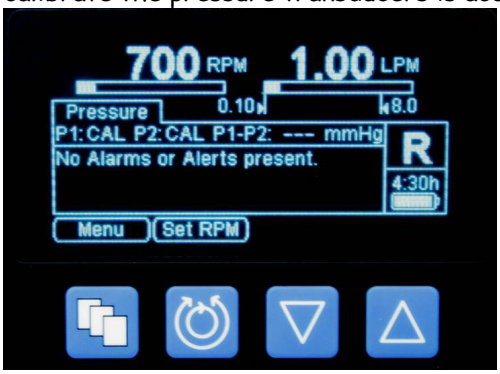
The Primary Console continues Blood Pump operation during an alert or **MOTOR ALARM** condition, and stops the Blood Pump during an alarm or **MOTOR STOPPED** condition. Both the visual and audio alert indicators are active, even if the problem has resolved, until the alert/alarm condition is acknowledged by pressing the ALARM ACKNOWLEDGE keypad. The user must acknowledge the alert/alarm to silence the audio indicator and to determine if the alert/alarm condition has resolved or is unresolved. If an alarm/alert condition occurs, the audible alarm/alert sounds and an alarm/alert message indicating the cause(s) of the alarm/alert appears on the display. Depressing the ALARM ACKNOWLEDGE keypad mutes the audible alarm. The alarm/alert message will be continuously displayed on the top two lines of **Row 2** of the Primary Console's display as long as the alarm/alert condition exists.

Pressure Display

Note: The pressure monitoring system has a functional range of (-) 100 mmHg to (+) 900 mmHg with a display resolution of 1 mmHg. If one channel exceeds either limit, the values displayed on the specific channel as well as the difference will be invalid, which is indicated by either "vvvv" for values below -100 mmHg respectively "^^^^" for values above 900 mmHg.

Configuring the Console

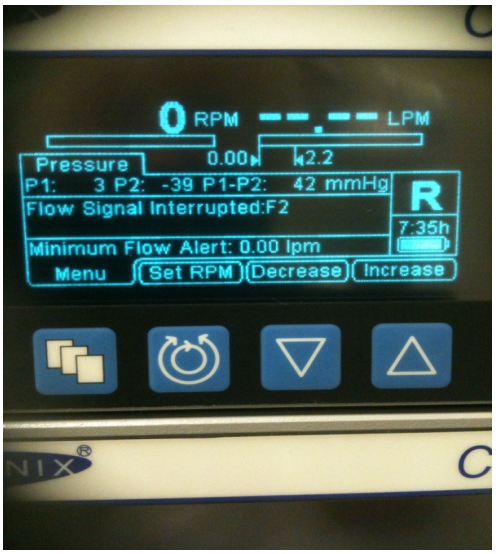
The ability to set Pump speed, set flow alert thresholds, select the flow alert sensitivity, select the language, select the set speed resolution, enable and disable the pressure measurement capability, and calibrate the pressure transducers is accessed through the **MENU** keypad.



Depressing the **SET RPM** keypad allows the user to increase or decrease the speed of the pump using the UP and DOWN **ARROWS**.

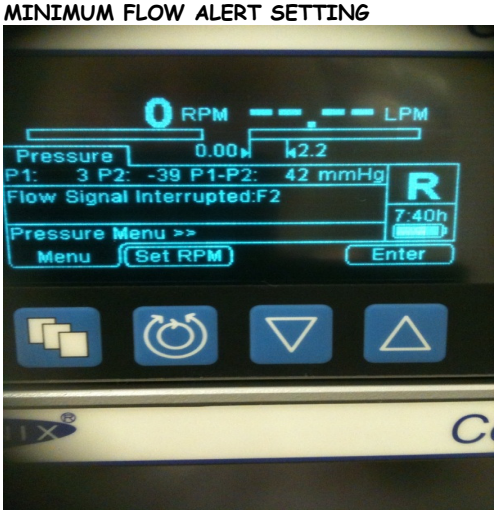
Depressing the **MENU** keypad leads to different user options that can be accessed by scrolling through the options and using arrow keys to adjust parameters:

MAIN MENU	EXTENDED MENU
▪ MINIMUM FLOW ALERT SETTING	- PRESSURE DISPLAY (for one or two pressure probes)
• MAXIMUM FLOW ALERT SETTING	- SPEED STEP RESOLUTION
• PRESSURE MENU	- FLOW RANGE SELECTION
- PRESSURE CALIBRATION	- FLOW LIMIT SENSITIVITY
- MINIMUM PRESSURE (P1) ALERT SETTING	- FLOW RECORDER SPEED(Monitor only)
- MAXIMUM PRESSURE (P1) ALERT SETTING	- SUPPORT TYPE
- MINIMUM PRESSURE (P2) ALERT SETTING	- LANGUAGE SELECTION
- MAXIMIUM PRESSURE (P2) ALERT	

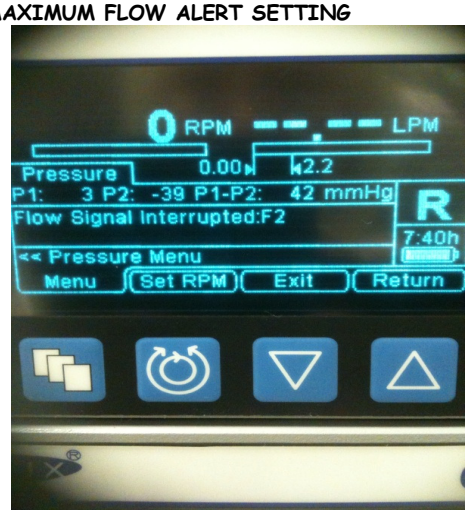


MINIMUM FLOW ALERT SETTING

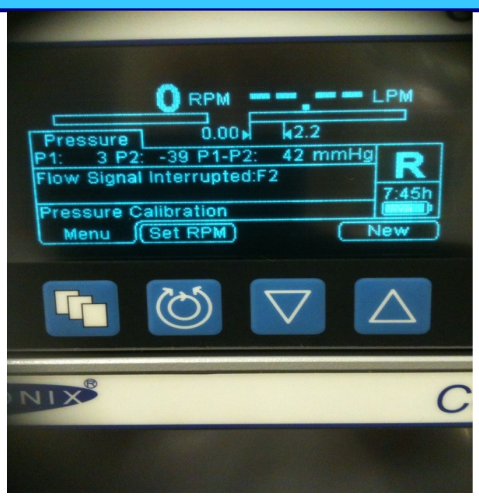
MAXIMUM FLOW ALERT SETTING



ENTER PRESSURE MENU

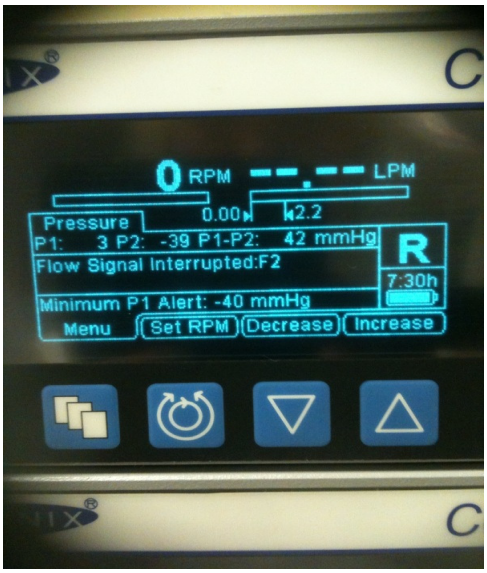


SCROLL THROUGH BY PRESSING MENU



NEW PRESSURE CALIBRATION

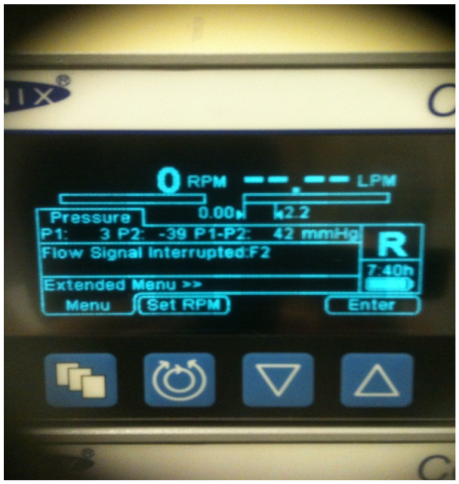
CALIBRATE PRESSURE 1 & 2



SET MINIMUM PRESSURE ALERT
FOR P 1 & P2

SET MAXIMUM PRESSURE ALERT
FOR P 1 & P2

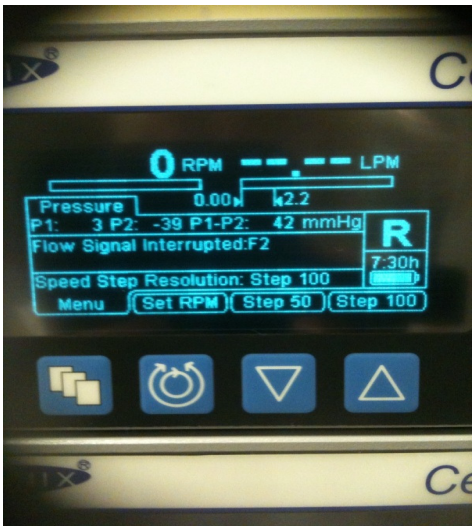
PRESS RETURN TO MAIN MENU



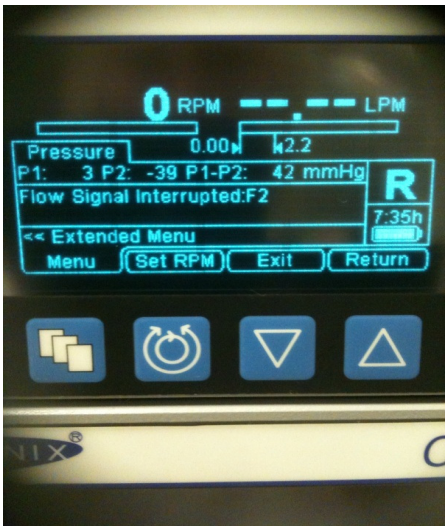
SCROLL DOWN MENU ENTER EXTENDED MENU

SCROLL THROUGH EXTENDED MENU

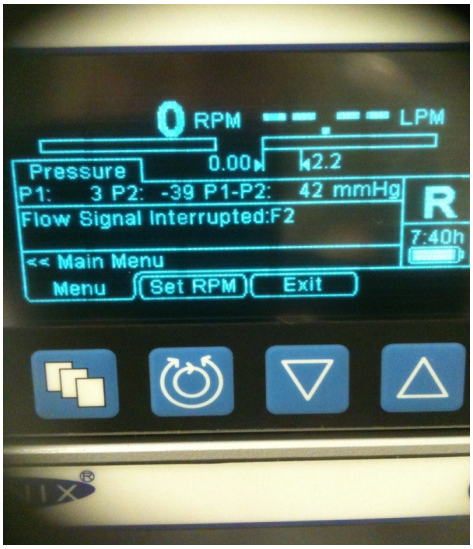
PRESSURE DISPLAY



ADJUST SPEED STEP RESOLUTIONS



RETURN TO MAIN MENU



EXIT MAIN MENU

Alarms and Alerts

The Levitronix CentriMag® Console alarm strategy is based on the following philosophy. Audio and visual advisories are divided into two groups, System **Alerts** and System **Alarms**, to warn the operator of conditions that may interrupt patient support or damage the Blood Pump, Motor Drive Unit or Console. A normal operating condition is free of any alerts or alarms and is classified as a green state of operation. **Alert Advisories** activate when the system is about to, or has entered, an unsafe but resolvable operating state (yellow state). **Alarm Advisories** activate when the system is about to, or has entered, an unsafe state of operation which may be hazardous to the patient or operator (red state). The table 8 below illustrates the fundamental strategy:

Table 8: Levitronix Console Alarm/Alert Advisory Strategy

Operating State	Advisory Level	Anticipated Operator Response
Green	None	None
Yellow	Alert	Resolve Fault Condition
Red	Alarm	Resolve Alarm Condition or Switch to back-up Console/Motor

Console / Circuit specifications		
	Pedivas <10 kg	**Centrimag > 10 kg**
Pump Speed Range (revolutions/min)	0 - 5500 RPM.	0 - 5500 RPM.
Pump Flow: Minimum (litres/min)	0.2 L/min	0.5 L/min
Pump Flow: Maximum (litres/min)	1.7 L/min	9.9 L/min
Recommended RPM at initiation of flow	1500	1500
Speed step resolutions	50ml	100ml

Levitronix ECMO Perfusion

Patient Weight	Tubing Pack (code)	Oxygenator	Pump Head (Max Flow l/min)	Art	Ven	Total Prime	Blood Volume	Max Gas Flow l/min	Min Gas Flow l/min
0 – 4.5 Kg	Pedivas CM4833	Medos 0800 LT	Pedivas 1.5	1/4	1/4	155ml	100ml	1.6	0.2
4.5 – 10 Kg	Pedivas CM4833	Medos 2400 LT	Pedivas 1.5	1/4	1/4	195ml	150ml	4.8	0.2
10 – 18 Kg	Intermediate CM4841	Medos 2400 LT	Centrimag 9.99	1/4	3/8	270ml	200ml (by discussion)	4.8	0.2
> 18 Kg	Centrimag CM4840	Medos 7000 LT	Centrimag 9.99	3/8	3/8	490ml	300ml (by discussion)	14	0.2

A litre of Plasmalyte 148 with 1000 iu of heparin added should be used to prime all circuits. Blood should be routinely added to 1st and 2nd sized circuits but only by prior discussion with consultant on a case by case basis for 3rd and 4th sized circuits.

Patient / Circuit specifications				
Patient weight	**< 4.5kgs**	**4.5-10kgs**	**10-18kgs**	**> 18kgs**
Arterial connector size	**1/4 "**	**1/4 "**	**1/4 "**	**3/8 "**
Venous connector size	**1/4 "**	**1/4 "**	**3/8 "**	**3/8 "**
Pedivas Pump Prime	14ml	14ml	-	-
Centrimag Pump Prime	-	-	31ml	31ml
Minimum Pump Blood Flow	200ml/min	200ml/min	500ml/min	500ml/min
Oxygenator	Medos 0800 LT	Medos 2400 LT	Medos 2400 LT	Medos 7000 LT
Oxygenator priming volume	55ml	95ml	95ml	275ml
Max Blood Flow through Oxygenator	800 ml/min	2400 ml/min	2400 ml/min	7000 ml/min
Max Gas Flow through Oxygenator	1.6 l/min	4.8 l/min	4.8 l/min	14 l/min
Min Gas Flow through Oxygenator	0.2l/min	0.2l/min	0.2l/min	0.2l/min
Flow Probe	1/4 "	1/4 "	1/4 "	3/8 "
Total Circuit prime	**155ml**	**195ml**	**270ml**	**490ml**

Cannulae Selection Guidelines							
VA			VV				
Biomedicus cannulae			Origen DL cannula		Avalon bi-caval cannula		
Weight	Arterial cannula size	Venous cannula size	Weight	Cannula size	Weight	Cannula size	Max flow
2 – 3 kg	8F	8F/10F	2.5 -3.5 kg	12F	2.5-5 kg	13F	0.5l/min
3 – 6 kg	10F	10F/12F/14 F	3.5 – 6 kg	15F	4 -9 kg	16F	0.9l/min
6 – 11 kg	12F	14F	5 – 11 kg	18F	<12 kg	19F	1.2l/min
11 – 14 kg	12F	14F/15F	>11 kg	N/A	<15 kg	20F*	1.5l/min
14 – 20 kg	14F	15F/17F	-	-	<20 kg	23F	2.0l/min
20 – 30 kg	15F	17F/19F	-	-	Size clinically **	27F	3.5l/min
30 – 40 kg	17F	19-21F	-	-	Size clinically **	31F	5.0l/min
> 40 kg	21F	21-23F	-	-	> 75 kg	31F PLUS additional drainage cannula	

*** Note the large step-up in size of the 20F vs the 19F Avalon bi-caval cannula. May not be suitable unless patient is quite long.**

**** Patients greater than 20kg there are no set guides re cannula size and the experience from Leicester is to size clinically with the patient.**

The Centrimag Motor

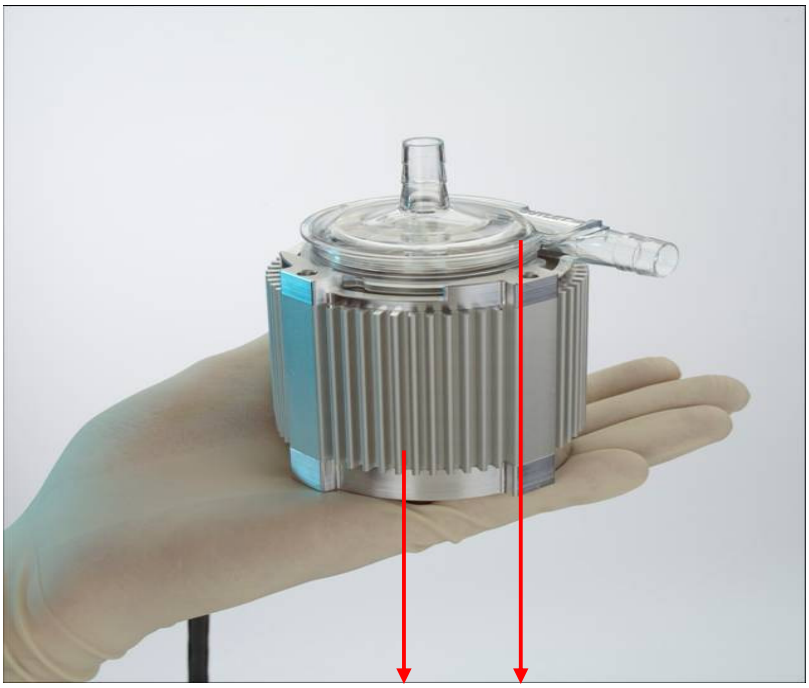


Fig 1

The drive motor **A** houses the pump **B**

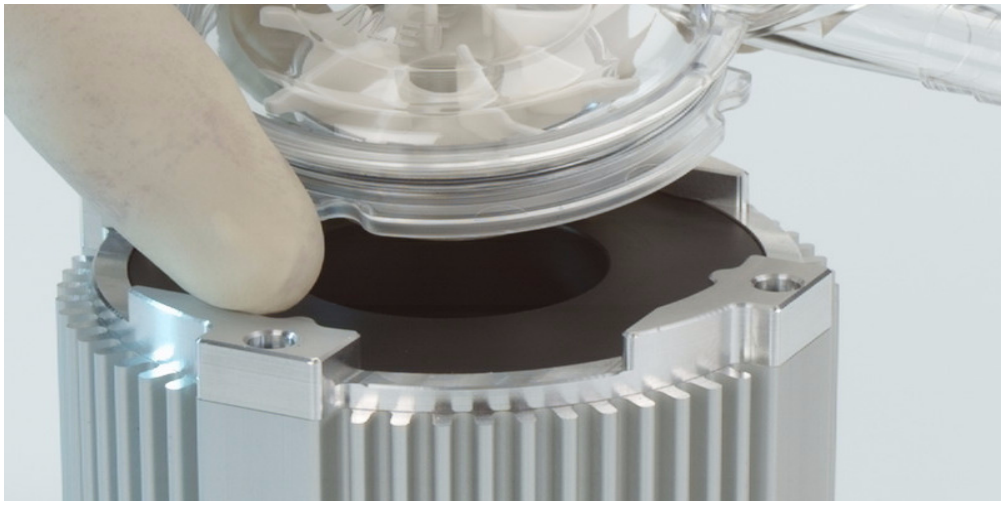


Fig 2

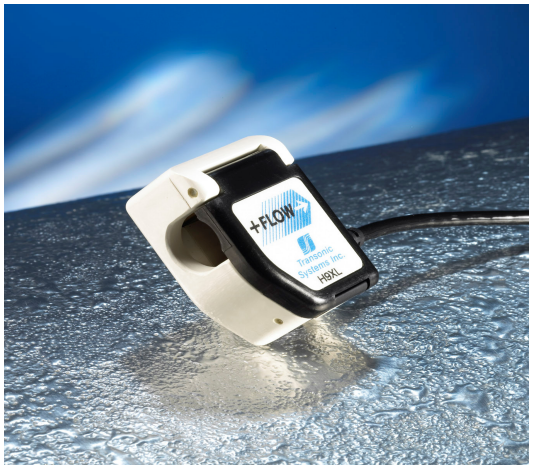
Match the grooves on the pump with those on the motor.

Rotate counter-clockwise until the pump locks into place.

Thread the retaining screw clockwise to secure into place.

Flow Probe

The flow probes provided with the 1st Generation Primary Console are **not interchangeable** with and will not work when connected to the 2nd Generation Primary console



Version I console flow probe

Version II console flow probe

Connect the flow Probe to Blood Pump Outlet tubing - ensure arrow is aligned in direction of flow.

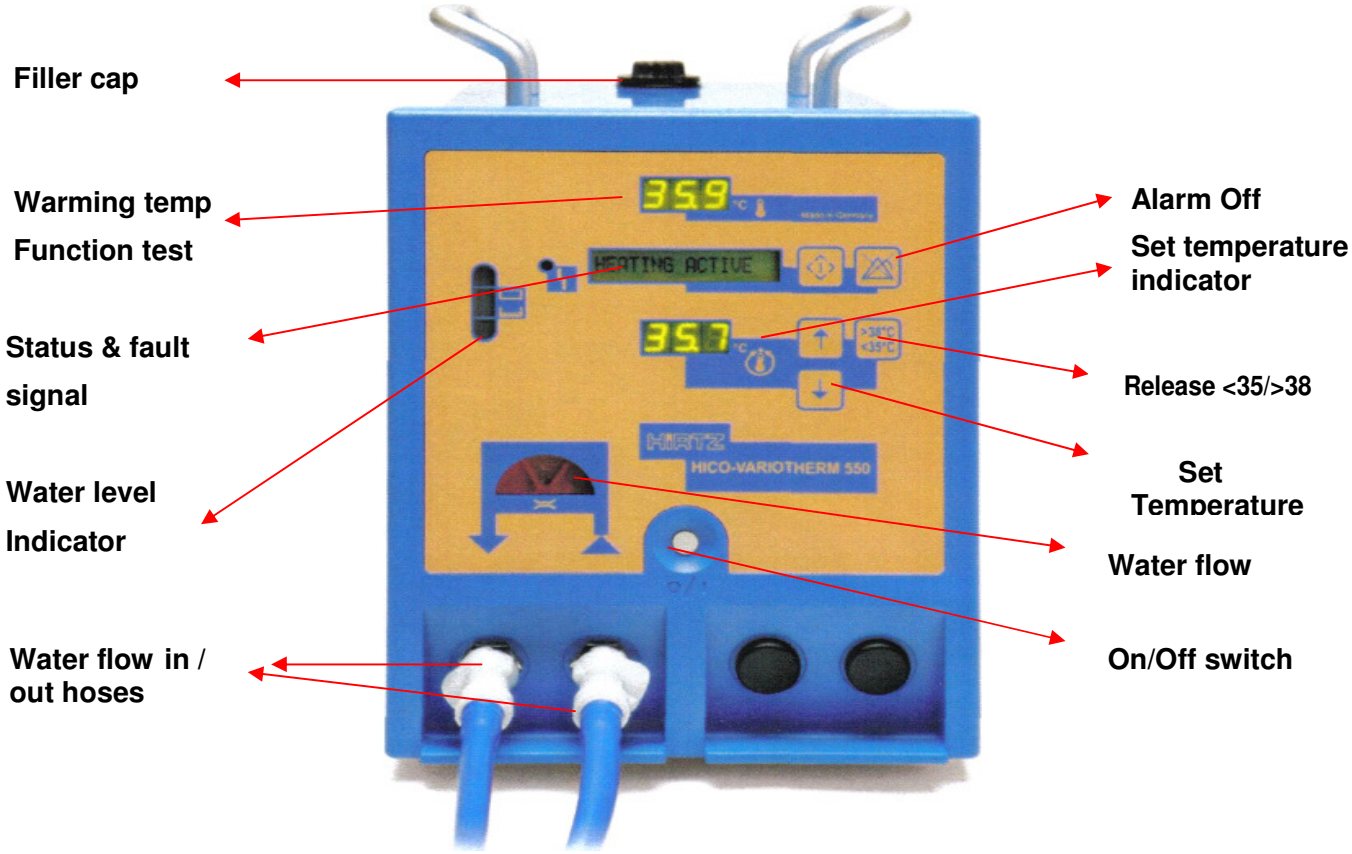
The flow probe should be sited on the circuit side of the bridge.

There are 2 sizes of flow probe:

H7XL fits 1/4 " tubing

H9XL fits 3/8 " tubing

Variotherm 550 water heather/cooler



If there is a fault with the pump unit's independent safety device, or it does not respond during the automatic or manual function test, the alarm signal "ALARM TEST FAIL" and "CALL CUSTOMER SERVICE" is displayed, along with the red fault light and the signal tone. The alarm cannot be suppressed, making it necessary to switch the unit off.

Should the alarm signal sound again after switching on and after a longer holding time (1-2 hours), the unit must be checked by the medical physics department.

If the water tank temperature falls below the measuring range (approx. 9 °C) the display shows the alarm signal "LOW TEMPERATURE" and "CHECK UNIT", along with the red fault light, the signal tone and the temperature indicator 000. The alarm cannot be suppressed, making it necessary to switch the unit off using the power switch.

The alarm signals "CHECK UNIT" and "CALL SERVICE" along with the red fault light and the signal tone are given for various defects. The alarm cannot be suppressed, making it necessary to switch the unit off using the power switch. In this case the unit must be checked by the medical physics department.

Power Failure Alarm

Should the power fail during operation, it is signalled by the signal tone and the red fault light on the pump unit. Call the perfusionist to change the heater.

The CDI 101 inline saturation/Hb/Hct monitor

The CDI 101 (Terumo) is a continuous Hb / Haematocrit and Oxygen saturation (SaO_2) monitoring system. It attaches by probe, clipping into a cuvette which is inserted during priming on the venous limb of the circuit.

The initial setup and testing will be performed by the perfusionist prior to cannulation.

The monitor should be calibrated twelve hourly to ensure accuracy. The Store button on the right side of the screen should be gently pressed which will internally store the displayed values. A venous sample should then be obtained for analysis in the gas machine. When the gas has been completed the recall button is pressed and a calibration window will appear which allows the ECLS specialist to input the corrected values. Pressing the OK button will then display the main screen.

If the monitor needs re booted (display values have been lost) during a run the probe will need to be removed from the circuit (unclip) and placed in the docking mechanism prior to switching off the machine. When the monitor is switched back on the display will show the above screen and the probe should be replaced on the circuit.

The CDI 101 has **no** battery backup therefore the CDI 500 should be used for all patient transfers.

Following decannulation it is vital that the probe is removed from the circuit cuvette and properly seated in the docking mechanism on the side of the monitor to prevent damage to the probe microchip.

The CDI 500

The CDI 500 allows for continuous monitoring of Hb /Haematocrit and Oxygen saturation (SaO_2) and up to eleven blood gas parameters including pH, PCO_2, PaO_2 and BE.

Probes can be attached via the cuvette to both the venous and arterial limbs of the circuit.

In line with the CDI 101, calibration of the CDI 500 should be performed twelve hourly.

The CDI 101 has **no** battery backup therefore the CDI 500 should be used for all patient transfers.

All case data is automatically stored to a removable flash card. If a hard copy is required this can be printed via the internal printer and stored in the patient's case notes.

Gas delivery system

Figure 8 Type: High/Low flow Oxygen flowmeter & Air/Oxygen blender

General Description:

The gas delivery system is composed of 3 basic components:

- A high & low flow O_2 flowmeter assembly
- An air/oxygen blender.

The high O_2 flowmeter has a range of 0-10 LPM (0.5 LPM graduations) and the low O_2 flowmeter 0-1 LPM (0.02 LPM graduations). Inlet connections to the flowmeter are indexed to gas type quick connects so as to prevent errors in hook-up. The air/oxygen blender has an O_2 concentration range of 21-100% with 10% graduations. The blended gas outlet of the blender is fed to the input of the high/low O_2 flowmeter assembly. The output of the flowmeter assembly are routed to the green gas conduit which inputs, after passing through a one-way filter and a blow-off valve, into the gas inlet of the membrane oxygenator.

A blow-off valve protects from high pressure in the gas phase in the event of an obstruction to the exhausting of gas from the membrane oxygenator (blood clot most likely cause). Pressure limit will be 50 mmHg in neonates and infants and 100 mmHg in older children.

Warnings and Precautions:

Test the flow-meter before hooking up to the membrane by opening the needle valve and observing the rise of the flow-meter ball. Closing the needle valve should cause the ball to drop in proportion to the

valve closing.

Periodically observe the flowmeter to ensure that the flow has not varied from the last setting. Medical air can contain moisture or debris that can cause the ball to stick in the glass tube. If this is suspected, tap on the face of the flowmeter. If the ball drops or rises significantly, consider replacement.

Keep the unit cleaned and free of blood and debris.

Do not over-tighten when turning off the flowmeters.

If pump blood gas reading differs drastically from the previous reading without having made any changes, check all the connections, check green tubing is securely connected to flow meter outlet and the membrane oxygenator, check all tubing for leaks and holes and check pressures into the gas delivery system before suspecting the membrane.

Never block the gas outlet of the membrane.

When operating, never leave unattended. Continuous supervision is mandatory for patient safety.

The usual start-up sweep gas flow rate is a one to one gas to blood flow ie the HiLite 0800 oxygenator usually 0.3-0.4LPM with a blood flow of 300-400mls/min. Patients with a high Co2 will require a half gas to blood flow ie 0.2LPM gas flow with a blood flow of 400mls/min to prevent rapid reduction in Co2 and resulting cerebral blood flow changes.

The Pressure Transducers

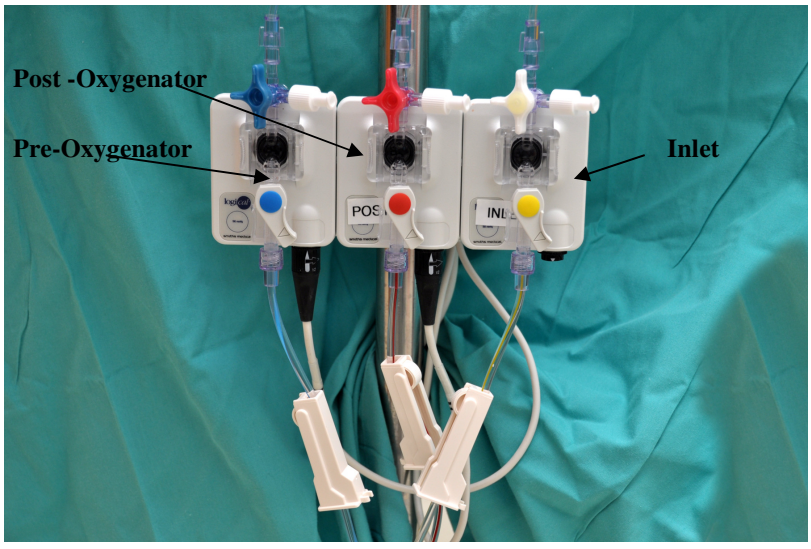


Figure 1

There are 3 pressure transducers **(Figure 1)** which form part of the monitoring system. These, and the flushing lines, are colour coded.

- Blue – pre-oxygenator P
- Red – post-oxygenator P
- Yellow – venous or inlet P

A flushing system similar to the one used for arterial lines is required but with a few differences. The giving set from the Heparin-saline infusion bag has a white roller clamp which is attached to a 3-way flushing line, which incorporates the yellow, blue and red flushing lines

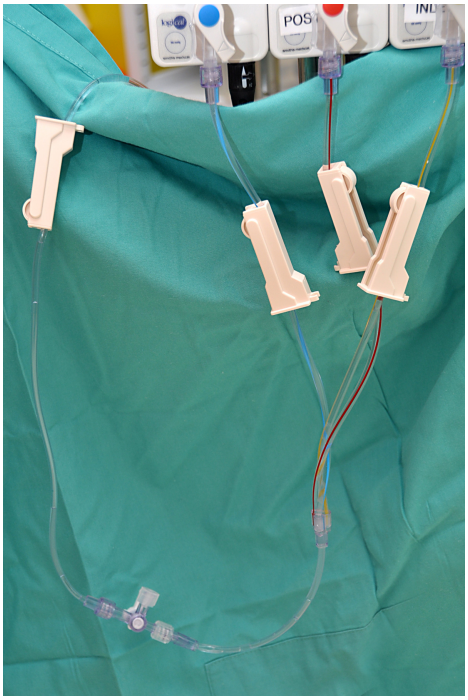


Figure 2

The white roller clamp will remain switched off and will only be turned on if blood is back-tracking beyond the pigtail and the pressure monitoring lines needs to be flushed. (Figure 2)

The blue monitoring line is attached to the pre oxygenator pigtail (#5) and the red monitoring line to the "blood-out" post oxygenator pigtail (#7) The yellow monitoring line is attached to the venous pigtail (#2) and will monitor the negative pressure generated by the pump.

All of the free ports on the 3 way taps should have a Bionector insitu .

Flushing Pressure Lines

White roller clamps are insitu on all of the 3 pressure monitoring lines below the transducers. All three pig-tails will be taped in an upright position. These measures should prevent backflow of blood in to the pressure monitoring lines.

As an additional precaution the pre and post oxygenator pigtails should be flushed if blood is backtracking. To do this, attach a 10ml luer-lock syringe containing heparinised saline to the side port of the 3-way stopcock joining the pressure monitoring line to the pigtail as in figure 3. Turn the tap off to the pressure monitoring line. As this is the high pressure side of the circuit (post-pump) remember to hold the syringe piston! Draw back as usual to de-bubble the connection and inject 2 mls to clear the pigtail. Return the tap to the original position.

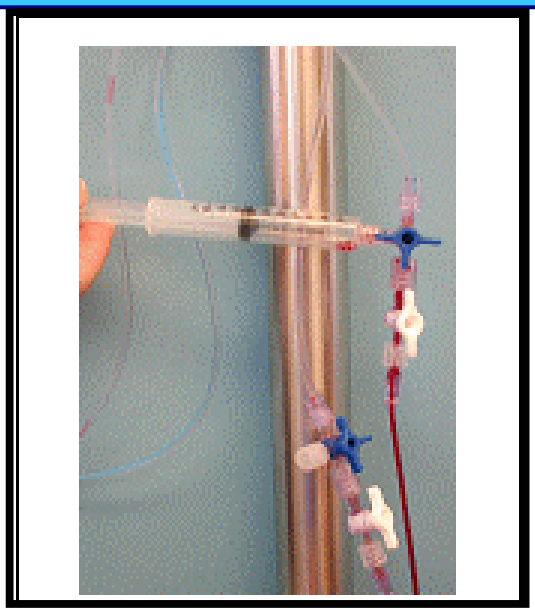


Fig 3

If blood tracks back up the yellow venous pigtail, it can be flushed as described above. Extreme care must be taken whilst doing this to avoid any air entrainment as this pigtail is under negative pressure.

The white roller clamp will need to be opened to do this – remember to close it afterwards.

Bionectors

As an added safety and infection control precaution all pigtails and stopcocks should have a Bionector in situ.

Bionectors should be changed every Wednesday and Sunday as per unit protocol. All pigtails should be aspirated and flushed once per shift to ensure patency. Caution should be exercised whilst flushing/changing Bionector on Pigtail #1 or Inlet pressure line Pigtail #2 as this may result in entrainment of air. If unsure re flushing/changing Bionector on inlet side contact Perfusion or ECLS Coordinator to assist.

Levitronix ECMO Set up Procedure

Hardware Required:

- Levitronix pump console
- Motor
- Motor Holder
- Flow probe ¼ for Pedivas or ⅜ for Centrimag
- Tubing Clamps

Disposables Required:

- Sterile Gloves
- 1000mls Plasmalyte 148 with 1000iu Heparin added
- Sterile pump head - < 10Kg Pedivas or > 10Kg Centrimag
- Oxygenator
- Tubing Pack

Set Up:

Ensuring aseptic techniques throughout inspect all packaging for integrity and sterility ensuring products are within stated expiratory dates.

1. Add 1000iu of Heparin to priming solution (1,000mls Plasmalyte 148)
2. Hang priming circuit on drip stand.
3. Open pump head using sterile gloves.
4. Attach blue tubing of table line set to connector marked inlet on the pump head.
5. Attach the length of tubing (with 2 luers) arrow to the outlet of pump head and other end to oxygenator inlet.
6. Attach red tubing to the outlet connector on oxygenator
7. Ensure all connections are firmly pushed over both barbs on each connector.
8. Place cable ties on connector and secure.
9. Attach a pigtail to all luer ports on tubing set tightening taps as you go.
10. Attach pigtails to top port and post membrane pressure port on oxygenator.
11. Connect gas line to blender.

Levitronix ECMO Priming Procedure

1. Clamp both blue (venous) and red (arterial) tubing approximately 4 inches from the recirculation bag.
2. Attach priming solution to the quick prime line, fill the recirculation bag and close clip.
3. De-air the recirculation bag by removing the cap from the air bleed (3-way tap). Ensure tap is closed and sterile cap replaced when complete.
4. Drop the table lines and remove pump head from motor. (Ensure recirculation bag is higher)
5. Remove the clamp from the blue line and allow the fluid to move through circuit.
6. Open and close tap at top of oxygenator to promote filling.
7. Ensure bridge is filled and clamp.
8. Remove clamp from the red line, take oxygenator out of holder.

9. To de-air oxygenator, turn on it's side (outlet port upwards) allowing all air inside membrane to rise up towards the recirculation bag.
10. Place oxygenator back on holder.
11. Insert disposable pump head into CentriMag motor by twisting anti-clockwise. Once inserted lock in place by tightening the small retaining screw.
12. Turn the pump console on and allow to self test.
13. Attach the flow probe to the arterial return tubing after the platelet pigtail, checking carefully the direction of flow indicated by the blue arrow.
14. Generate forward flow by pressing set RPM followed by the up arrow.
15. Once a healthy flow has been established all air should be removed from circuit, oxygenator and pigtails.
    Note: Flow will be required to remove the smaller bubbles.
16. Hang the table lines back on the drip stand.
17. It is important to check pump head for air. To do this, stop the pump, remove the disposable head and agitate gently. Do not strike the head with any object. Re-attach and restart pump and do final check for air.
18. Any air collected in the re-circulation bag can be removed to prevent any potential for air entrainment.
19. Place Bionector connectors onto all pigtails.
20. Connect up the 3 pressure monitoring lines, zero, and place Bionector connectors onto all other available ports. (See Protocol for ECMO Pressure Monitoring Set Up)
21. With the pump still running, firstly clamp the red outlet tubing above the bridge. Remove the clamp from bridge and clamp blue venous tubing above bridge. (A B V)
22. Turn off the pump and the system is ready for use.

Blood Priming

Blood should be routinely added to the 1st and 2nd size circuits only. Please ensure priming drugs are added to PRBC unit by ECMO specialist prior to proceeding.

For 3rd Size intermediate circuit 3/8 / 1/4 with 2400 Oxygenator and 4th size Centrimag circuit 3/8 / 3/8 with 7000 Oxygenator *NO BLOOD REQUIRED.

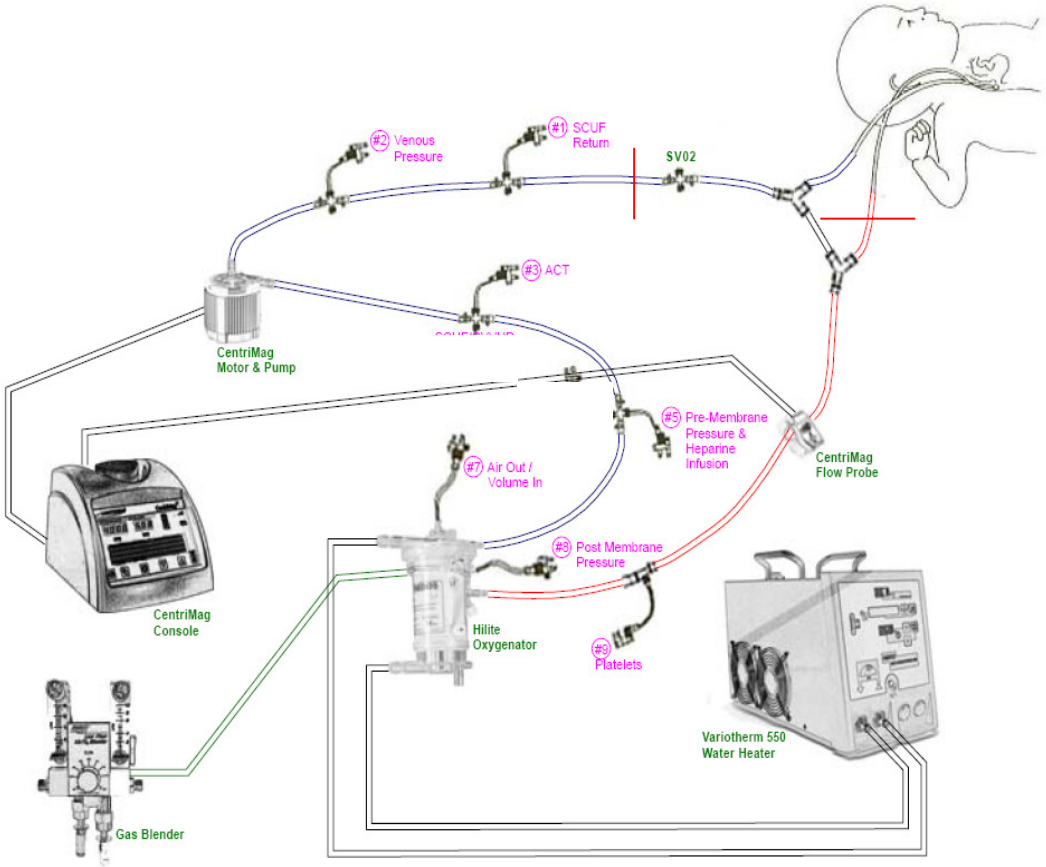
***Unless requested by consultants e.g. post cardiac patient, low Hb or circuit change then:**

Volume to be Added

- Pedivas circuit 1/4 / 1/4 with 0800 oxygenator – 100mls PRBC
- Pedivas circuit 1/4 / 1/4 with 2400 oxygenator – 150mls PRBC
- Intermediate circuit 3/8 / 1/4 with 2400 Oxygenator – 200mls PRBC (by discussion)
- Centrimag circuit 3/8 / 3/8 with 7000 Oxygenator – 300mls/full unit PRBC (by discussion)

Levitronix ECMO Blood Priming Procedure

1. Place one clamp on arterial return line above bridge (A) and one between pigtail #1 and bridge (B) as per diagram below.
2. Use a 50ml luer lock syringe and draw up 50ml blood from the bag bubble free
3. Connect blood syringe to ECMO circuit pigtail # 1 (SCUF Return)
4. Open tap and slowly push blood into circuit. This will force fluid up the bridge on the venous side and into the priming bag.
5. This should be repeated once again for the first circuit size (100ml) and twice for the second size (150ml), three times for third size (200mls) and the full unit for the largest circuit size.
6. Once complete remove clamp B and place on venous return line above bridge.
7. Circulate through bridge until required for use and check a blood gas.
8. Remember there is **NO COMPLIANCE** in this circuit so in order to remove fluid for sampling you will have to replace at the same time.
9. For sampling, fill a 10ml syringe with saline and attach to a pigtail and leave tap open whilst you draw off from circuit. This should avoid cavitation.



Once circuit is primed complete first part of perfusion checklist and transfer circuit to patient bedside. The inlet pressure monitoring should be setup and zeroed at bedside prior to cannulation (see protocol). Following cannulation complete the perfusion handover checklist.

Set Alarms

- Minimum Flow Alert: Pedivas set 50ml below established flow
  Centrimag set 100ml below established flow
- Maximum Flow Alert: Pedivas set 250ml above established flow
  CentriMag set 200ml above established flow

NB. High and low flow alarm limits cannot be set within 0.3l/min of each other

- Flow Limit Sensitivity: Set to Normal
- Pressure Display: Active
- Speed Step Resolution: Step 50 for Pedivas / Step 100 for Centrimag
- Language: English

Trouble shooting Levitronix centrifugal pump

Never clamp venous (blue) lumen without clamping arterial (red) lumen first as this may cause cavitation of gas in blood within device.

- **Retrograde flow**

Retrograde flow can occur at low RPM or during significant increases in BP and will cause acute clinical deterioration. Retrograde flow will be displayed on Levitronix console as - - - - . If this is displayed immediately clamp arterial tubing (post pump head).

- Action: Immediately clamp arterial tubing (post-oxygenator)

  Observe that flow probe is correctly placed on tubing

  Increase RPM by approximately 10%.

  Slowly release clamp observing for signs of forward flow.

  Once support has been achieved RPM may be weaned to give desired blood flow.

- **Pump failure**
- See "emergency procedures"
- If pump has been stopped for more than 5 mins there is a significant risk of thromboembolism.
- Follow cannula flushing guideline
- **Clots in circuit**
- See "Anti-coagulation management" section
- **Reduction in flow**

Can be caused by fall in patient's intravascular volume or by obstruction to flow into **venous (blue) cannula**.

- Action:Assess patients filling pressures (CVP) and inlet pressure

  Check cannula and Venous tubing

  Volume 5ml/kg PPS

  Reduce rpm by 10% to allow filling of RA before slowly increasing rpm watching

  flows on pump console.

  See also laminated "emergency ready reckoner" on pump trolley

- **Clamping circuit**
- Always clamp red lumen (arterial limb) first
- Only clamp red (arterial) then blue (venous) if changing pump head or flushing cannulae
- **Defibrillation**
- This can be safely done on ECMO
- The patient's haemodynamic stability should be carefully monitored during this time.
- Ensure rpm and flow of ECMO returns to baseline
- Switch on back-up pump in case of unanticipated pump failure

- **Pump failure**

1. Clamp red lumen (arterial limb).
2. Turn on power back-up console (switch at R side)
3. Turn off main console pump by pressing stop button for 2 secs this allows flow measure and pressure settings to be continuously viewed as the pump head is changed)
4. Once impeller is stationary unlock retaining screw & rotate head clockwise, lift out from motor.
5. Transfer head to back up motor. Insert and twist anti-clockwise then tighten retaining screw (ensure groove matches screw position).
6. Acknowledge "pump not inserted" alarm as cannot increase revs till alarm acknowledged.
7. Increase revs to 1500rpm.
8. Unclamp red lumen
9. Slowly increase revs to previous level
10. Attach flow meter to back up console
11. Remove pressure cables from main console and plug into back-up and re-zero
12. Call perfusion for replacement console

Safety checks

Undertake with each shift change

Primary Console

- Plugged into mains power
- On AC power indicator illuminated
- Battery level meter indicating battery fully charged
- Flow limit sensitivity set to normal
- Upper and Lower flow limit alarms set:
- Pt <10Kg – set 50ml/min below current flow
- Pt >10Kg set 100ml/min below current flow

NB. High and low flow alarm limits cannot be set within 0.3L/min of each other.

High flow limits should therefore be set 0.3L/min above low flow set limits.

Backup Console

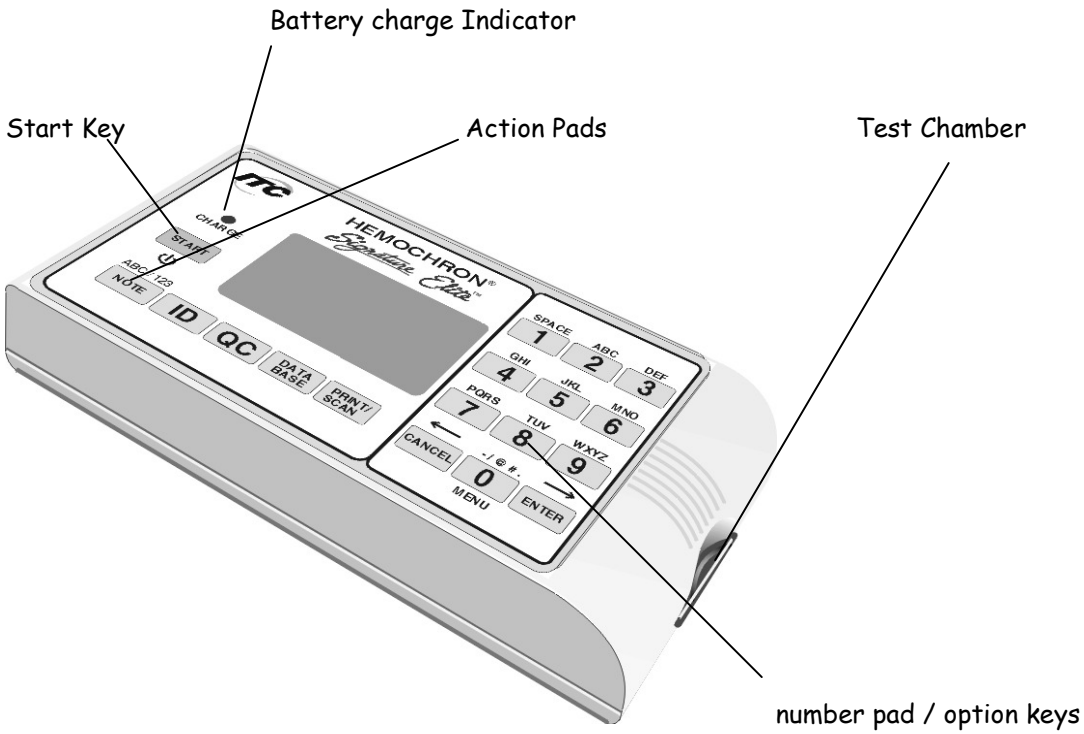
- Backup console and motor present.
- Console switched on and self-test successfully completed at start of ECMO run and daily thereafter.
- Backup motor positioned so as to enable easy transfer of pump head in an emergency.
- Backup console plugged into mains power at all times (mains power indicator not illuminated when console switched off)

The Elite Haemochron ACT Analyser

Anticoagulation is necessary during various medical and surgical procedures to counter-balance the natural thrombotic response of blood upon its exposure to a foreign surface such as the ECMO or VAD circuit. It is most commonly used for heparin anticoagulation monitoring during bypass surgery, Extracorporeal Membrane Oxygenation (ECMO), haemofiltration, haemodialysis and critical care (CVVH-Continuous Venous to Venous Haemodialysis). Without optimal anticoagulant therapy, clot formation would occur within minutes. Close monitoring and control of anticoagulation is desirable to ensure clot free blood flow while minimising bleeding complications during the ECLS run.

The Hemochron Signature Elite utilises a mechanical endpoint clotting mechanism in which testing occurs within the disposable ACT cuvette. Following whole blood sample introduction, the instrument precisely measures 15µl of blood and automatically moves it into the test channel within the ACT cuvette. The remainder of the blood sample, not needed for testing, is automatically drawn into the waste channel of the cuvette. Sample/reagent mixing and test initiation are performed automatically, requiring no operator interaction. After mixing with the reagent, the sample is then moved back and forth within the test channel and monitored by the analyser for clot formation.

The clot detection mechanism consists of two LED optical detectors aligned with the test channel of the cuvette. The speed at which the blood sample moves between the two detectors is measured. As clot formation begins, blood flow is impeded and the movement slows. The microcoagulation instrument recognises that a clot endpoint has been achieved when the movement decreases below a predetermined rate. Electronic optical detection of a fibrin clot in the blood sample automatically terminates the test causing the instrument's timer to display the coagulation time in seconds. The whole blood ACT test result is displayed by the instrument's digital timer as the celite equivalent ACT value in seconds in order to provide a familiar clinical format and thus facilitate accurate clinical test result interpretation.



Supplies required for testing

1. Hemochron Signature Elite Microcoagulation Analyser

2. Transformer (9 volt) and mains cable

3. **ACT-LR cuvettes** – a self contained disposable test chamber preloaded with a dried preparation of **celite**, potato dextrin, stabilizers and buffers. Each cuvette is individually packaged in a foil pouch with a dessicant. Each box contains 45 individually pouched cuvettes. Cuvette pouches are stamped with a lot-specific expiration date.

4. When refrigerated (2-8°C), the foil-pouched ACT+ cuvette is stable until the marked expiration date usually 12 months. **Room temperature cuvettes are good for a maximum of 3 months but must never exceed the marked expiration date.** A box of cuvettes should be removed from the fridge when setting up for a cannulation and then 1 box as required during run. **Re-dating is necessary if stored at room temperature**. A re-dating label is included on the side panel of each box of cuvettes and should be completed. DO NOT expose to temperatures in excess of 37°C.

6. Cuvettes must be at room temperature prior to use. For cuvettes stored at refrigerated temperature (Allow one hour.) **This can affect results!**

Test Requirements: prior to opening cuvette and inserting into device ensure that the following are available at the bedside.

1. Patient label with bar code

2. Operator bar code

3. Appropriate cuvette

4. Sterile Drape

5. Plastic syringe 1ml

6. Plastic Syringe 3ml x 2

7. Saline for pigtail flush

7. Cliniwipe

8. Sterile gloves

Collection Procedure:

The ACT test is optimally performed using 0.05ml of **fresh whole blood**. The cuvette requires a minimum volume of 15µl to perform the analysis, and will display a "Sample too small" if insufficient sample is applied. Samples with any of the following characteristics should be **discarded** immediately, and a **fresh whole blood** sample collected prior to performing any test:

- Sample contamination with indwelling intravenous (i.v.) solutions.
- Sample contamination with alcohol cleansing solution.
- Samples with visible clotting or debris accumulation.

NOTE: The amount of blood required to adequately flush the line until it is free of contaminants is dependent on the amount of solution contained within the line. The volume required to fully clear an ECMO circuit pigtail is 1ml.

Blood samples demonstrating any of the above may interfere with the ACT assay.

Test Instructions

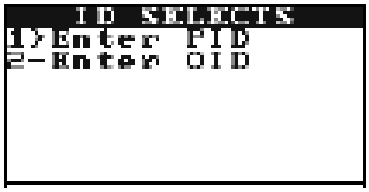
1. Prior to collection of a sample put on apron, wash hands and prepare sterile field and supplies, switch on device (press start) and select correct cuvette, ACT-LR.

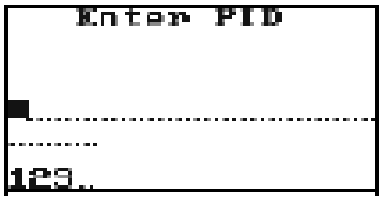
2. **Insert** Cuvette into the right side of instrument with the blood reservoir facing up. Cuvettes must be at room temperature prior to use. **DO NOT force a cuvette into the instrument. DO NOT use expired cuvettes.** Once device has warmed cuvette to 37oc, the instrument display will indicate "Add Sample" and "Press Start" and 5min count down will commence.

and

The instrument will remain in the ready mode for five minutes. At the end of five minutes, a "Start timeout" will occur indicating that the current cuvette be discarded and a new cuvette placed in the instrument

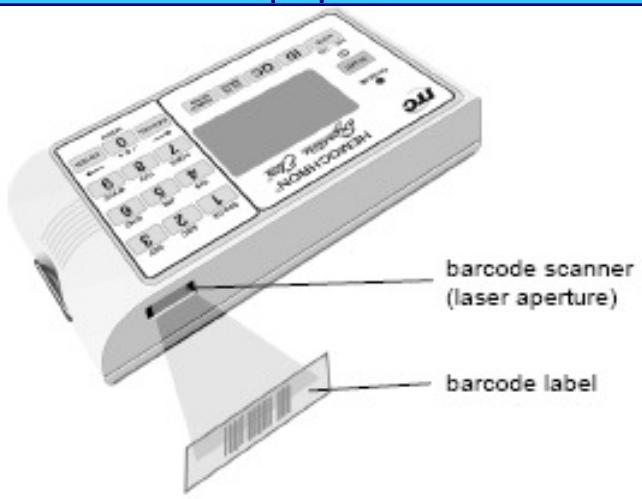
Enter patient ID, press ID on device and select/enter PID (patient identification) by selecting number 1 on device. Press and hold print/scan button and position approx 4" from bar code of patient label until device bleeps. The patient ID may also be manually entered, press NOTE button to change between numbers/letters and press and hold enter button to store. To return to main screen press cancel on keypad.



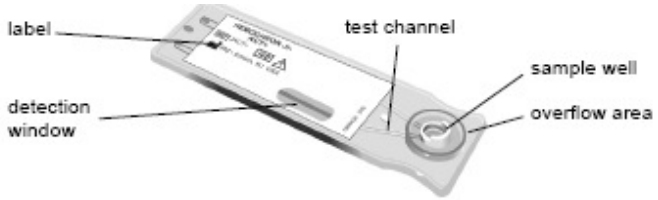


4. Enter operator ID, press ID on device and select/enter OID (operator identification) select number 2 on device. Press and hold print/scan button and position approx 4" from bar code of personnel ID label until device bleeps. The Operator ID may also be manually entered, press NOTE button to change between numbers/letters and press and hold enter button to store. To return to main screen press cancel on keypad.

5. Scan cuvette barcode, press Print/Scan button then select next page of menu by selecting number 7 on device, then select number 3 "Test scan" Device will prompt operator to press scan. Hold cuvette packaging in front of scan port and hold Print/scan button until device bleeps and the lot number of the cuvette appears in window. To return to main screen for test, press cancel 3 times. These 3 steps will take approximately 30 secs.



6. Obtain blood sample from patient as per guideline and **immediately** dispense one drop of fresh whole blood into the sample well of the test cuvette, filling from the bottom of the well up. A sufficient quantity of blood must be added directly to the center of the sample well to fill it flush to the top. Should a large drop of blood extend above the top of the center sample well, push it over into the outer sample well. **Press** the **Start Button** on the instrument to begin the test. The instrument will make an audible beep.



7. **When complete**, the instrument will again beep and display the result. Electronic optical detection of a fibrin clot in the blood sample automatically terminates the test causing the instrument's timer to display the coagulation time in seconds. **DO NOT remove cuvette!** The test result will only remain displayed until the cuvette is removed. **Record test result** on CIS/ECMO chart. The device will store upto 600 test results.

Caution: All used cuvettes should be considered as potentially infectious, handled with care and disposed of by following standard waste disposal policy.

Results

Results that **exceed "400" seconds** must be **reported** out as **greater than 400 seconds** when using **ACT-LR cuvettes**.

Critical Values:

NOTE: Test results must correlate with patient condition, and/or treatment, abnormal results must be repeated. All results are entered in CIS/ECMO chart as part of the patient record. Critical values require adjustment of Heparin and repeat testing every 20/30mins until within parameter. Results which remain abnormal must be reported to the ECMO physician and documented in the patients nursing records.

1. Temperature errors **must** be reported to Gillian Wylie / Bioengineering at ext. 89329 or 80132. If it is after hours, weekend or holiday, a replacement device will be available in the 2nd floor ECMO Office

2. **DO NOT** use cuvettes past their expiration date or cuvettes that have been improperly stored. If kept at room temperate box must be dated with new 3 month expiry date.

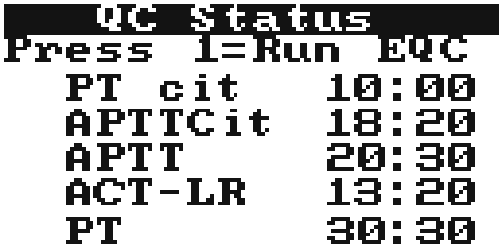
3. **DO NOT** force a cuvette into the instrument. If resistance to insertion is encountered, gently remove the cuvette and examine the cuvette slot. Remove any obstruction before attempting further use of the instrument. If unable to remove easily, contact Gillian Wylie / Bioengineering at ext. 89329 or 80132.

4. **DO NOT** use excessive force in depressing the START key.

5. **DO NOT** drop the instrument. If dropped, perform electronic quality control (EQC). If EQC is acceptable, instrument can be used. If unacceptable, contact Gillian Wylie / Bioengineering at ext. 89329 or 80132.

Quality Control

The HEMOCHRON *Signature Elite* instrument performs a "self-check" every time it is activated and a test is performed. When a test is initiated by inserting a cuvette, system checks are automatically performed. An electronic quality control (EQC) test should be performed once a shift. There is an internal EQC within the unit. Prior to initiating an EQC test ensure any used cuvettes are removed then press QC key and select 1 The test chamber warms to temperature and the EQC test begins. The results are displayed while the test is progressing. When the test is completed, the results are displayed on the screen and written to the QC database. Press **CANCEL** to exit the screen. The Internal EQC will check two levels of QC and the temperature, and it will store each result.
If one result fails, the test will stop and record all results as failed. If the user aborts the internal EQC test, the test is not saved to the database or printed.



A Liquid Quality Control will be run on each batch of cuvettes that are sent from the company by perfusion or the ECLS coordinator, ensure that all boxes removed from the fridge have been initialled as passed. If in doubt contact ECLS Coordinator or Perfusion

ECMO physiology

A thorough understanding of oxygen and carbon dioxide exchange during VA and VV perfusion is a prerequisite for successful management of the patient on ECLS. To understand the effect of ECLS on oxygenation, one must review the following concepts of oxygen content, arterio-venous differences, oxygen delivery and oxygen consumption. Oxygenation is dependent on oxygen content and delivery as defined below

Oxygen Content

Oxygen content (ml O_2/100ml blood or volume %) is defined as the total oxygen in whole blood that is available to tissues. The oxygen content is rarely measured directly for clinical applications. Blood oxygenation is more commonly expressed in terms of PaO_2 or haemoglobin saturation. However oxygen content is an important consideration in the physiologic management of critically ill patients. Oxygen exists in two forms in the blood – dissolved or bound to haemoglobin. The formula below is used to calculate oxygen content:

Oxygen Content: (measured as mlO_2/100ml blood, or vol%) = O_2 bound to Hb + dissolved O_2

= [Hb (g/dl) x Sat% x 1.34] + [PaO_2 (mmHg)x 0.0031] and is measured as ml O_2/dl

Example:
Hb = 15 g/dl
SaO_2 =100%
PaO_2 =200 mmHg
O_2 content = (15 x 1.00 x 1.34) + (200 x .0031) = 20.1 + 0.6 = 20.7 ml O_2/dl blood (vol%)

Haemoglobin concentration is the major variable determining oxygen content when the SaO_2 is 100%

In a practical sense on ECLS the major determinant of oxygen content is haemoglobin concentration since blood loss and blood transfusions can directly influence oxygen carrying capacity. The oxygen saturation of blood leaving the oxygenator is >90%. However it should be appreciated that increasing PaO_2 beyond 100mmHg contributes little additional benefit. Even if the PaO_2 is >400mmHg only 1.2 mlO_2/100ml blood is added to the O_2 content of the blood.

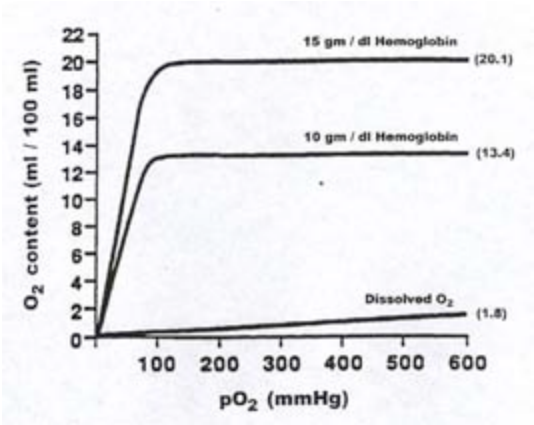


Figure 1 Oxygen content of blood

The Oxygen Dissociation Curve

The relationship between PaO_2, saturation, and O_2 content is shown by the oxygen dissociation curve. (Figure 2) Large increases in oxygen saturations occur with increased PaO_2 until levels of 50 mmHg.

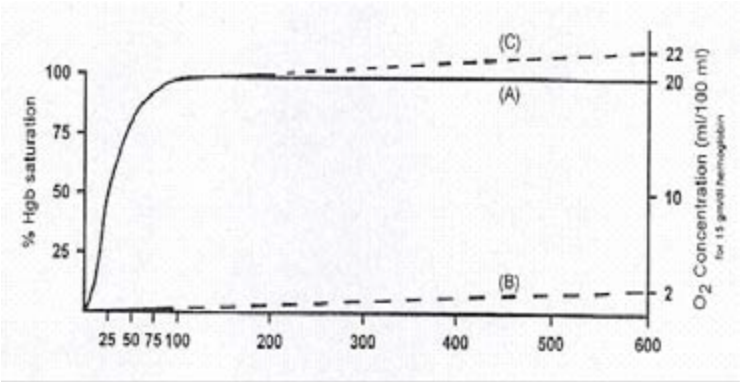


Figure 2 Oxygen dissociation curve

Oxygen Delivery (DO_2)

In the normal infant (or adult) venous blood is pumped by the heart through the lungs, where oxygen is absorbed by haemoglobin. It is then pumped through the systemic circulation where oxygen is released to the tissues. "Oxygen sufficiency" will be present when oxygen delivery equals or exceeds tissue oxygen consumption. Oxygen delivery is the amount of oxygen delivered to the peripheral tissues each minute and is determined by two primary variables: oxygen content of the blood and the blood flow (cardiac output).

DO_2 delivery = O_2 content x blood flow (cardiac output)

$=[(Hb \times Sat \times 1.34)+(PO_2 \times 0.0031)] \times C.O.$

Cardio-respiratory haemostasis tends to maintain systemic oxygen delivery at the normal level. In anaemia and hypoxia the cardiac output will increase until oxygen delivery is normalised.

NOTE: Flow is equal to cardiac output (C.O.)in the patient but can be easily controlled during VA ECLS by increasing the amount of blood flowing in the ECLS pump and therefore decreasing that done by the heart. As noted above, DO_2 can be effected most by [Hb] and ECLS flow, since saturations are usually 98% or greater. It is therefore imperative to keep the Hct at 40% or greater.

Oxygen Consumption (VO_2)

The other side of the oxygen balance equation is tissue oxygen consumption. It is defined as the volume of oxygen consumed by the body in one minute. Under steady state conditions, the amount of oxygen absorbed across the lung in the process of pulmonary gas exchange is exactly equal to the amount of oxygen consumed by peripheral tissues during metabolism (the Fick principle) regardless of the status of pulmonary function.

By subtracting the venous oxygen delivery to the right heart from the arterial oxygen delivery to

the body oxygen consumption can be calculated.

VO_2 = Arterial oxygen delivery – Venous oxygen delivery

Fick Equation:

Oxygen consumption (mlO_2/min) = Cardiac Output (L/min) x [13.4 x Hb (g/dl)] x [SaO_2 (%) – SvO_2 (%)]

If the arterial blood is fully saturated, the venous saturation decreases proportionate to the amount of oxygen extracted from the arterial blood. Thus if the oxygen extraction ratio is 20% the venous saturation will be 80%; if the oxygen extraction ratio is 33% the venous saturation will be 67% etc.

Oxygen consumption is controlled by tissue metabolism. It is **decreased** by rest, paralysis and hypothermia and **increased** by activity, infection, hyperthermia and increased levels of catecholamines and thyroid hormones. Oxygen consumption in the sick newborn can be doubled by sepsis, preceding hypoxia and stimuli associated with stress.

The relationship between oxygen delivery (DO_2) and oxygen consumption (VO_2) is reflected in the oxygen content of the venous blood. Venous oxygen content represents residual oxygen in the blood after oxygen has been released to the tissues.

Typical values for venous and arterial blood at different levels of haemoglobin are identified in Figure 3.

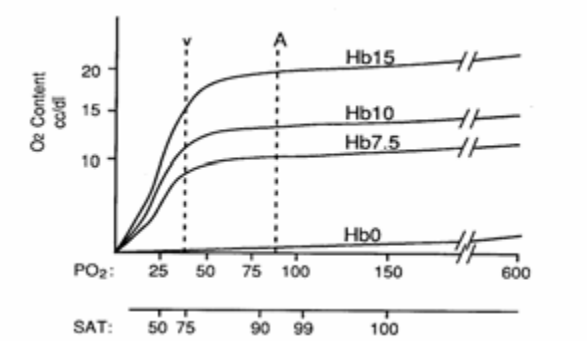


Figure 3 Measuring O_2 in blood

Notice there is more oxygen in normal blood (Hb 15g/dl) with a PO_2 of 40 mmHg (≈5.2kPa) than there is in anaemic blood (Hb 10g/dl) with a PO_2 of 100 mmHg (≈13kPa)

Relationship between oxygen delivery and consumption

Normally oxygen delivery is five times consumption and the venous saturation is 80%. If delivery is less than twice consumption the venous saturation is less than 50% and consumption becomes dependant on delivery. The relation ship between oxygen consumption (VO_2) and oxygen delivery (DO_2) are shown in Figure 4.

In a practical sense on VA ECLS the patient is transfused to a desired haemoglobin level and the ECLS pump flow increased to achieve adequate oxygen delivery as reflected by the venous saturations regardless of oxygen consumption. An SvO_2 of >70% generally reflects adequate ECLS support. The concepts of oxygen delivery and content are depicted in the next few examples of the

normal newborn (Figure 5), the infant in respiratory failure secondary to PPHN (Figure 6) and the effect of VA (Figure 7) and VV ECLS (Figure 8).

Suppose we have a 3kg infant who has an Hb of 14.5g/100ml, SaO_2 =100%, PaO_2=100mmHg as shown in figure 5. The **oxygen content** is computed as:

CaO_2 = (Hb x PaO_2 x1.34) + (PaO_2 x 0.0031)
= (14.5 x 1.0 x 1.34) + (100 x 0.0031)
= 19.7 ml O_2/100ml blood (vol%)

If cardiac output is known to be 120ml/kg/min or 360ml/min. the **oxygen delivery** is:

DO_2 = CaO_2 X flow (*C.O.*)
= 19.7 ml O_2/100ml x 360ml min
= 71 ml/min 0_2 or 23.7 ml/kg/min

Similarly, if the venous PO_2 and saturation are known as (PvO_2 = 40 mmHg, SvO_2 = 75%), the venous oxygen content can be calculated. Using the same formula, the venous oxygen content is 14.7 vol%.
The **arterial venous oxygen difference** ($AVDO_2$) = CaO_2 – CvO_2.
Using this formula the $AVDO_2$ = 19.7 vol% - !4.7 vol% = 5 vol% which is a normal value we can calculate oxygen consumption (VO_2) by the equation:

VO_2 = $AVDO_2$ x flow
= 5ml O_2/100ml x 360 ml min
= 18ml O_2/min (or 6ml/kg/min)

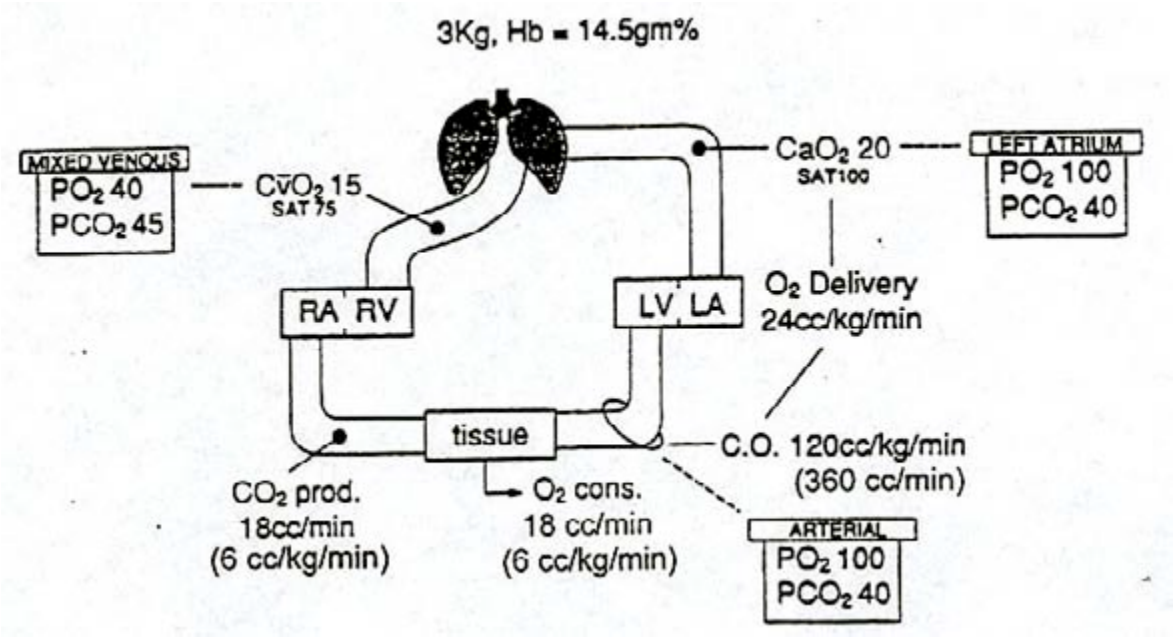


Figure 5 Normal physiology

Assuming the same patient now developed newborn respiratory failure with 50% right-to-left shunt (50% of the venous blood does not pass through the lungs (figure 6), the oxygen content of the

"mixed" blood (from aorta and shunt) can be calculated.

Thus a half volume of blood with an oxygen content of 14.7 vol% added to a half volume of blood with an oxygen content of 19.7 vol% results in a volume of blood with a content of 17.2 vol%. If we assume that our patient's oxygen requirements are the same, then the only way to satisfy oxygen balance is for his blood to circulate faster. Cardiac output (C.O.) is increased through endogenous reflexes to maintain normal oxygen delivery. Since oxygen content is decreased by 13% (from 19.7 to 17.2), C.O. must be increased by 13% to maintain normal oxygen delivery.

This ductal shunt has imposed the need to increase cardiac output from 120 ml/kg/min to 135 ml/kg/min to support the same oxygen demand. If cardiac output did not increase and consumption remained constant, venous oxygen content would decrease, ultimately resulting in an even lower arterial oxygen content and PaO_2. With less oxygen delivered, relatively more oxygen is consumed from each decilitre of blood.

Eventually, oxygen supply would be inadequate to meet requirements and shock with anaerobic metabolism would ensue. Infants increase their oxygen delivery by increasing heart rate and cardiac output; they extract more oxygen from haemoglobin (to the limits imposed by the Hb/O_2 dissociation curve) and they can switch to anaerobic metabolism, producing increasing amounts of lactic acid. The end result would be a very ill baby with tachycardia, acidosis and low mixed venous PO_2.

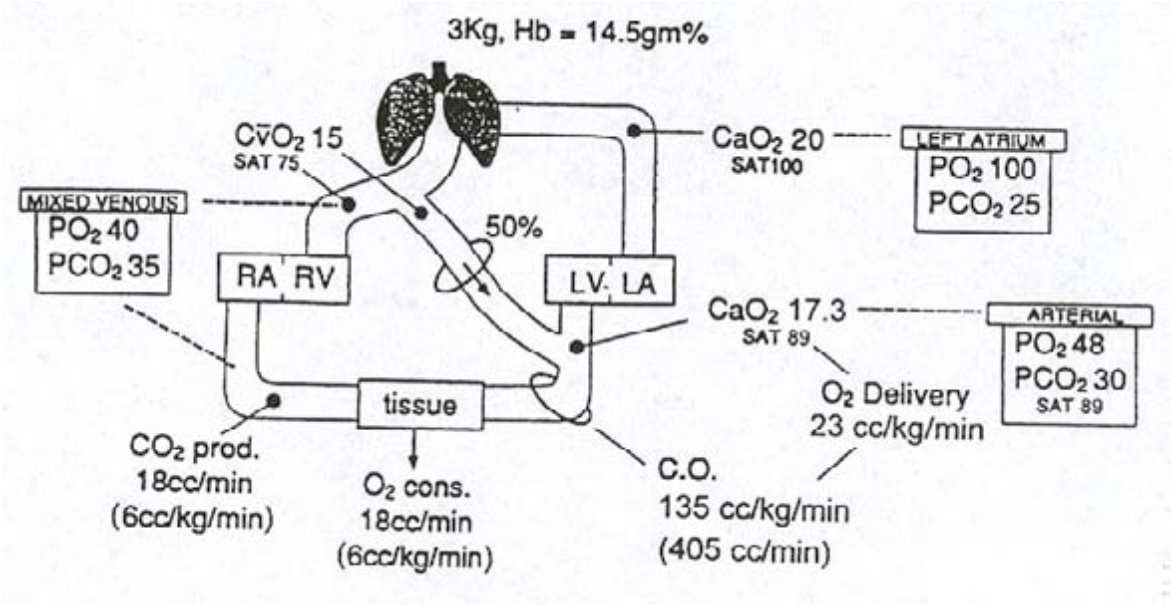


Figure 6 Normal lungs but PPHN and PDA present

Take the example one step further. His condition has deteriorated and we place him on VA ECLS support (figure 7).

He is cannulated and ECLS flow is 360 ml/min (120 ml/kg/min). Assume that an additional 30 ml/kg/min remains flowing through his right atrium and right ventricle into his lungs. Also assume for simplicity that his lungs make no contribution to oxygenation. Blood coming from the left

ventricle has the same content as venous blood. Assume the venous content is 15 vol% and the post oxygenator blood has an oxygen content of 21.0 vol%. The oxygen content of the combined cardiac and ECLS flow as it is mixed in the descending aorta is 20 vol%. At this flow rate, an oxygen consumption of 18 ml/kg/min would be nicely supported, leaving a venous oxygen content of 15 vol%. Any contribution by the lungs would lower the ECLS delivery requirements and allow weaning. As the patient recovers, lung function improves and the lungs transfer more oxygen. The resultant mixture of extracorporeal and pulmonary blood results in a higher oxygen content so the ECLS flow can be reduced. More blood is returned to the lungs and the flow is gradually weaned. The flow is regulated to keep the oxygen content at the prescribed levels. Once the patient has recovered, ECLS is stopped allowing 100% of the venous return to flow through the lungs.

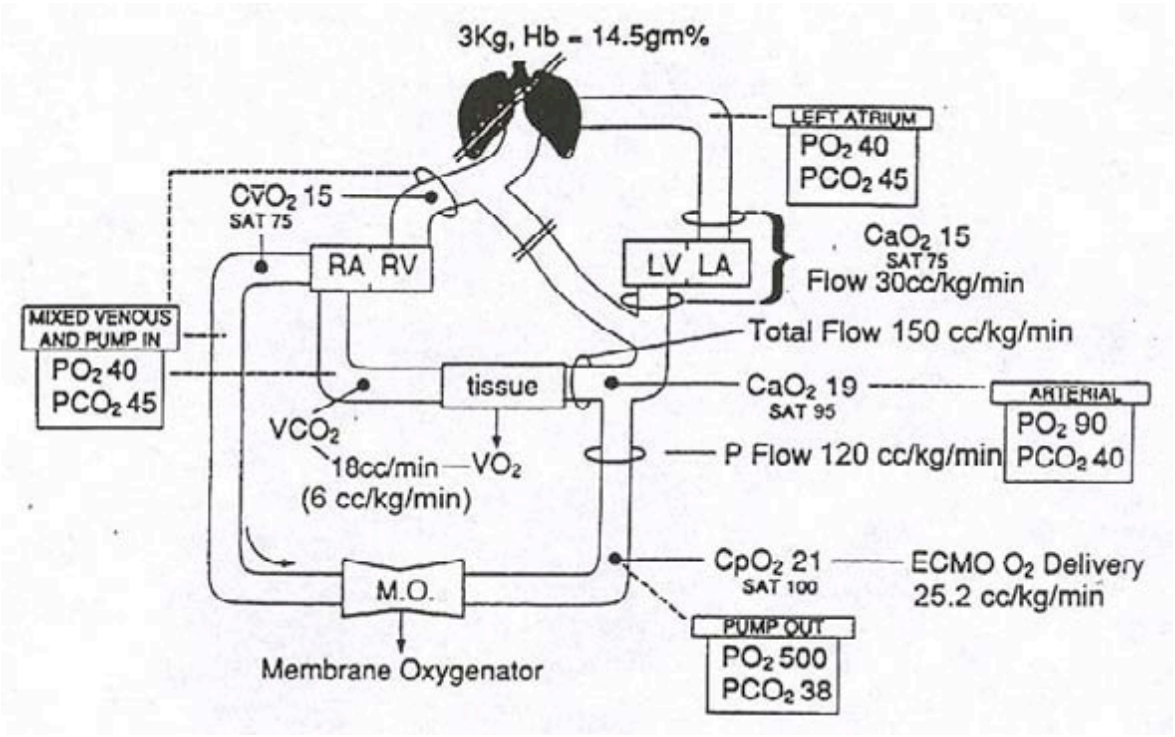


Figure 7 PPHN on ECMO

If the infant is treated with VV perfusion rather than VA, blood is drained from and returned to the right atrium, so blood flow through the right ventricle, pulmonary artery, ductus and pulmonary circulation is normal. Since carbon dioxide is readily diffusible, relatively low flow VV perfusion is sufficient to remove all metabolically produced CO_2. However, during VV perfusion, oxygenation of

the blood is limited by the amount of oxygen each decilitre can absorb. In VV perfusion, the oxygenator blood is returned to the right atrium where it mixes with blood returning from the tissues, raising the saturation of blood in the right ventricle, pulmonary artery and ultimately the arterial circulation. Compared to VA ECLS, the saturation of the venous drainage blood is higher, the amount each decilitre can absorb is less and more decilitres per minute must be passed through the oxygenator to achieve the same oxygen delivery on VV bypass. The VV ECLS patient's haematocrit is maintained 5% higher than that used for VA to aid oxygen delivery.

A typical example of VV perfusion for a neonate is shown in figure 8. To avoid confusion, the blood gases in the venous drainage blood during VV perfusion are designated PqO_2, $PqCO_2$, SqO_2 and CqO_2. In addition blood gases in the pump outlet blood are designated PpO_2, $PpCO_2$, SpO_2 and CpO_2. Notice that the haemoglobin is now increased to 15.2 mg/dl (5% more than previous examples), the arterial PO_2 is 52 mmHg (7kPa) and the content is 19 ml/dl. This patient would be quite similar to the untreated hypoxic physiology shown in figure 6 if the haematocrit had not been increased. The cardiac output has reflexively increased to 135 ml/kg/min in order to maintain normal systemic oxygen delivery at 26 ml/kg/min.

The blood leaving the tissues has an oxygen content of 15.3 ml/dl. The blood mixes in the right atrium with the perfusion blood from the oxygenator which has a content of 21.5 ml/dl and a PO_2 of 500 mmHg. The mixed blood in the right atrium has a content of 19 ml/dl (vol%) and a PO_2 of 52 mmHg (7kPa). This blood passes through the right ventricle out into the pulmonary artery and into the systemic circulation. Assuming there is no native lung function, the pulmonary artery blood and the aortic blood have the same blood gas composition. While some of the blood passes from the right atrium into the pulmonary artery (135 ml/kg/min in this example), the remainder is diverted through the venous drainage catheter into the circuit. This drainage blood has a PqO_2 of 45 mmHg (6kPa) and a CqO_2 of 16.4 vol%. This difference from the patient's true venous blood oxygen content is caused by recirculation of oxygenated blood from the pump back into the drainage catheter (generally about 20 % of total pump flow)Another important difference between VV and VV perfusion is that the arterial PO_2 will be no higher than pulmonary artery PO_2, when there is no native lung function.

Consequently it is necessary to manage VV patients at arterial saturation levels between 85-95% with PO_2 levels of 6-7.5 kPa and haematocrit levels slightly higher than those on VA. As long as the cardiac output is able to compensate for this hypoxaemia, systemic oxygen delivery is more than adequate to meet normal systemic requirements.

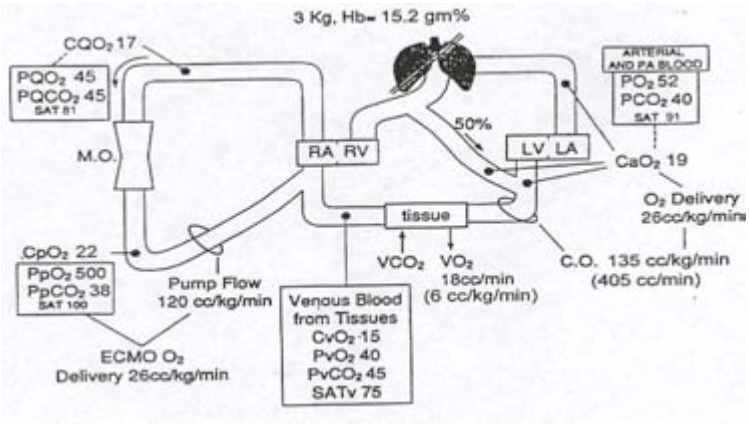


Figure 8 Lung disease with VV DL

Physiology of the ECLS circuit

Resistance of the circuit

Resistance to flow is dependant on the dimensions and geometry of the tubing used and the characteristics of the fluid used. Poiseuille's Law defines it by the following equation:

$$R = \frac{8\eta l}{r^4}$$

where 8h is the constant of proportionality; l (cm) is the length of the conduit; h (Poises, Poise = dyne-sec/cm2) is the coefficient of viscosity; and r (cm) is the internal radius of the tubing.

Therefore, the longer the tubing, the greater the resistance, or the larger the radius, the less resistance. The arterial (or venous return) catheter (D) is the high resistance component of the circuit, this is where the diameter of the circuit will significantly change.

Resistance can also be described by the following formula:

$$R = \frac{P_1 - P_2}{Q}$$

where P1 - P2 is the difference between inlet and outlet pressures, and Q is the flow. Usually expressed as mmHg/LPM.

$$\frac{\Delta P}{Q} = \text{Resistance} = \frac{8\eta l}{\pi r^4}$$

ECLS Catheters

All cannulae used with the centrifugal pump will be wire reinforced to prevent collapse of the cannulae under high negative inlet pressures excerted by the pump. The length and internal diameter of the tubing and cannula will affect the overall resistance of the ECMO circuit. Ie A "short large lumen" catheter such as the Biomedicus 10 Fr (in the neonate) is ideal for lower resistances and thus lower pressures in the circuit.

Avalon double lumen catheters allow blood to be drained from the SVC and IVC with dedicated ports in these areas if the cannula is appropriately sited. The return aperture is directed towards the tricuspid valve to minimise recirculation.

The resistance of the arterial (or venous return) catheter and the blood flow will determine the post membrane pressure in the circuit since the tubing has very low resistance in the neonatal flow ranges.

Maximal flows (ml/min) for each catheter have been determined These have been designated 'M' numbers. The lower the number, the lower the resistance.

NOTE: If unusually high pressures post-membrane are noted, rule out the following:

- Kinked catheter
- Clotted catheter
- Catheter tied too tight (unusual with Biomedicus catheters)
- Flow too high for the size of the catheter
- Catheter against the wall of the aorta or dissection of the aortic arch

NOTE: If haemolysis occurs, think of obstruction in the circuit.

Blood flow

Blood flow varies directly as the fourth power of the radius of the tubing and is inversely proportional to the length of the tubing.

$F \propto r^4$ and $F \propto \frac{1}{L}$

Therefore the venous catheter will determine the amount of blood flow going through the circuit. To maximise flow, a short-as-possible large lumen venous catheter should be used ("short and thick does the trick"). Since flow determines oxygenation (see next Section), a large venous catheter is preferred.

Laminar and Turbulent Flow:

To decrease haemolysis and/or fibrin degradation and formation of clots, laminar or streamlined flow is preferred. Therefore changes in tubing diameter or acute angles in the circuit are to be avoided. Stagnant areas of flow created by abrupt angles or changes in diameter, called "Eddy Flow Patterns", are depicted below in Figure 10. It is in these areas that cell aggregates and fibrin start to form resulting in sources of clot formation.

In summary circuit and catheter design must take all of these factors into consideration, especially for the "low flow, low heparinisation" state of ECLS which creates more risk for clot formation than the high flow, high heparinisation state of cardiopulmonary bypass. The effect of circuit design on haemolysis, clot formation, and pressure changes must be fully understood before changing or adding new components to the circuit.

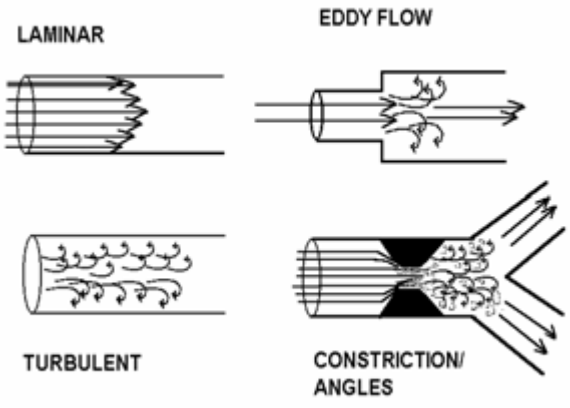


Figure 10 Types of flow current

Physiology of ECMO

ECLS blood flow must be maintained at a sufficient rate to adequately perfuse the baby and allow "rest" of the lungs. In most infants this can be achieved at flows of 100-120 ml/kg/min for VA ECLS and 120-150 ml/kg/min for VV ECLS. The cardiac output (C.O.) in infants is difficult to measure because of the right to left shunts, but has been estimated to be from 120-300 ml/kg/min. Doppler studies of left ventricular output have shown that the sick neonate may have an output of 200-300 ml/kg/min. Because of the difficulty of measuring C.O. in the neonate, an estimated of 200 ml/kg/min is used to calculate the percentage flow. (i.e. a 3 kg infant on 360 ml/min flow is on 60% bypass using 200 ml/kg/min as the estimated C.O.).

The centrifugal pump like the roller pump is classed as non-pulsatile. Although it is unusual to require 100% bypass flows (which would flatten the pulse contour), pulsatility is altered (see Figure 11). This altered pulsatility does have an effect on organs such as the kidney. These alterations appear to change the production of renin resulting in alterations in electrolyte requirements. Renin levels have been shown to be high in infants on ECLS.

The effect of ECLS on the total renin-aldosterone axis has been studied. Infants on ECLS require large amounts of K^+ (4 mmol/kg/day) and less Na^+ (1-2mmol/kg/day) which is thought to be secondary to the effect of ECLS on the kidney (see Electrolyte Management on ECLS).

Acute cardiac dysfunction

Cardiac stun is observed not infrequently on commencing VA ECLS. The pulse pressure narrows to <15 mmHg (mild) or <10 mmHg (severe), though the MAP remains stable. It tends to occur in the sicker patients. A number of theories exist as to its cause (high outflow pressure; poor coronary artery oxygenation / perfusion; cardiac "rest") but the important issue is that it will resolve spontaneously within 24-36 hours in almost all cases. During this period, flows are held at 50-60% CO and the blender is weaned as appropriate. Cardiac stun does not occur with VV ECLS.

Cardiac arrhythmias can occur if the pump prime electrolyte concentrations are not within the physiological range. This is especially important in the case of VV ECLS where the circuit does not support cardiac function.

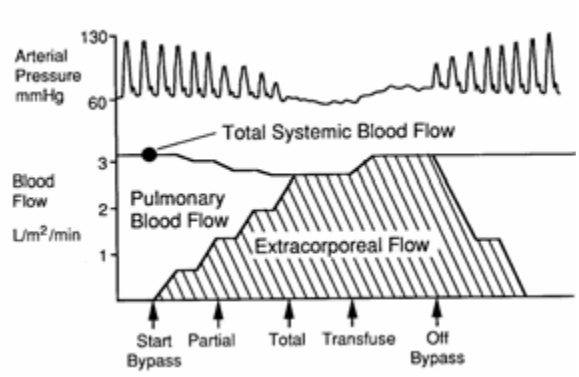


Figure 11: Physiological measurements during VA cardiopulmonary bypass

The artificial lung: gas flow requirements

The gas flow to the oxygenator is regulated by the flow-meters and an oxygen blender to give differing fractional oxygen concentrations (FiO_2). The gas flow to the oxygenator must be limited to ensure that the pressure on the gas side of the oxygenator is lower than that on the blood side. If this is not maintained, air embolus and/or rupture of the fibres can occur. This reversal of pressures may occur by exceeding the rated flow of the oxygenator, or by obstructing the gas outlet port on the oxygenator. Cases of massive air embolus resulting in death have been reported, so understanding of this concept is imperative. The management of the gas flow to the oxygenator is discussed below. A review of oxygenation concepts discussed earlier in this book may be of help.

Oxygen regulation

Oxygen transfer is not affected by changes in gas flow because the O_2 gradient is so large (100 kPa in the sweep gas to circa 5 kPa in the venous blood). With the 800LT oxygenator, the O_2 flow is usually kept between 0.4 and 0.6 L/min, with a maximum rated flow of 2.0 L/min (rates above 2.0 L/min may cause supersaturation and air embolus and rupture the oxygenator fibres). The functioning oxygenator is always working at its maximum capacity for O_2 transfer, therefore the blood outlet PO_2 is regulated by the following:

1. Inlet PO_2 and O_2 content (i.e. the amount of oxygen each ml of blood can take up in the membrane lung)
2. The blood flow - the 800LT will transfer 55-70 ml/min and thus has a rated blood flow (max) rate of 800 ml/min; exceeding this rate will cause oxygenation to decrease.
3. The surface area of the oxygenator

When the oxygenator is functioning normally, to lower the blood outlet PO_2, you must decrease the FiO_2 going into the oxygenator by blending oxygen/air into the oxygenator gas. To raise the outlet PO_2 when you are on FiO_2 = 1.0, a larger oxygenator must be used. Remember, that the blood will be maximally saturated with PO_2 >13.5 kPa and thus raising the PO_2 above this level will not increase oxygen delivery to the patient. To increase patient PaO_2, the blood flow to the patient from the pump must be increased, i.e., more of the C.O. of the patient must be taken over by the pump to give oxygenated blood to the patient.

ECLS flow is limited by the amount of venous blood drainage available, and this in turn is dependent on the size of the venous catheter and its position. Improper catheter placement and size will limit the ability to oxygenate the patient. The major way to increase oxygenation in the ECLS patient is to keep Hct between 40-45 and then to increase ECLS flow to take more of the C.O. through the ECLS circuit.

Oxygen exchange is:

- Independent of sweep gas flow rate
- Dependent on O_2 concentrations (blood and gas)
- Dependent on blood flow rate
- Dependent on oxygenator diffusion characteristics
- Dependent on blood path thickness
- Constant for a given oxygenator
- Dependent on oxygenator surface area

Examples of O_2 regulation: (Hb = 15 g/dl)

If PO_2 = 200 mmHg:
O_2 content = (15 x 0.99 x 1.34) + (0.0031 x 200) = 20.72 vol. %

If PO_2 = 500 mmHg:
O_2 content = (15 x 0.99 x 1.34) + (0.0031 x 500) = 21.65 vol. %

Therefore, increasing PO_2 to 500 mmHg did not significantly change O_2 content. If you were flowing at the same rate, it did not change OD, i.e., if flows were at 200 ml/min, then OD = 41.2 and 43.2 respectively.

Carbon Dioxide regulation

Note:
No CO_2 is added to the centrifugal circuit therefore pCO_2 is adjusted via adjusting gas flow.

Starting off with gas flows corresponding to blood flow is a reasonable starting point e.g. 0.5L/min blood flow then set oxygen gas flow at 0.5L/min. Patients with high pCO2 will require a reduced sweep gas flow to ensure there is no rapid reduction in pCO2. This is normally attained by halving the normal predicted gas flow rate.

CO_2 exchange through the oxygenator is much more efficient than O_2 exchange. The rate of CO_2 transfer is 6 times greater than that of O_2. Because the CO_2 gradient is usually not large (oxygenator 0 kPa and blood 4.6-5.3 kPa), CO_2 transfer is reliant on sweep gas flow. The blood and gas PCO_2 levels equilibrate very fast, so to remove more CO_2 you must move the sweep gas through at a faster rate. You can think of CO_2 removal as akin to that in the lung, i.e., increasing ventilation (increasing sweep gas flow rate for ECLS) will decrease PCO_2, and decreasing ventilation (decreasing sweep gas flow rate for ECLS) will increase PCO_2.

It must be remembered that with VA ECLS, the patient's brain will preferentially see the pump PCO_2 and therefore levels less than 5.3 kPa may result in a decreased respiratory rate in the patient. As ECLS flows are decreased and once the patient has taken over greater than 50% of the CO, low pump PCO_2 will cause the patient to stop breathing, and the patient's $PaCO_2$ will rise. Treatment is to drop the gas flow thereby increasing the pump PCO_2 to between 6-6.7 kPa. This will stimulate the patient to breathe and blow off the excess CO_2.

Factors affecting CO_2 elimination

- Relative concentrations of CO2 on either side of the oxygenator.
- Surface area of the oxygenator available for transport.
- Sweep gas flow rate across the oxygenator.

Thus, CO_2 exchange is:

- Independent of blood flow.
- Dependent of gas diffusion gradient.
- Dependent on sweep gas flow rate.
- Dependent on oxygenator surface area.

Example of CO_2 regulation:

Example Pump ABG: cH 50 (pH = 7.30)
PCO_2 = 7 kPa

PO_2 = 48 kPa (with O_2 sweep flow = 0.4 L/min; CO_2 sweep flow = 0.02 L/min)

To decrease PCO_2: increase O_2 L flow to 0.3

Water excretion

Water vapour is formed when cool gas passes over a warm blood surface. Not only does it cool blood, it can condense in the gas phase, blocking gas transfer ("pulmonary oedema"). The gas flow should be kept above 0.2 L/min to keep this from happening. If oxygenator failure is evident and water vapour is thought to be a possible cause, then the sweep gas (O_2) flow should be increased to the maximum flow rate of the membrane in an attempt to blow off the water vapour. The first sign of this problem may be larger amounts of water droplets coming from the oxygenator gas exit port, and an increase in pump PCO_2. This will be seen before a drop in pump PO_2 occurs. This is an emergency, as total oxygenator failure can occur.

Veno-arterial & Veno-venous ECMO

Veno-arterial ECMO:

In VA bypass, blood drains from the right atrium through a catheter placed in the right Internal Jugular Vein with the tip of the catheter in the right atrium. The blood is oxygenated and returned to the patient through the right Common Carotid Artery. This mode of bypass provides excellent support for the heart and lungs. Pulmonary artery pressure should drop from the mechanical effect of draining the right atrium. If myocardial ischaemia and dysfunction is a major component in the patient's pathology, VA bypass offers excellent support (figure 12).

Advantages of VA bypass:

- Excellent support of the heart and lungs
- Only one surgical site
- Excellent oxygenation at low flows
- Not dependent on cardiac function
- SvO_2 can be used to monitor oxygen delivery

Disadvantages of VA bypass:

- Any particles,bubbles or emboli in the circuit could be infused into the patient's arterial system (i.e. the BRAIN)
- Carotid ligation
- Potential hyperoxia of blood supplying the brain
- Alteration of normal pulsatile flow pattern
- Cardiac stun

Cardiac Stun

Cardiac stun is poorly understood. Myocardium contractility diminishes, and a pulse pressure is barely detectable on the arterial trace. On VA ECLS support this is not life threatening because there will be adequate systemic perfusion. The diagnosis of stun is made on echocardiography, and reduced pulse pressure may indicate tamponade or incorrect arterial cannula placement.

An explanation for cardiac stun is the sudden exposure of the hypoxic myocardium to "hyperoxic" blood. A possible method of preventing this is to ensure that at the onset of VA ECLS, the blender is set lower than 100% (e.g. 60%). It should be recognised that in most cases of cardiac ECLS,

adequate oxygenation will occur at the alveolar level and that VA ECLS predominantly offers mechanical support, so keeping the blender at this level should not be detrimental.

In summary, during VA bypass **with no native lung function**, the factors that increase oxygenation include.

- Increased ECLS flow
- Decreased pulmonary blood flow
- Increased haemoglobin
- Increased CvO_2 (decreased oxygen consumption)

The factors that decrease oxygenation are

- Decreased ECLS flow
- Increased pulmonary blood flow
- Decreased haemoglobin
- Decreased CvO_2 (increased oxygen consumption)

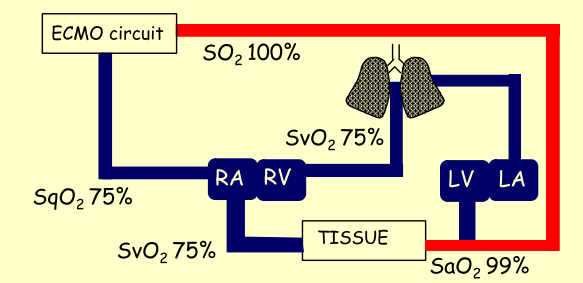


Figure 12 VA ECLS

Veno-venous ECMO:

Types of VV support:

1. Veno-venous single catheter/dual lumen support (OriGen/Avalon):

In VV single catheter/dual lumen bypass the patient's blood is removed from the right atrium via the wider venous channel and re-infused via the narrower "arterial" lumen into the right atrium aiming at the tricuspid valve. Specialised catheters have been developed (12FG, 15FG & 18FG OriGen) for neonates and a range of Avalon cannula for larger patients including adults (see Figure 14).

2. Veno-venous two catheter support:

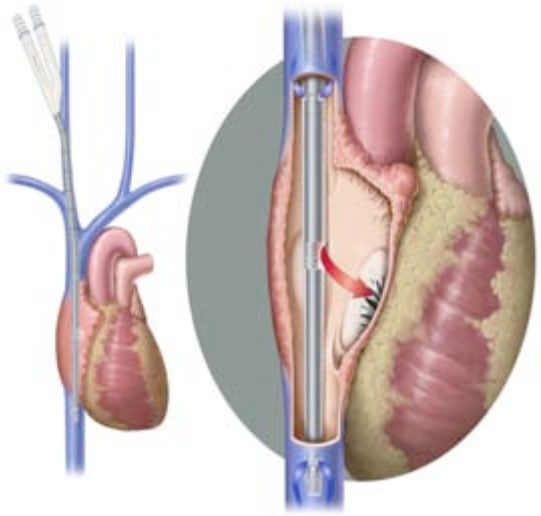
In larger infants and children, a two catheter technique may be used. Usually the blood is siphoned from the right atrium via a double lumen catheter inserted via the right Internal Jugular Vein if drainage / oxygenation was inadequate this may be augmented by inserting a single lumen cannula into the femoral vein(s) in the groin.

Advantages of VV support:

- No carotid ligation
- Particles in the system will theoretically go into the lungs, although with R to L shunts this may not be true
- Hyperoxygenated blood enters the PA and thus may help decrease the pulmonary artery pressure

Disadvantages of VV support:

- Very high flows are required to achieve adequate oxygenation
- Dependent on the cardiac function
- Occasionally requires IJV ligation – if cannula not inserted percutaneously.



Avalon cannulae draining from SVC and IVC

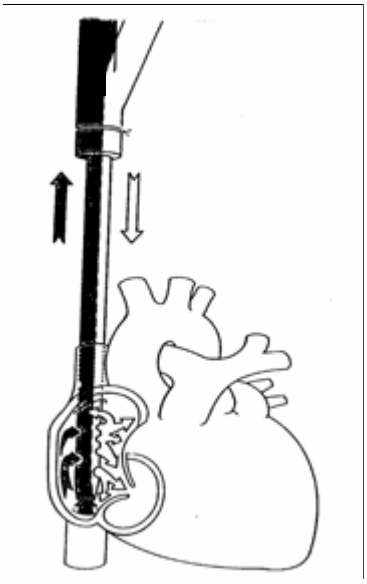


Figure 14 Double lumen VV cannula in right atrium

Circulatory effects of VV-ECMO

Many of the characteristics unique to VV ECLS derive from the fact that both drainage to and re-infusion from the ECLS circuit occur in the central venous circulation – often through the same blood vessel. This has several important clinical implications.

Both VV and VA ECLS are referred to as "bypass" but unlike VA ECLS, VV does not really "bypass" the heart or lungs. Since the volume of blood drained from and returned to the central venous system is equal, VV ECLS does not reduce right ventricular pre-load, pulmonary blood flow, left atrial return or ventricular output. The absence of a change in left ventricular afterload may eliminate the isolated left ventricular "stun" syndrome seen in a subset of VA supported patients.

Echocardiographic studies demonstrate that patients managed on VV ECLS may have improved cardiac function. In contrast to VA ECLS, the oxygen saturation of the blood delivered to the pulmonary artery is higher because oxygenated blood is delivered to the right atrium. The effects of higher oxygen saturation in the pulmonary arteries may decrease pulmonary vascular resistance and right ventricular afterload. In addition, avoidance of increased left ventricular afterload and improved oxygen delivery to the coronary arteries may improve myocardial performance.

Oxygenation during VV-ECMO

The success of VV ECLS is dependent upon the manipulation and management of several factors unique to this therapy, most importantly oxygenation and ventilation. Since VV ECLS provides no direct circulatory support, it can be difficult to achieve the same level of oxygen delivery compared to VA ECLS. However when recirculation is minimal and cardiac output is supported, oxygen delivery to the patient from the ECLS circuit can be similar. Oxygenation is optimised when the haemoglobin concentration is around 15g/dL, when recirculation is low and when the venous drainage catheter is large enough to achieve 120-140 ml/kg/min flow. In practical terms, 3 variables can be used to monitor oxygenation during VV ECLS.

·**Arterial oxygen saturation**

Measured by blood gas sampling or pulse oximetry this is a good reflection of oxygen sufficiency.

·**Pre-oxygenator PaO_2 or saturation**

During VA ECLS this accurately reflects the mixed venous saturation and determines the adequacy of adequacy of oxygenation. During VV ECLS pre-oxygenator saturation over estimates mixed venous saturation because of recirculation. When recirculation is high, values on "venous saturation" monitor will have a high saturation even if the patient's true mixed venous saturation is dropping and oxygen delivery is inadequate. A low value (<70%) may also reflect suboptimal oxygen delivery. An indirect but very important marker of suboptimal oxygen delivery may be increasing metabolic acidosis, rising serum lactate and reducing urine output

.

Recirculation

An understanding of the concept of recirculation and appropriate interpretation of blood gas results is critical to successful application of VV ECLS. Recirculation is defined as "the portion of blood returning to the ECLS circuit immediately after being infused to the patient from the ECLS circuit. The Avalon cannula which drains blood from the IVC and SVC usually results in less recirculation than the Origen cannula.

All patients on VV ECLS have some degree of recirculation. This may present as decreasing patient arterial saturation and increasing pre-membrane saturation. In addition the blood draining from the right atrium is the same colour as the blood returning from the pump.

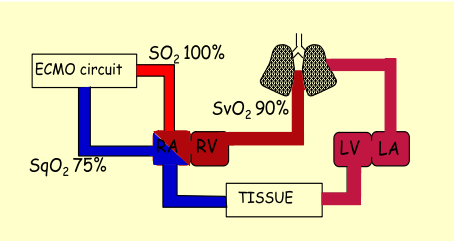


Figure 15 "Ideal" VV circuit with no recirculation

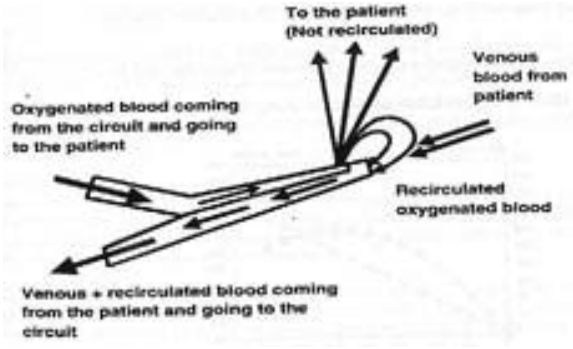


Figure 16 Recirculation diagram in VV double lumen catheter

Four factors can affect recirculation and oxygen delivery:

1. Pump Flow

If pump flow is high, the negative pressure drawing blood from the right atrium back into the ECLS circuit is higher. The increased suction pressure causes streaming of oxygenated blood from the arterial lumen to the venous drainage lumen of the double-lumen catheter. The amount of oxygen delivery provided to the patient first increases and then decreases as pump flow increases beyond optimal flow and recirculation.

Pump flow is the factor affecting recirculation over which the ECLS specialist has the most control. If recirculation is high, try to wean the pump flow. If patient saturations improve or stay the same it may be possible to wean again. If they decrease, the flows need to be increased and other causes for recirculation need to be addressed.

2. Catheter Position

Correct catheter position during cannulation is critical for optimal support during VV ECLS. Echocardiography during the procedure will ensure that the double lumen catheter is positioned so that oxygenated blood returning via the "arterial" lumen is directed towards the tricuspid valve. In larger patients, if the tips of the drainage and return catheters (i.e. catheters inserted in the femoral vein and internal jugular vein) are directed at each other from close range, recirculation will increase.

If recirculation seems to be increased, inadvertent changes in catheter position should be ruled out by CXR or echocardiogram. Surgical repositioning may be necessary.

3. Cardiac output

If the oxygenated blood delivered to the right atrium is rapidly moved to the right ventricle it is less accessible to the drainage catheter. In contrast, during cardiac standstill, all of the oxygenated blood flowing in to the right atrium would drain back into the ECLS circuit because it has nowhere else to go.

Cardiac output is the product of heart rate and stroke volume. Tachycardia should be managed adequate sedation and a quiet environment. Stroke volume should be optimised by increasing intravascular volume and by using cardiotonic drugs as indicated. Improving cardiac output will increase oxygen delivery.

4. Size of the Right Atrium

Intravascular volume, or more precisely, right atrial volume, also influences recirculation. When the oxygenated blood is delivered to a very small right atrium it is more likely to flow directly back in to the ECLS circuit than if the oxygenated blood is diluted in a large volume of desaturated blood in a normal sizes right atrium. If hypovolaemia is the issue volume expanders and blood products are indicated.

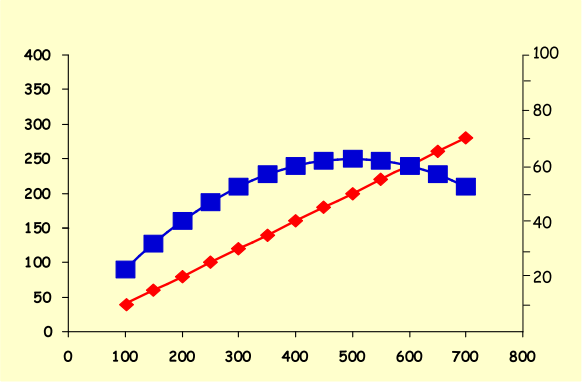


Figure 17 Oxygen delivery with increasing pump flow in VA (red) and double lumen VV (blue) ECLS

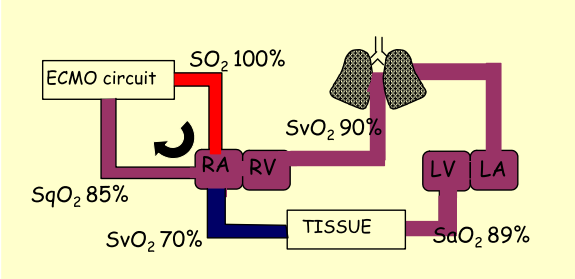


Figure 19 Effect of recirculation on VV double lumen circuit

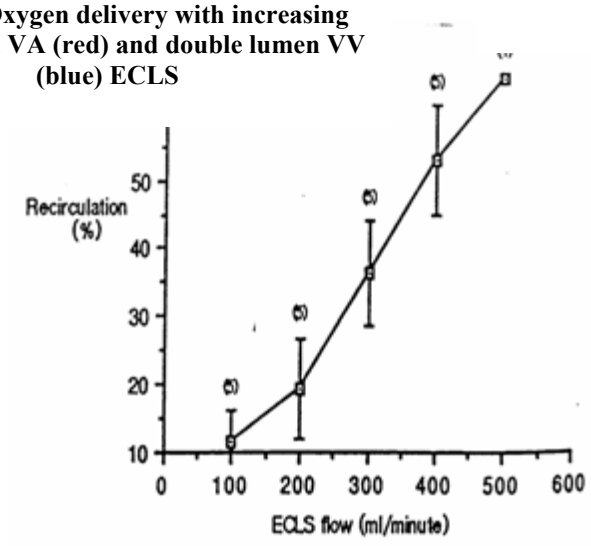


Figure 18 Percentage of re infused oxygenated blood, which is again immediately drained into the bypass circuit, thereby decreasing the efficiency of bypass.

In summary

During VV perfusion with no native lung function the factors that increase oxygenation are:

- Increased ECLS flow
- Decreased percentage of re-circulated pump flow
- Increased haemoglobin
- Increased CvO_2 (decreased oxygen consumption)
- Increased cardiac output

During VV perfusion with no native lung function the factors which decrease oxygenation are

- Decreased ECLS flow
- Increased percentage of re-circulated pump flow
- Decreased haemoglobin
- Decreased CvO_2 (increased oxygen consumption)
- Decreased cardiac output

Patient selection

Neonatal ECMO patient selection

Conventional ventilatory management saves the lives of most neonates with respiratory failure. ECLS is currently used only for neonates who are responding poorly to optimal ventilatory, medical, and surgical treatment, or those in which it is felt that continued high ventilation pressures will cause significant lung damage. We generally consider neonates for ECLS if they have an estimated 50-60% or greater mortality risk despite optimal therapy.

ECLS is a temporary artificial lung. If a neonate's respiratory disorder can be reversed within a 21 day period, it is feasible for ECLS to safely support the patient. It is not practical to use ECLS to treat a chronic disease process such as BPD secondary to ventilator induced lung damage. Therefore we limit initiation of ECLS to the first ten days of assisted ventilation, although it should be remembered that this is an arbitrary number and clinical evaluation of ventilator induced lung injury should be the determining factor. A proper evaluation of the chest x-ray findings must be done before consideration for ECLS support. The common causes of respiratory failure in neonates are generally reversible with ECLS support within a few days.

Many premature infants have severe RDS and would appear to be good ECLS candidates, but 40% of these infants will develop a haemorrhage in the germinal matrix or ventricles without the stress of ECLS. The heparinisation and/or the alterations in pulsatility of blood flow created by ECLS causes a significant incidence of severe intracranial haemorrhage, and thus a prohibitive mortality in this group of infants. Therefore, the infant considered for ECLS should normally be at least 34 weeks gestation or ≥2000 grams. Development of new non-thrombogenic circuits may make ECLS feasible for these infants in the future, but research is still required in this area before this will be possible.

Infants must be systemically heparinised on ECLS and therefore any infants with bleeding complications such as a major intracranial haemorrhage (> grade I) or major pulmonary haemorrhage should not be considered for ECLS.

In selected cases, babies with Grade II IVH may be considered for ECLS with the use of Aprotinin, a Protease inhibitor. Coagulopathies should be corrected prior to ECLS in infants who are septic, etc.

Infants who are failing more conventional therapy are candidates for ECLS support. While it is possible to transfer a child on iNO it is not yet possible to use HFOV. Therefore transfer of a child to an ECLS centre should be considered before initiating HFOV. We are working with neonatal colleagues throughout Scotland to develop criteria for early discussion of potentially suitable ECLS candidates.

Historically at Yorkhill, the Oxygen Index (OI) has been used as an index of severity of lung disease

$$OI = \frac{P_{aw} \times F_iO_2 \times 100}{P_aO_2}$$

[Where Paw is mean airway pressure (mmHg) and PaO_2 is post-ductal (mmHg)]

An OI >40 carries a 60% mortality with present conventional treatment.

Although OI can be a useful measure of severity of lung disease it is not the only indication that ECLS is the optimal treatment. Changes in intensive care and the introduction of therapies such as high frequency oscillation, surfactant therapy and inhaled nitric oxide have reduced the need for ECLS in neonates with respiratory failure. Generally when calculating the OI for patients who are currently on HFOV +10 to their index and the same for patients who are receiving inhaled nitric oxide.

Paediatric respiratory ECMO patient selection

Paediatric respiratory ECMO is a far more controversial therapy than neonatal ECLS. This is not surprising considering the many differences between neonatal and paediatric respiratory failure. Paediatric respiratory failure encompasses a more heterogeneous group of diseases. According to ELSO Registry data the most common diagnoses for paediatric ECMO support are viral pneumonia, acute respiratory failure, non-Adult Respiratory Distress Syndrome (ARDS) and ARDS. ARDS can be caused by diseases as various as viral pneumonia, bacterial pneumonia, bronchiolitis, sepsis, trauma, smoke inhalation and near drowning. These diseases do not usually have the dramatic component of pulmonary hypertension which is often associated with neonatal respiratory failure and they tend to resolve more slowly.

Selection criteria

The ideal utilisation of ECLS for any age group would be for those patients who have a high likelihood of death without bypass support and a high likelihood of survival with it. At this time there are no definitive standardised paediatric ECLS selection criteria. The general approach is to consider ECLS when other forms of therapy are failing. This common-sense approach is understandable, since HFOV and inhaled Nitric Oxide therapy are less invasive and seem to carry less risk than ECLS. However there remains the major question of optimal timing. ECLS runs will be long and ultimately futile if ventilator therapy and the underlying disease have already caused irreversible lung injury. There also remains the completely unanswered questions about which patients should simply not be considered as "ECLS candidates", i.e. which conditions or diagnoses should preclude patients from receiving ECLS. Patients with hypoxic-ischaemic arrests resulting in neurological injury, irreversible lung disease or malignancies and /or immuno-compromised state have generally been excluded from ECLS support. However the decision not to institute ECLS because an arrest has taken place may cost the patient his life at a time when his or her neurological outcome cannot even be evaluated. Many centres would also support children with underlying neoplasia or those in an immuno-compromised position. The decision has to be made within the multi-disciplinary team on a case by case basis in these difficult cases.

At present, and until more definitive inclusion and exclusion criteria are formulated, the decision for ECMO support in a paediatric patient will involve discussion with all staff involved in the care and a risk/benefit analysis undertaken.

There has recently been significant discussion regarding patients suffering with Pertussis who are referred fro ECMO. The UK 4 centres group has agreed that children > 30 days of age may be considered for ECMO. VA ECMO is the preferred option in this setting. We have updated the manual with a guide for leucodepletion using an in-line filter which Perfusion staff will site. The results over the next 6 months to 1 year will be reviewed and guidance updated.

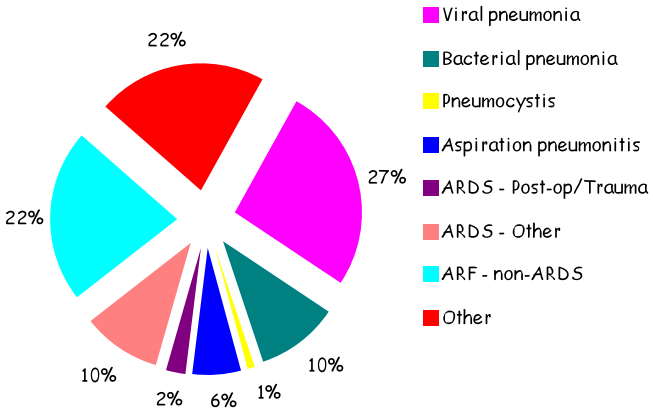


Figure 2 RHSC Paediatric cases by category

Paediatric cardiac ECMO patient selection

Indications for Cardiac ECMO

The main indication for cardiac ECLS is post-operative low cardiac output syndrome (LCOS) associated with ventricular failure. This most often occurs after long and complex surgery associated with extended periods of cardiopulmonary bypass, prolonged cross clamp times and moderate or deep hypothermia. The pre-existing cardiac anatomy may make cardiac failure more likely, particularly if this has involved surgery to the coronary arteries. Most cases of cardiac failure result in severe pulmonary oedema. ECLS support has the advantage of ensuring oxygenation as well as supporting the heart.

Markers of low cardiac output:

- Increasing tachycardia
- Low mixed venous oxygen saturation (absolute value < 40%)
- Arteriovenous oxygen difference > 35%
- Rising filling pressures
- Incrementing inotrope requirement or the need for a third agent
- Toe core difference > 4°
- Sternum open or required to be reopened
- Oliguria
- Lactate increasing or failing to fall
- Persisting metabolic acidosis

Where possible we attempt to identify the potential need for ECLS pre-operatively: this patient group are characterised by a marginal preoperative ventricular function and/or a planned procedure that will radically alter the haemodynamics of the circulation. Those where the

severity of LCOS is unexpected tend to require shorter runs and have a more favourable outcome.

Cardiac and pulmonary function should be deemed reversible, although this prediction is not reliably clear in the immediate post operative setting. It is contentious as to whether someone who is not considered appropriate for transplantation should be commenced on ECLS. However, ECLS as a bridge to transplantation is an established technique in centres that undertake paediatric cardiac transplantation. Late survival rates up to 43% have been reported.

There is a strong argument that the use of ECLS in the postoperative cardiac patient should be restricted to those who have had a complete repair of the underlying lesion. Several centres have reported poor results when using ECLS after palliative procedures, but patients with single ventricle physiology are the fastest growing group of cardiac support on the ELSO registry.

Scenarios following cardiac surgery which may require ECLS:

- Myocardial dysfunction post-cardiomyotomy
  - Failure to separate from CPB
  - Progressive low cardiac output state
    - Arrhythmia
    - Cardiac arrest
    - Hypoxia-respiratory insufficiency
    - Pulmonary hypertension
    - Right heart failure
    - Shunt occlusion
- Elective augmentation: post-cardiomyotomy in HLHS
- Stabilisation between elective procedures
- Peri-transplant:
  - As a bridge
  - Post transplant to support RV failure induced by pulmonary hypertension
  - During acute rejection

The literature supporting emergency cardiopulmonary support is mounting. Survival rates vary in different series from 45-80%. This indicates that paediatric cardiac patients are salvageable and deserve an aggressive approach with rapid deployment of ECLS. Early restoration of cardiac output improves survival. Survival decreases with duration of CPR from time of *witnessed* arrest. We have little experience of using ECLS in the cardiac arrest setting (E-CPR). Other groups have reported variable success, with survival to discharge being reported following over 90 minutes of cardiac massage before initiation of ECLS. The potential use of ECLS in this setting is clearly dependent on the speed with which support can be initiated, though there is increasing evidence to support the statement that it is the effectiveness rather than the duration of CPR that is important, and if there is evidence of persisting neurological function then ECLS should be commenced.

A 60% survival to discharge rate has been reported by a group that could initiate crystalloid primed ECLS within 15 minutes of cardiac arrest. Other groups report a survival to discharge rate of between 11 and 64% for initiation of E-CPR up to and over 60 minutes after the cardiac

arrest. As the circuit technology improves with the development of small, low-prime volume, centrifugal circuits, it is likely that commencing with a clear primed circuit will become more common. The rate limiting step for the use of E-CPR is staff availability. A clear primed circuit can be used immediately upon the request of the clinical team, although access to fresh O-negative blood is readily available.

When should Cardiac ECMO be started?

There is a lack of consistent accepted threshold criteria on which to commence cardiac ECLS. Unlike the oxygenation index in respiratory ECLS there is not a reliable predictor of mortality in cardiac ECLS. It is accepted that early initiation of ECLS, ***prior to*** a cardio-respiratory arrest offers the best chance of success. This avoids the end organ morbidity associated with prolonged periods of low cardiac output. However, ECLS is invasive and associated with life threatening complications.

Acknowledging an extremis situation which is not improving should mandate consideration and formal discussion between intensive care, cardiology and cardiothoracic surgery. Clearly the more indicators of low cardiac output state, the greater the need for a period of recovery.

Our unit's agreed criteria are in the appendix section of the cardiac chapter.

ECMO Patient referral process

NICU

- **Internal referral**
  - Case discussion between "responsible" clinicians including Nurse-in-charge of NICU

- **External referral**
  - Call placed through to NICU main desk (0141-201-0255/0258)
  - Initial details taken re patient status, location, phone numbers etc and recorded on the ECMO referral form by nurse-in-charge.
  - Contact on call Neonatologist inform of referral and establish bed availability.
  - On call Neonatologist to discuss with referring centre re advice, suitability of referral and transport options
  - Contact transfer team to arrange retrieval
  - Inform surgical colleagues, perfusion staff, Cardiology, Radiology and blood bank if ECMO retrieval to be undertaken
  - All referrals should be recorded even if refused and reason for refusal noted ie clinical/staffing/bed availability

PICU

- **Internal referral**
  - Case discussion between "responsible" clinicians including Nurse-in-charge of PICU.
  - Inform perfusion staff, surgical colleagues, cardiac consultant and technicians and blood bank.

- **External referral**
  - Call placed through to PICU main desk (0141-201-0081/3) or on dedicated retrieval phone line (0141-201-6923).
  - Initial details taken re patient status, location, phone numbers, blood group, size of circuit & membrane if on ecmo already etc and recorded on the retrieval form. Ensure that ECMO referral box is highlighted.
  - Even if the referral is finally declined a referral form should be filled in
  - Inform PICU Nurse-in-charge of referral and establish bed availability.
  - Contact Duty PICU Retrieval Consultant to discuss and enact solution to case.
  - Inform surgical colleagues, perfusion staff, cardiac consultant and technicians and blood bank if ECMO retrieval to be undertaken.

Please ensure all referrals are recorded inspite of outcome as this is a quality indicator for the ECMO service

Preparation for ECMO

Once a patient referral has been accepted, the allocated ECMO specialists will assume responsibility for liaising with the rest of the team, ensuring that the bed space is prepared and that preparation for the cannulation is processed smoothly. Cannulation requires the orchestration of a large group of individuals and equipment therefore the RHSC Clinical Cannulation Guideline has been developed to aid this process.

Quick guide to cannulation for ECMO

Staff required

1. On call Paediatric or Cardiovascular surgeon +/- Cannulating Paediatric Intensivist
2. Theatre Staff – scrub & circulating nurse
3. Consultant Neonatologist/Intensivist
4. ECLS specialist x2
5. Perfusionist (contact at appropriate time - not too early)
6. If veno-venous double lumen cannulation Cardiology Consultant and/or technician
7. Radiology for CXR and Cranial ultrasound if appropriate age

The following equipment and supplies must be available:

- **Theatre equipment:** prepared by theatre nurses
- **Diathermy:**
- **Head light:**
- **Extra wall suction:** from theatre
- **ECLS cannulae:** from ECLS store room, see guide sheet for sizing (cannulator responsible for choosing size and obtaining cannulae)
- **ECLS circuit:** primed by perfusionist

Blood products:

Inform blood bank early, as supplies may need to be brought in from BTS. Neonates will require O negative blood ordered if blood group not available. Remember to obtain a cross match specimen prior to starting ECLS. *See below for procedure for ordering blood when patient not yet "on-site".*

The ECLS circuit for the centrifugal pump is low volume and can generally be **clear primed** for patients over 10kgs with Plasma-Lyte 148 which is a buffered, balanced electrolyte solution. A decision may be made to top the patient up with PRBC as ECLS flow is initiated. A 50ml syringe can be attached to the circuit and PRBC given as volume when flow is commenced. Perfusion will add 1000units Heparin to each 1000ml bag. If patient is less than 10kgs then circuit will be blood primed. All circuit changes will be blood primed and in circumstances such as low HCT/post operative cardiac a clinical decision will be made by ECMO clinician. Generally, due to the smaller foreign surface of tubing, platelets will not need to be bolused into the circuit as ECLS is commenced. However, patients with a low pre ECMO platelet count

(<100,000) will need to be transfused as ECLS is commenced. A FBC & Coagulation screen should be obtained as soon as possible following cannulation and platelets can be topped up as required.

Blood products will need to be ordered from blood bank for each patient prior to cannulation to cover emergencies and routine top up once support is initiated. At cannulation 1 unit of blood should be requested in the "blood bank box" which can be checked re patient details without opening the box and then returned to blood bank within 3hrs for re-issuing if not utilised.

Mode of Support	Timing of run	Weight range	Products to be available daily in blood bank	Products required at bedside for cannulation
ECMO	**Cannulation**	**<5kg**	• **2 Priming units (use O –ve blood in emergency).** • **1 Large volume PRBC top up - (O -ve blood in emergency)** • **4 Paedipak PRBC for top up** • **A split of 4 Paedipak platelets for top up**	• **1 Priming unit with additions – Give to perfusion** • **1 Large volume PRBC top up in cold bag at bedside** • **1 Paedipak PRBC drawn up at bedside** • **1 unit Platelets if last count <100,000**
		5-10Kg	• **2 Priming units (use O -ve blood in emergency).** • **3 large PRBC for top up** • **3 units' donor apheresis platelets.**	• **1 Priming unit with additions – Give to perfusion** • **1 Large volume PRBC top up in cold bag at bedside** • **I unit Platelets if last count <100,000**
		>10Kg **Clear Prime routinely but D/W Intensivist as cardiac / low Hb patients may require blood prime)**	• ***2 Priming units (if required)*** • **3 large PRBC for top up** • **3 units' donor apheresis platelets**	• ***If required 1 Priming unit with additions – (Give to perfusion)*** • **1 Large volume PRBC top up in cold bag at bedside** • **1 unit Platelets if last count <100,000**
ECMO	**During run** If ongoing bleeding present consider previous 24hrs usage when ordering as the above is only a minimum base line	**<5kg**	• **1 Priming unit** • **4 Paedipak PRBC for top up** • **A split of 4 Paedipak platelets for top up**	
		>5kg	• **1 Priming unit** • **3 large PRBC for top up** • **3 units' donor apheresis platelets**	
Patients < 4.5 kgs for respiratory ECMO may be supported on the roller pump therefore all respiratory patients in that weight group should have 2 priming, 4 Paedipak top up and 4 platelets ordered for them. If they are subsequently managed on the centrifugal pump this can be reviewed.				

Blood product ready reckoner (See Guideline 6a)

Drug preparation:

Prior to draping, ensure reliable (preferably central) intravenous access is available and accessible.

- **Heparin bolus** 50 units/kg (x 2)
  - 1st bolus is given immediately prior to insertion of cannula with adequate flush. Check with the surgeon re timing.
  - If ECLS is not established within 20mins inform the surgeon. Check ACT- if <350 2nd bolus should be given. Inform the surgeon.
- **Anaesthesia**

- Fentanyl 10-20 mcg/kg
- Vecuronium 0.1-0.2 mg/kg (if not currently muscle relaxed)

- **Emergency Drugs**
  - Adrenaline
  - Atropine
  - Calcium
  - Sodium Bicarbonate

    Have minijets open and dosages worked out based on patient weight.
- **NaCl 0.9% flush** 10 ml x 4

Volume colloid and blood already drawn up in syringes (remember to filter all blood products). 50 ml syringes can be used for circuit volume once on ECLS. Use 20ml syringes for neonates during cannulation as volume will be given to the patient.

Heparin infusion, made up as per protocol, should be attached to circuit and pump programmed prior to cannulation.

Arterial monitoring is **essential** during cannulation for ECLS and must be in place before draping for cannulation and heparinisation.

Patient position / preparation:
The patient will be placed with their head towards the foot of the bed/bassinette. Neonates nursed on radiant warmer (check x-ray plate available). Paediatric patients should be nursed on the cardiac beds. All patients should be nursed on appropriately sized repose mattresses.

Place a roll under the shoulders to extend the neck and turn the infants head to the left to expose the right side of the neck for ECMO cannulation. Care should be taken to ensure that the ET tube is in a good position prior to cannulation (check CXR) and that it is not dislodged or kinked when positioning the head. If the patient is oscillated this **may** need to be discontinued and hand ventilation initiated during the cannulae insertion. **Ensure diathermy pad is attached**.

Immediately after cannulation:
Once ECMO has been commenced, an ACT should be obtained. If ACT is <300 heparin infusion should be commenced at 50units/kg (2mls/hr). ACT should be checked every 15min until stable. If patient has significant ongoing blood loss see the *Protocol for Management of Post op Cardiac Patients* for actions. A full circuit check should be undertaken and handover sheet filled out with the perfusionist. Obtain FBC, U&E, Coagulation screen and ABG. Adermal pads should be placed under the shoulders and occiput and the heels and elbows accessed for further pads.

The correct positioning of cannulae is checked on a plain x-ray immediately following cannulation. There is a radio-opaque mark at the distal end of the smaller venous Biomedicus cannula (8-14F) and this point will typically be located close to the RA / IVC junction. ECHO will confirm the position of the VVDL cannula and if there are concerns with drainage problems or position (this is the best way of checking cannula position and blood flow). Please ask the cannulator to return the patient's head to neutral position where possible after cannulation (prior to final ECHO) to aid left sided cerebral venous drainage. Once the cannulae are secured to the external skin, (observe for pulling to skin / twisting), use

the ECMO circuit gripper plate to secure the lines. All ECMO parameters, fluids and drugs must be written up post cannulation.

Remind the cannulator to write up the procedure in the notes, with size of cannula, depth and any concerns regarding the procedure.

Unsuitable cannula position when starting ECMO usually causes increased negative pressures on the venous pressure line and blood flow problems (although not always immediately). Likewise, apparently 'good' cannula positioning that does not permit full flows (for that cannula) should be investigated. Do not let the cannulating team or perfusionist disappear unless all is well (good blood flows achievable + reasonable circuit pressures). Decisions on whether to adjust cannulae position will be made in conjunction with the cannulator.

Procedure for ordering blood when patient not on site

A **Cannulation Blood Product Downtime pack** which consists of 1 Blood Bank (BB) form and 4 Porters collection forms (for large priming unit, top up and platelets) will be available on each ECMO cart. The forms should be completed for each external patient following discussion with blood bank. These should be used for ordering ECMO cannulation blood products when patient is not yet on the RHSC system. If the patient is already admitted then normal procedures should be followed.

First **contact with BB following referral** of a patient from another hospital will be by phone, directly followed by the placement of completed downtime forms in pod system. Forms must have patients full name (baby where appropriate instead of first name), DOB and reported blood group. The products ordered separately on each form will include emergency unit, top up unit, and Platelets. Any form not utilised during the cannulation will be discarded by BB. BB will order in O-ve blood and A –ve or low titre O platelets and immediately label them when they arrive on site.

When the products are required the porter should be phoned to collect them and will retrieve downtime collection form from BB. A cross match sample should be obtained from the patient ASAP following admission (ideally pre-cannulation) and the electronic Trakcare system should be used for all further blood product requests.

Clear primed circuits

The new centrifugal circuit can be primed quickly by perfusion in the event of a clinical emergency and therefore a request for a standby clear primed circuit should be rare. Where a request is made by medical staff for a circuit to be "on standby" for an inpatient a formal request via Trakcare must be made at the same time for cannulation blood products. Blood bank will order in blood and platelets and label them for "ECMO Use" This must be reviewed every 24hrs and de-reserved when no longer required.

Cannulation

Personnel Required:

- Paediatric or cardiovascular surgeon
- ECLS Physician
- ECLS Specialists
- Theatre Staff
- Perfusionists
- Cardiology (echocardiogram for VV DL)
- Radiology for pre-cannulation CxR and Skull Ultrasound

Medications Required:

- Vecuronium (0.1mg/kg)
- Fentanyl (10-20mcg/kg)
- Resuscitation drugs
- Volume PPS/Saline
- Heparin (2x50iu/kg bolus & Heparin infusion)
- Surgicel and/or Fibrin Glue

Cannulation requires the orchestration of a large group of individuals including those responsible for the bypass circuit, those responsible for the surgical aspects, and those responsible for the medical aspects of the procedures. The above medications and emergency drugs should be at the bedside. Blood and/or PPS should be available for emergencies. The patient should be paralysed if not already, and given anaesthetic doses of Fentanyl (10-20 mcg/kg). This can be given as a split dose to reduce the concern for hypotension.

Team Brief

This is essential prior to any cannulation ar indeed any procedure taken on an ECMO patient. This allows a quick summary and highlight of plan and delineation of roles and responsibilities during cannulation.

Role allocation during ECMO cannulation:

The ECLS Physician acts as the anaesthetist, monitoring the vital signs and giving medications during the procedure. One ECLS specialist assists the perfusionist in management of the circuit, while the second ECLS specialist assists in patient monitoring and recording. The cannulator concentrates on cannulation.

Positioning of Patient:

The patient is positioned with his head to the "foot" of the bed. The patient's neck is then hyper-extended with a shoulder roll and the head turned to the left. It is important at this point to check that the ET tube is secure and not kinked. Be sure that the diathermy ground pad is placed at this time. The patency of IV access should be checked and easily available once draping has occurred. An iv extension line may be needed. All monitoring should be satisfactory before draping occurs.

Perfusion:

The primed circuit will be brought to the patient's bedside prior to cannulation. Perfusion will connect

the circuit gases. Sweep O_2 will be set to deliver a one to one gas blood flow at initiation of support. The pressure transducers will be set up and zeroed to the circuit. Pre and post membrane pressure lines are connected to the console and venous (inlet) pressure line to the patients monitor. The perfusionist will take responsibility for setting initial RPM of 1500 and adjusting to achieve required flows as support is increased. An ECMO specialist may be required to assist by holding up the ECMO tubing for cannulation and until lines have been secured.

Prep and Drape:

A wide prep of the patient's neck and chest is then done with Betadine solution or 2% Chlorohexidine. The right ear is prepped into the field. Clear drapes are available to assist clear access to patient during cannulation. Towels may be used to cover the infant's head. Steri-drape may be used to secure the towels to the skin of the neck.

Anaesthesia:

The patient is sedated with Fentanyl (10-20 mcg/kg) and paralysed with Vecuronium (0.1 mg/kg). These drugs are given by the ECLS physician. Prior to the skin incision the tissues of the right neck are infiltrated with 0.5% lignocaine with 1:200,000 adrenaline.

The surgeons will identify the internal jugular vein (IJV) and carotid artery. As soon as the carotid artery is isolated, the heparin loading dose should be given by the ECLS physician. Although it is the surgeon's responsibility to ask for the Heparin to be given at this stage the whole team should assume collective responsibility for timing of the Heparin bolus. This should be noted and written down by the ECMO specialist. In the majority of cases the bolus will be 50 units/kg. If ECLS has not been established within 20 minutes of the initial bolus inform the ECLS Physician/Surgeon. A further heparin bolus may be necessary. An ACT of 350 sec. should be maintained while there is stagnant blood in the catheters – i.e. pre institution of ECLS.

Surgical VA open assisted cannulation:

Incision:

After injecting with lignocaine/adrenaline, a 1-2 cm incision is made over the right sternocleidomastoid muscle. The cutting current of the diathermy is used for the skin incision which is continued through the subcutaneous tissue. All visible blood vessels are coagulated. The fibres of the sternocleidomastoid muscle can then be spread apart with a hemostat. Alternatively the muscle may be retracted anteriorly or posteriorly depending on the anatomy. The carotid sheath is opened. Usually the common carotid artery is isolated before the vein. It is important to remember that the vagus nerve usually lies posterior to the vessels in the sheath. The nerve should be identified and avoided. The artery is then encircled with proximal and distal 2-0 silk ties, held with clamps but not tied. The internal jugular vein is left alone at this stage. It is isolated after cannulation of the artery. This is to avoid spasm of the vein which occurs even with minimal handling.

Preparation of Cannulae:

ECLS catheters from size 8-14 French (in neonates) are used. Choose the cannula size to approximate the size of the vessel. To estimate the position of the cannula, identify the sternal notch and the xyphoid process. The arterial cannula will be placed approximately one-third the distance from the sternal notch to the xyphoid (usually 3.5 cm in a neonate). After determining this position, note the position of entrance into the artery on the cannula.

The venous cannula position is estimated by a point half way down from the sternal notch to the xyphoid

(usually 6-7 cm in a neonate). Remember that the neck is hyperextended and the chest is hyperinflated. When these two factors are corrected the cannula will descend. Allow for this when you determine catheter length. Biomedicus arterial and venous cannulae are the most suitable.

Arterial Cannulation:

The arterial cannula is placed first. The patient is heparinised with 50 units/kg of heparin sodium. After waiting 60-90 seconds the cephalad ligature on the common carotid artery is tied. A bulldog clamp is then placed at the proximal artery. Be sure the artery is filled with blood before placing the bulldog. This will dilate the artery. Traction on the proximal silk tie may cause severe spasm of the artery. An arteriotomy is then made with a #11 blade, (two traction sutures of 6-0 prolene can be placed on the sides of the arteriotomy to aid cannulation). Lubricated Garrett dilators are used to dilate the arteriotomy to the approximate size of the cannula chosen. Before placing the cannula, be sure that a sterile tubing clamp is occluding the cannula. The lubricated arterial cannula is then advanced as the bulldog clamp is released. The cannula is secured with the proximal 2-0 silk ligature, tied over a 1-1.5 cm piece of vessel loop ("bootie"). A second proximal 2-0 silk ligature is placed about the artery and cannula and ligated over the bootie. The distal 2-0 silk ligature is likewise tied around the cannula and then one of these strands is tied to a strand from the initial proximal ligature. Test for good flow through the cannula by transiently releasing the tubing clamp.

Venous Cannulation:

The vein is dissected and surrounded with two, 2-0 silk ties. Be careful not to use the tie to assert traction or the vein will go into spasm. A bulldog clamp is placed first on the distal vein closest to the chest inlet to allow blood to distend the vein and then the cephalad 2-0 silk is ligated. The venotomy is made with a #11 blade (two stay sutures of 6-0 prolene can be placed as traction sutures). You'll be glad you have them if the vein tears! The vein is dilated with lubricated Garrett dilators. The venous cannula is likewise lubricated and a sterile tubing clamp placed on the upper portion of the cannula.

With insertion of the venous cannula, pressure on the liver must be applied to ensure that blood flow is out of the catheter and that air is not sucked into the venous catheter (the IJV catheter is in the RA, and thus a negative pressure chamber, which will suck in air). This can take 8 -10 ml blood, so the MAP should be monitored closely during catheter placement.

As the assistant presses on the liver the cannula is inserted as the bulldog clamp is removed by the assistant. Bleeding may occur from the side holes in the cannula, and it may be necessary to transfuse at this point if there is delay in inserting the catheter beyond the side holes. There is often a slight hold up at the thoracic inlet. Do not push or the vein will rip. Instead, use gentle downward and backward pressure. The cannula is secured by ligating the proximal 2-0 silk ligature over a bootie. A second proximal 2-0 silk ligature is passed around the vein and cannula and secured with the bootie under the knot. The distal ligature is tied around the cannula and then a strand from this, as well as one of the proximal ligatures are tied together. Flow is tested by placing pressure over the patient's liver as the tubing clamp is temporarily released. Obtain an ACT.

Percutaneous VA cannulation:

It is rarely appropriate to perform percutaneous VA ECLS cannulation. Venous cannulation is not difficult but arterial cannulation is. It requires a great deal of skill and it is very easy to damage the carotid artery or even to advance too far and damage the aortic valve. It should only be performed in exceptional cases by very experienced surgeons who can deal with any emergencies arising.

Percutaneous Veno-venous cannulation

In respiratory ECMO cases this is a more physiological method of support. In neonates size constraints may make VV ECLS inappropriate. Most older patients will be supported by VV ECLS unless exceptional circumstances dictate otherwise (e.g. post-op cardiac, sepsis).

Veno-venous cannulation is the normal first line approach for patients requiring ECLS support for respiratory reasons. This will usually be either a 2 catheter or single catheter (double lumen) system.

This is normally achieved by the route using the routine Seldinger technique. A Doppler ultrasound probe can be used to better locate the Internal Jugular Vein (IJV).

Initial venous access uses a percutaneous sheath kit (MeritMedical Prelude + needle sheath introducer). A 21G needle and a 0.018" guidewire are inserted aiming to pierce the IJV 1-2cm above the clavicle aiming the needle towards the right nipple. The guidewire is advanced into the RA under echocardiographic control. The skin incision is widened to accept the appropriate size cannula and a (previously) heparinised saline primed 4F (7cm) step-up sheath (& associated introducer) is inserted to the hilt.

The original guidewire and the introducer are removed and the larger 0.038" guidewire from the OriGen percutaneous introducer kit (OriGen PCTa) is introduced and its position in the RA confirmed by echocardiography. The sheath is then removed taking care not to dislodge the guidewire. The vein is dilated with the appropriate dilator(s) (see below) and the cannula is inserted with its introducer over the guidewire.

There are three dilators and two guide wires in the Origen™ cannulation set:

- The Tan dilator for the 12FG DL cannula
- The Tan followed by the Blue dilator for the 15FG DL cannula
- The Tan followed by the Blue followed by the Green dilator for the 18FG DL cannula.

When inserting an Origen cannula the (long) introducer should protrude from the cannula tip as far as the surgeon wishes to insert it. The introducer is then steadied and the cannula in advanced over the introducer with a "wiggling" motion to facilitate the step-up from introducer to cannula as it enters the IJV. Once confirmed in the RA, the guidewire and introducer are removed together and the 'venous' limb of the cannula is clamped with a tubing clamp. The 'arterial' limb bung is then removed and a further tubing clamp used to occlude this limb. Ensure the 'arterial' limb of the cannula is uppermost.

The cannula is now ready to connect to the ECLS circuit.

When inserting an Avalon Bi-caval cannula, initial access is as for the Origen. Once the sheath is in place, the Avalon Elite Vascular Access Kit is utilised. The main difference is that the 0.038" guidewire needs to be directed into the IVC under echocardiographic or fluoroscopic (depending on size) control. The technique for dilatation is similar to the OriGen but the cannula introducer comes with the cannula and is customised for the specific cannula. It is introduced under echocardiographic or fluoroscopic (depending on size) control as in a classic Seldinger technique, ensuring the tip of the cannula is in the upper IVC and the middle (return) side-hole is sited in the RA. Ensure the 'arterial' limb of the cannula is uppermost.

The open-assisted procedure:

A modified percutaneous approach can be used for double lumen veno-venous cannulation.

The jugular vein is exposed but not dissected from surrounding structures by a small transverse incision

in the neck. The vein is cannulated by a percutaneous needle puncture between the ear and the incision. The needle can be watched entering the vein and the 0.038" guidewire is inserted. The vein is dilated as appropriate for the cannula to be inserted. The cannula and its introducer are inserted together as described for the percutaneous technique above.

Notes on cannulation:

1. At the upper limit of our age range it is conceivable that we would need to augment venous drainage. This would need to be done by establishing surgical access to a femoral vein to add a further large bore venous cannula.
2. Echocardiography is essential when inserting a double lumen cannula into the right internal jugular vein to ensure accurate positioning of the catheter tip in the right atrium.

Connecting the ECLS circuit:

Always connect the arterial cannula to the ECLS circuit before the venous cannula. (The arterial side of the system is identified as the tubing coming from the oxygenator). **It is the cannulator's responsibility to ensure that the system is connected correctly.** The cannulae and connectors are then filled with heparinised saline. The arterial cannula should be held upright in the surgeon's left hand and then the non-sterile arterial tubing (with tubing clamp as far away as possible to give maximum room for attaching the tubing) will be given to the cannulator by the perfusionist or ECLS specialist.

The arterial cannula and tubing are then filled with heparinised saline until all air bubbles are removed from both. Remove and replace the tubing clamp on the cannula (don't forget to put your thumb over the end) to release any bubbles trapped by the clamp. The connector is placed into the tubing as heparinised saline is irrigated onto the connection site. After they are securely joined again check for any air bubbles including under the tubing clamp on the cannula. If any are present the connection must be broken and the procedure repeated. If no air bubbles are present the sterile tubing clamp can be removed from the arterial cannula. The venous cannula is connected in a similar way. Again remove all air bubbles from the cannula and tubing by filling each with heparinised saline. After the connection has been made, again check the tubing for any air bubbles. If none are found the sterile tubing clamp can be removed from the venous cannula.

Proper cannula position for the arterial cannula will be confirmed on X-ray if the tip of the cannula is at the level of the T4-T5 vertebral body or the level of the carina. The venous cannula should be in the right atrium and, with the patient's neck hyperextended, optimal position of the tip (metal dot) will be approximately 2.0-2.5 cm above the level of the right hemidiaphragm.

The patient is now ready to be placed on ECLS. The Perfusionist will set the initial RPM to 1500 for all circuit sizes. The venous side will be unclamped by the Perfusionist then the "bridge" will be clamped and then the arterial side of the system will be unclamped (V-B-A). Flows will be slowly increased to ensure the desired flows can be achieved in VV ECMO. This is usually 50-60% of cardiac output for neonates (120ml/kg VV, 100ml/kg VA) (200ml/kg = 100% for neonates) and up to 100% for older patients. Patient MAP and circuit inlet pressures should be closely observed during this period. It is important to remember that the centrifugal pump is not servo regulated, therefore a maximum operating venous pressure of -50cmH_2O should be targeted (upto -99 for large patients on VV ECMO) . Excessive negative inlet pressures significantly increases the risk of cavitation and air in circuit. Volume may need to be given in small aliquots if this is a problem. Cannula size, maximum rated flows for circuit and cannula position should all be checked if venous pressures continue to be problematic.

Closure of the Wound:

Cover the non-sterile tubing with a towel. Prior to fixing the cannula(e) and closing the wound (open technique), be certain that the cannulae are in good position as confirmed by ECHO. Any site of bleeding is looked for and coagulated. The wound is then packed with thrombin-soaked Surgicel, or Fibrin glue if appropriate. The skin is approximated with a running 4-0 vicryl on an atraumatic needle. After the skin is closed and the suture tied but not cut, the same suture is used to secure each cannula separately at the skin level, and then both cannulae are tied together using the same 4-0 vicryl. Next the area behind the patient's right ear is anesthetised and, using a 2-0 silk or Ethibond (larger for bigger patients) suture on a GI needle, each catheter is secured separately. The cannulas are then tied together. The incision site should be cleaned and an IV3000 dressing applied. If the site is oozing despite the application of pressure a small piece of Kaltostat can be placed over the insertion site prior to the IV3000 dressing. (See cannula care protocol)

Lastly, be sure that the tubing is fixed securely to the bed without any traction on the cannulas. Leave enough "play" in the tubing to be able to move the patient's head slightly. The head should be held midline to prevent obstruction of venous return on the contralateral side of the neck.

Cannula care:

Cannulation sites should be managed as per the "cannula care" protocol in the protocol section. This allows optimal and consistent cannula securing and skin cleansing to minimise the risks of secondary infections.

Exchanging a percutaneously inserted OriGen cannula

Indications

- Inadequate flows due to too small cannula.
- Clots in the cannula.
- Kink in the cannula.

Preparation

- This procedure should be undertaken under ECHO guidance.
- Use the appropriate size introducer for the cannula to be exchanged.
- Insert the 0.038" guide wire to just inside the tip of the introducer.
- Estimate the length of introducer to the tip of the cannula to be exchanged.
- Cut retaining sutures and withdraw the in situ cannula to the level of SVC/RA junction for safety.
- Ensure the patient is as prepared as possible to come off ECMO support (including having an adequate ACT).

Procedure

- Come off bypass (A-B-V) and clamp both limbs of the OriGen cannula.
- Cut the ECMO tubing close to the cannula.
- Insert the introducer/guide wire down the "venous" limb of the cannula until you feel it reach the tip of the cannula while taking off the tubing clamp at the same time.
- Don't advance the introducer past the cannula tip but do advance the guide wire at this stage under ECHO guidance.
- Holding the introducer steady, withdraw the cannula over the introducer.
- Put gentle pressure on the exit site and hold the introducer steady.
- If the replacement cannula is the same size, insert it over the introducer taking great care not to let the introducer advance further into the heart (risking perforation and tamponade).
- If the cannula is to be larger, remove the introducer over the guide wire ensuring the guide wire remains in the RA. Insert the larger introducer and cannula as you would for a first time cannulation.
- Reconnect tubing (ensure correct orientation) and reinstitute ECMO support (V-B-A).
- Check cannula position and flows before fixing the cannula in situ.

Notes

- Always do under ECHO guidance though it is difficult to separately identify the component parts (cannula, introducer, guide wire) on ECHO.
- This is not a procedure to be undertaken lightly. Ensure it is appropriate.
- There is a real danger of tamponade if the introduced is advanced beyond the cannula tip (main reason for withdrawing cannula to upper RA before proceeding).
- If the "J" end of the guide wire is advanced into the cannula without the introducer there are 2 potential problems:
    - There will be significant back-bleeding from the cannula around the guide wire
    - The "J" may exit a side hole in the cannula and get stuck, necessitating removal of both cannula and guide wire (not good!)
- If the straight end of the guide wire is used there is a higher risk of cardiac perforation than with the "J" end.
- Take the time to rehearse all moves in advance so every member of the team is familiar with their role.

ECMO patient management

This chapter is separated into subsections highlighting the subtle differences in patient management between neonatal, respiratory and cardiac ECMO support as well as giving an overview of factors such as coagulation, renal support, microbiology, nutritional support, CNS surveillance.

Neonatal ECMO

Introduction

Extracorporeal Life Support (ECLS) has a long history which is interwoven with the development of cardiopulmonary bypass. Dr John Gibbon began working on techniques for extracorporeal support in the 1930's after a patient died from a pulmonary haemorrhage. This was followed by years of refinement in the laboratory and a pump oxygenator was used to treat the first patient in 1953. In 1982 Bartlett published the initial ECMO experience with 45 neonates. ECMO had only been used when maximal conventional therapy was exhausted and the infants were considered moribund. With >50% survival in patients considered to have a 90% mortality interest in ECMO for newborn respiratory failure was high but even though these early results were promising the lack of a randomised control trial caused many medics to continue to doubt it's safety and efficacy.

The UK Collaborative Neonatal ECLS Trial was undertaken throughout the UK to assess whether a policy of referral for ECMO has a beneficial effect on survival to 1 year without severe disability in comparison with conventional management. Between 1993 and 1995, 185 mature (gestational age at birth >35 weeks, birth weight >2kg) newborn infants with severe respiratory failure (oxygenation index _40) were enrolled from 55 hospitals in a randomized comparison of either referral to one of five specialist centres for consideration of ECMO or continued intensive conventional management at the original hospital. The most common diagnoses were persistent pulmonary hypertension due to meconium aspiration, congenital diaphragmatic hernia, isolated persistent fetal circulation, sepsis, and idiopathic respiratory distress syndrome. Of the infants allocated ECMO, 84% received this support. Recruitment to the trial was stopped early (November, 1995) by the trial steering committee on the advice of the independent data-monitoring committee, because the data accumulated showed a clear advantage with ECMO. 124 children were enrolled before December, 1994; those who survived to 1year of age underwent neurological assessment at that age

Overall, 81 (44%) infants died before leaving hospital and two are known to have died later. Death rates differed between the two trial groups; 30 of 93 infants allocated ECMO died compared with 54 of 92 allocated conventional care. The relative risk was 0·55 (95% CI 0·39—0·77; p=0·0005), which is equivalent to one extra survivor for every three to four infants allocated ECMO. The difference in survival applied irrespective of the primary diagnosis, disease severity, and type of referral centre. The benefit of ECMO was also found for the primary outcome measure of death or disability at 1 year (among 124 children enrolled before December, 1994). One child in each group has severe disability (overall Griffiths' developmental quotient <50, or untestable), and 16 (ten ECMO, six conventional management) have impairments with a lesser degree of disability (UK ECMO Trial Group, 1996).

Yorkhill ECLS Programme

Since starting the ECLS programme at Yorkhill in 1992, we have undertaken almost 410 ECLS runs. Initially, the service was for neonatal respiratory conditions but has since expanded to include paediatric respiratory conditions and later cardiac ECLS. We have also supported a couple of older children and

adults in exceptional circumstances. Results are encouraging and are comparable with other major centres providing ECLS. Overall neonatal respiratory survival is 78%, paediatric respiratory is 72% and cardiac survival is 51%.

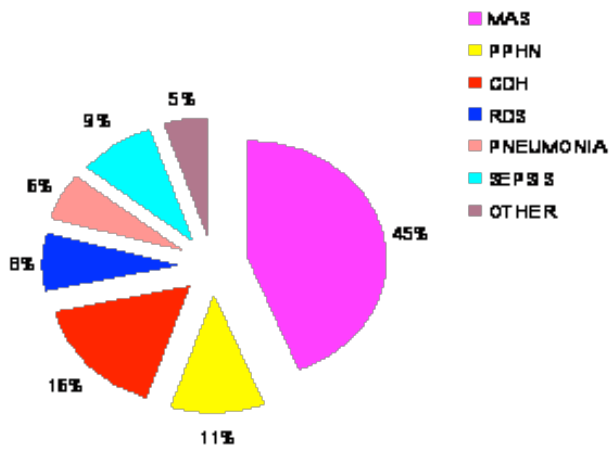


Figure 8 RHSC Neonatal cases by diagnosis

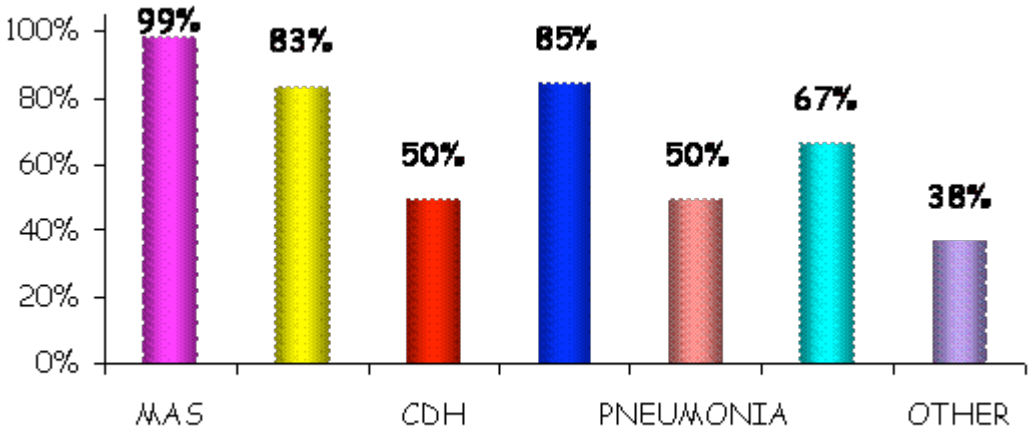


Figure 9 RHSC Neonatal survival by diagnosis

Aim of Neonatal ECMO

Selecting patients for ECLS and timing of treatment are two difficult aspects. Due to the invasive nature of ECLS and its significant associated risks, ECLS has always been reserved to treat only those neonates in whom other less invasive and dangerous therapies have failed. It was originally reserved for patients who predictive mortality was 80% (Cornish, 1995). However as expertise has improved and other patients have been treated, selection criteria have broadened to include patients for whom the benefits of ECLS would outweigh its risks.

Criteria for entry into the United Kingdom Collaborative ECLS Trial:

- Weight > 2000g and >34 weeks gestation
- Not more than 7 days of high pressure ventilation
- Less than 28 days old
- Unresponsive to maximal conventional management
- OI > 40 or $PaCO_2$ > 90 mmHg
- No contraindication to ECLS support:
- Congenital/acquired CNS abnormality
- Irreversible cardiopulmonary disease
- Period of asystole (outside post-delivery period)
- Proven NEC
- No reason to stop conventional care:
    - Major congenital / chromosomal anomalies
    - Severe encephalopathy

Neonatal ECLS Criteria 2010

Criteria **relatively** unchanged from UK Trial, but:

- We encourage early consultation and transfer of patients who are at risk of needing ECLS
- OI <25
- All CDH
- Smaller and younger babies now considered
- Some chromosomal defects are not a contraindication (e.g. Down's Syndrome)

Neonatal Respiratory Physiology

Development of Lung Maturity:

By 24 weeks gestation gas exchange is theoretically possible. From this time to term, further terminal branching of the airways occurs with the development of saccules which become the alveolar ducts. Alveoli begin to appear at 32 weeks. These continue to develop until 2 years of age. Surfactant is secreted by type II alveolar cells in the lungs. It is composed of several phospholipids which lower the surface tension forces of the alveolar walls. This is important for the initial opening of the alveoli and the prevention of subsequent atelectasis. The major constituent of surfactant is lecithin phosphatidylcholine. It comprises 50-75% of all the phospholipids. It first appears in significant quantities at 22-24 weeks gestation and continues to increase with gestation. By 35 weeks the ratio of lecithin to sphingomyelin is 2:1 which signifies pulmonary maturity. The presence of another phospholipid, phosphatidyl glycerol (PG), is also an indicator of lung maturity. Production begins just before 34 weeks gestation.

Foetal Lungs:

The lungs of the fetus are filled with fetal lung fluid. This fluid is produced in the periphery of the lungs and migrates out of the airways into the amnion where it becomes a constituent of the amniotic fluid. Fetal lung fluid functions to maintain the patency of the airways allowing formation of the developing airspaces. At birth, fetal lung fluid is eliminated from the airways by two mechanisms. Vaginal delivery compresses the chest and forces fluid from the lungs. This fluid is then reabsorbed by the lymphatic system after delivery. To initiate respiration the following steps must occur:

- Entry of air must overcome opposing forces (60 cm H20 pressure may be needed for the first breath)

- Some air must remain in the lungs at the end of expiration so that the lungs do not collapse, establishing a functional residual capacity (FRC)
- Pulmonary vascular resistance must fall to allow blood into the lungs
- With the removal of the placenta, a redistribution of the cardiac output must occur

Caesarean section reduces the efficiency of fetal lung fluid clearance increasing the risk of Transient Tachypnoea of the Newborn (TTN).

Diseases which increase fetal lung maturity:
Maternal:

- Toxaemia
- Hypertensive renal disease
- Hypertensive cardiovascular disease
- Sickle cell disease
- Narcotic addiction
- Diabetes Group

Fetal:

- Premature rupture of membranes disease
- Growth retardation

Diseases which delay fetal lung maturity
Maternal:

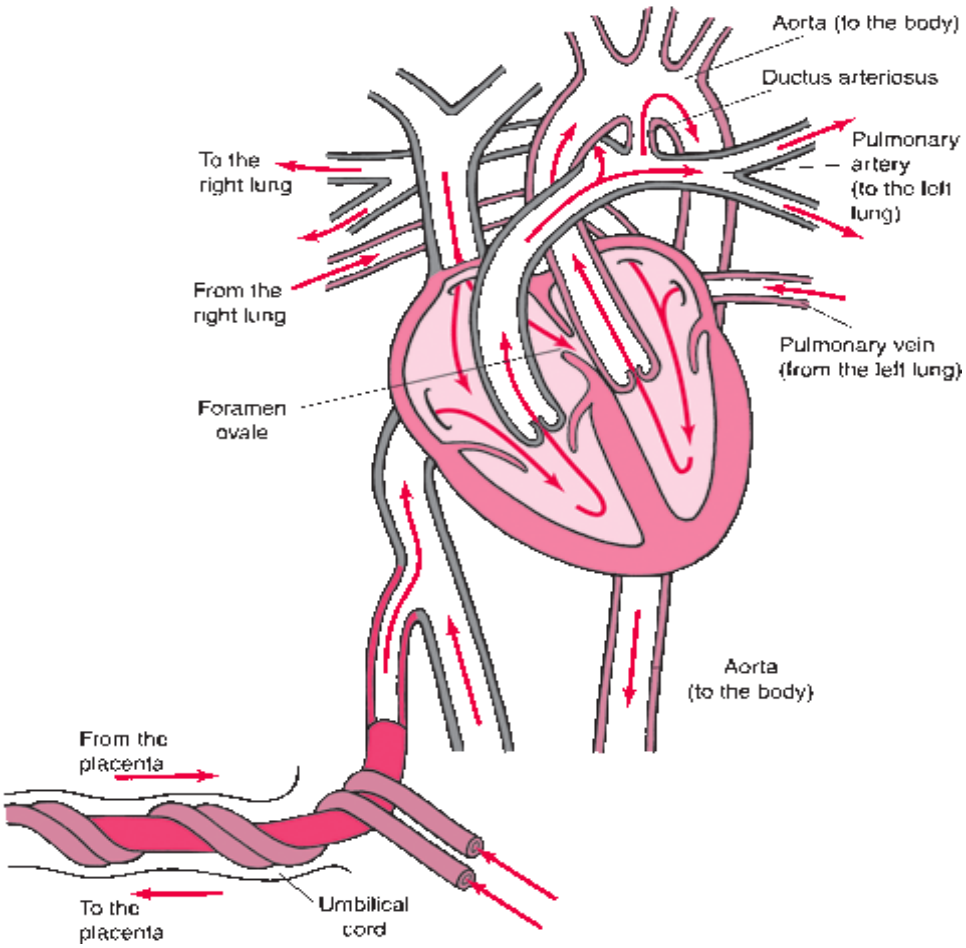
- Diabetes group
- Chronic glomerulonephritis

Foetal:

- Hydrops foetalis
- Smaller of identical twins

Foetal Circulation
Gas and nutrient exchange (replenishment) in the fetus is accomplished by the placenta. The newly replenished blood from the placenta returns to the fetus via the umbilical vein. Fetal circulation is necessarily different from adult circulation to facilitate movement of blood from the placenta to the organ systems of the fetus and back again. In the fetus, external respiration of the fetus occurs in the placenta. Therefore, only the portion of cardiac output required to sustain the metabolic demands of the developing lung is required to perfuse the pulmonary circulation. This constitutes about 10% of the total cardiac output.

Blood flow through the pulmonary vasculature is reduced by hypoxic pulmonary vasoconstriction (HPV) and two vascular shunts; the *foramen ovale* and *ductus arteriosus*. HPV increases pulmonary vascular resistance and the vascular shunts provide less resistant pathways for blood to flow. The *foramen ovale* allows blood to flow from the right atrium (higher pressure) to the left atrium (lower pressure). The *ductus arteriosus* allows blood to flow from the pulmonary artery (higher pressure) to the aorta (lower pressure).



Foetal circulation is accomplished as outline below:

Blood replete with nutrients and gas flows from the placenta to the umbilical vein toward the fetus with:

- 50% going directly into the portal circulation (liver) and 50% directly through the ductus venosus to the inferior vena cava and on to the right atrium.

At the right atrium blood flow has two directional opportunities

- Either blood flows from the right atrium through the foramen ovale to the left atrium, to the left ventricle, out to the aorta and body, to the umbilical arteries and back to the placenta (most blood flow will follow this route).
- Or blood flows from the right ventricle to the pulmonary artery where blood flow divides again:
- Either blood flows through the ductus arteriosus to the aorta out to the body to the umbilical arteries and back to the placenta.
- Or (about 10%) of the blood flow will go through the lungs via pulmonary circulation, to the left atrium to the left ventricle out to the body to the umbilical arteries and back to the placenta.

Indications for neonatal ECMO

Respiratory Distress Syndrome:

Respiratory Distress Syndrome (RDS) is due to a relative deficiency of surfactant most commonly occurring in premature infants. The lack of surfactant causes atelectasis and subsequent respiratory failure. While it is most common in premature infants it is possible for term infants to be affected in certain situations such as an infant of a diabetic mother or in cases of elective caesarean section (especially before 39 weeks). In other situations surfactant can be inactivated by meconium, infection or asphyxia. It is possible to stimulate surfactant production using maternal steroid injection (usually betamethasone) 12 to 48 hours before birth. RDS is a complication in about 1% pregnancies. Approximately 50% of the neonates born at 26-28 weeks of gestation develop RDS, whereas <30% of premature neonates born at 30 to 30-31 weeks develop RDS.

Enormous strides have been made in our understanding of the pathophysiology and management of these infants, leading to improvements in morbidity and mortality.
Advances include:

1. The use of antenatal steroids to enhance pulmonary maturity
2. Appropriate resuscitation facilitated by placental transfusion and immediate use of continuous positive airway pressure (CPAP) for alveolar recruitment.
3. Early administration of surfactant
4. Using gentle modes of ventilation to minimize damage to the immature lungs.

<u>Pathophysiology of RDS</u>

The cause of RDS is relative deficiency of surfactant, which decreases lung compliance and functional residual capacity with increased dead space.
The resulting large ventilation-perfusion (V/Q) mismatch and right-to-left shunt may involve as much as 80% of the cardiac output.

- Inadequate amount of active surfactant
- Alveolar collapse
- Increased work of breathing
- Respiratory failure
- V/Q mismatching hypoxia and hypercapnia
- Hypoxia and acidosis cause increased pulmonary vascular resistance and increased V/Q mismatching
- Capillary leak of proteinaceous material (Hyaline Membranes)

Progressive signs of respiratory distress are noted soon after birth and include the following:

- Tachypneoa
- Expiratory grunting (from partial closure of glottis)
- Subcostal and intercostal retractions
- Cyanosis
- Nasal flaring

In near-term infants the increased pulmonary vascular resistance causes right to left shunting through the foramen ovale and/or ductus arteriosus

Several diagnoses may coexist and complicate the course of RDS, including the following:

- Pneumonia, often secondary to group B beta-haemolytic streptococci.

- Metabolic problems (eg, hypothermia, hypoglycaemia).
- Haematological problems (eg, anaemia, polycythemia).
- Transient tachypnea of the newborn which usually occurs in term or near-term neonates, often after caesarean delivery.
- Aspiration syndromes which may result from aspiration of amniotic fluid, blood, or meconium.
- Pulmonary air leaks (eg, pneumothorax, interstitial emphysema, pneumomediastinum, pneumopericardium)
- Congenital anomalies of the lungs (eg, diaphragmatic hernia).

Management of RDS:

Exogenous surfactants are available to treat RDS. Curosurf (porcine surfactant) and Survanta (bovine surfactant) are the two natural surfactants available. Exosurf is an artificial surfactant which is slower to act due to the lack of surfactant proteins. There is debate as to whether surfactant should be given prophylactically to all at risk infants or reserved as rescue therapy for neonates requiring ventilation.

Increased inspired oxygen should be given to all infants who are hypoxaemic. If the oxygen requirement is climbing, the pCO2 is raised or there is marked respiratory distress nasal CPAP (continuous positive airway pressure) should be considered. Nasal CPAP recruits collapsed alveoli, and increases the functional residual capacity which increases lung compliance reversing the changes seen in RDS. If nasal CPAP fails then the child needs intubation and positive pressure ventilation.

Ventilator strategies:

PEEP (positive end expiratory pressure) will help to keep the alveoli from collapsing at the end of inspiration. A setting of 4-5 cmH2O is usual. Lower settings may not be effective while high settings can interfere with venous return to the heart. Peak pressures should be kept as low as possible to decrease the risk of lung injury due to excessive stretch of the alveoli. Inspiratory times should be set between 0.3 and 0.5 seconds. If the child is active then trigger ventilation is appropriate. 4 hourly blood gases (more frequent if unstable) should aim for CH 60-40 (pH 7.20-7.40), PaCO2 6-6.7 kPa, and PaO2 6.7-8.7 kPa.

Long-term complications:

Chronic lung disease (CLD) is a complication of any neonatal lung injury. It is defined as oxygen dependency at 28 days, clinical respiratory distress and an abnormal CXR. High inspired oxygen and lung damage from over-distension by ventilation are the most important causes. Infection, persistent ductus arteriosus and poor anti-oxidant systems are contributing factors. The most common underlying reason for the development of CLD is prematurity with RDS and therefore as smaller babies are rescued the incidence of CLD is increasing. As the child with CLD grows then lung growth will improve lung function. Bronchodilators, diuretics and steroids are other treatment options.

Meconium Aspiration Syndrome (MAS)

The passage of meconium can occur before birth secondary to acute or chronic hypoxia or stress. Infants can aspirate meconium in utero or at the time of delivery when the first breath occurs. The majority of meconium aspiration is mild but can be severe with the patient exhibiting marked respiratory distress. In the industrialized world, meconium in the amniotic fluid can be detected in 8-25% of all births after 34 weeks' gestation. Of those newborns with meconium-stained amniotic fluid, approximately 10% develop meconium aspiration syndrome.

Pathophysiology of MAS:

Two forms of lung pathology can occur:

- emphysema resulting from ball-valving with partial obstruction
- atelectasis resulting from total obstruction (Figure 1)

Both processes are usually present making the management of these infants very difficult.

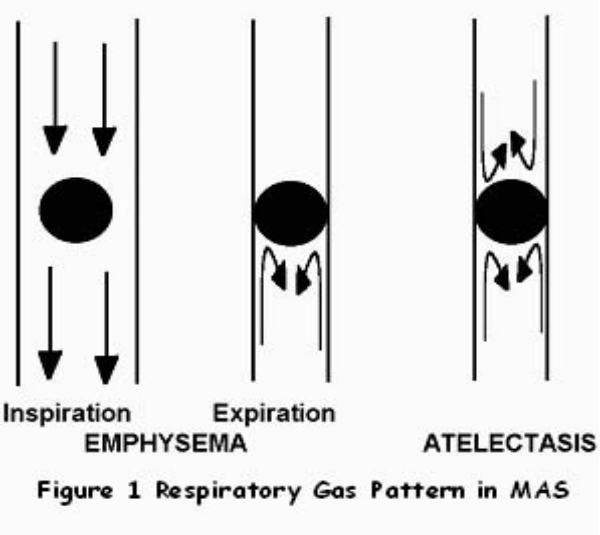


Figure 1 Respiratory Gas Pattern in MAS

Surfactant dysfunction:

Several constituents of meconium, especially the free fatty acids have a higher minimal surface tension than surfactant and strip it from the alveolar surface, resulting in diffuse atelectasis.

Chemical pneumonitis:

Enzymes, bile salts, and fats in meconium irritate the airways and parenchyma, causing a release of cytokines resulting in a diffuse pneumonia that may begin within a few hours of aspiration.

Persistent pulmonary hypertension of the newborn (PPHN):

All of these pulmonary effects can produce gross ventilation-perfusion (V/Q) mismatch. To complicate matters further, many infants with meconium aspiration syndrome (MAS) have primary or secondary persistent pulmonary hypertension of the newborn (PPHN) as a result of chronic in utero stress and thickening of the pulmonary vessels. Finally, although meconium is sterile, its presence in the air passages can predispose the infant to pulmonary infection.

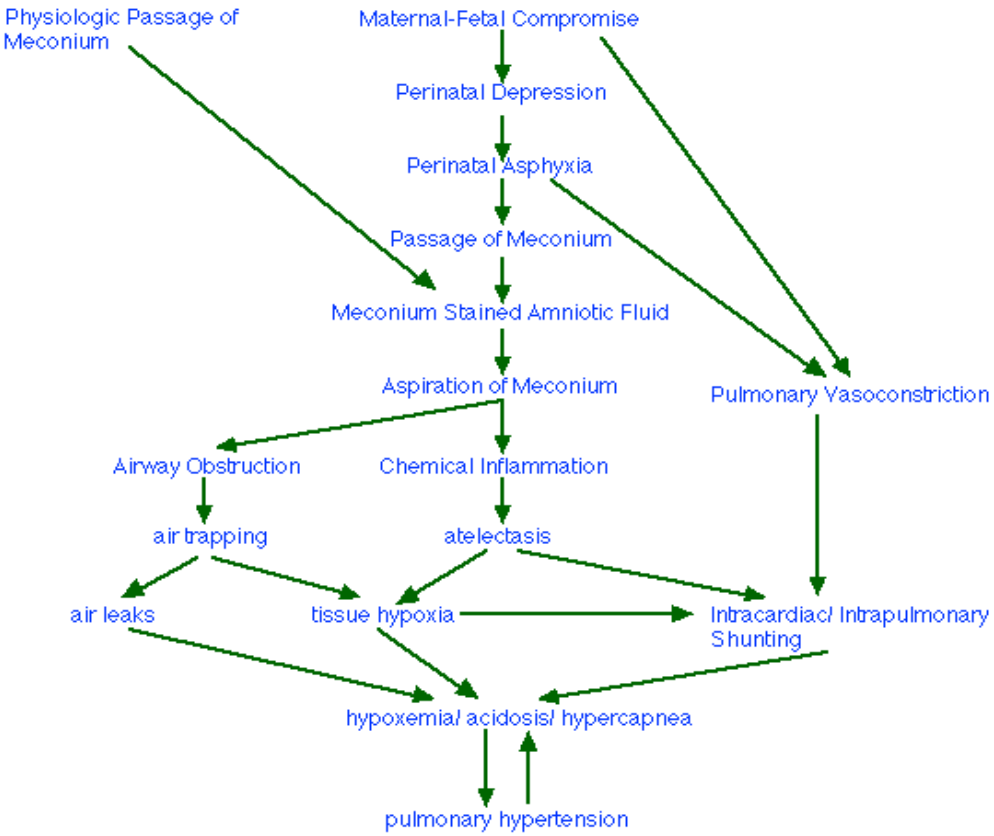


Figure 2: Pathophysiology of Meconium Aspiration Syndrome

The mortality rate for meconium aspiration syndrome resulting from severe parenchymal pulmonary disease and pulmonary hypertension is as high as 20% and should be considered a neonatal emergency with attendance of a paediatrician at the delivery to aspirate meconium from at risk infants.

Management of MAS:

- Nurse in high inspired oxygen
- Accept higher pCO_2 levels in effort to avoid intubation.
- If ventilated sedate or paralyse to reduce air leak
- Try to use low pressures to reduce air leak
- Use longer inspiratory times 0.5-0.7 seconds to overcome the high airway resistance
- Use slow rates (30-40) to avoid air trapping
- Surfactant therapy is now commonly used to replace displaced or inactivated surfactant.
- Inhaled nitric oxide is the pulmonary vasodilator of choice

Persistent Pulmonary Hypertension of the Newborn

PPHN occurs when there is a pathological increase in the pulmonary vascular resistance sufficient to divert blood away from the pulmonary circulation through anatomic shunts into the systemic circulation. Therefore a significant amount of de-oxygenated blood will mix on the systemic side causing significant hypoxaemia. PPHN can only occur when there is a decrease in the amount of blood flowing to the lungs, and is not simply a description of a raised pulmonary vascular resistance which will be present in a variety of diseases. For example in the setting of significant lung pathology such as MAS the pulmonary

vascular resistance will remain high. If there is then a change such as decreasing oxygenation or increasing acidosis the pulmonary vascular resistance may increase sufficiently to shunt blood away from the lungs causing decreased lung perfusion and the clinical development of PPHN.

Recent data suggest that the PPHN syndrome may occur as often as 2-6 cases per 1000 live births. PPHN is a frequent complicating factor in the term or near-term newborn with parenchymal lung disease, such as MAS or pneumonia. An increased incidence of PPHN is reported for mothers who use selective serotonin reuptake inhibitors (SSRIs) during the last half of their pregnancies. As recently as 15 years ago, the mortality rate reached 40%, and the prevalence of major neurologic disability was 15-60%.The introduction of extracorporeal membrane oxygenation (ECMO) and other new therapies has had a major effect on reducing the mortality rate associated with PPHN. In the UK Collaborative ECMO Trial the mortality rate decreased from approximately 60% in the group randomly assigned to receive conventional therapy to 30% for the group randomly assigned to receive ECMO. If all available therapies are used, the mortality rate appears to be less than 10%. However, the prevalence of major neurologic disabilities among surviving newborns remains approximately 15-20%.

Aetiology of PPHN:
PPHN is most commonly associated with 1 of 3 underlying aetiologies:

The first and most commonly encountered scenario is **acute pulmonary vasoconstriction** due to acute perinatal events, such as:

- Alveolar hypoxia secondary to parenchymal lung disease, such as respiratory distress syndrome (RDS) or pneumonia
- Hypoventilation resulting from asphyxia or other neurologic conditions
- Hypothermia
- Hypoglycaemia

The second cause, **idiopathic PPHN** is associated with a normal chest radiograph and no parenchymal lung disease. Newborns with idiopathic PPHN present with pure vascular disease. This syndrome typically results from:

- an abnormally remodelled pulmonary arterial bed, perhaps secondary to chronic stress in utero which causes prenatal hypertrophy of muscle layers in the pulmonary arterioles and primary failure of the pulmonary artery to relax
- Alveolar Capillary Dysplasia.
- Other potential associations include maternal use of NSAIDs, such as Ibuprofen or Naproxen, or SSRIs in the last half of pregnancy

The third cause of PPHN is **hypoplasia of the pulmonary vascular bed** caused by.

- Congenital diaphragmatic hernia, an abnormality of diaphragmatic development that allows the abdominal viscera to enter the chest and compress the lung.
- Maternal oligohydramnios
- Congenital cystic adenomatoid malformation, though PPHN is rarely associated with this malformation, even if the defect is large.

Some other causes of PPHN:

- Vasospasm and constriction secondary to hypoxia or acidosis
- Polycythemia, hypocalcaemia, and hypoglycaemia

• Specific mediators may be through the prostaglandin and/or leukotriene pathways

PPHN is characterised by:
• Right to left shunts through the PDA and/or PFO secondary to pulmonary hypertension
• Mixing of oxygenated and deoxygenated blood in the aorta and/or left atrium
Lung hypoperfusion due to the shunting

Doppler ultrasound can now diagnose directional shunts at both the PDA and PFO. Clinically one can look at pre and post ductal saturations. With a ductal shunt, only blood which is before the entrance of the PDA (pre-ductal) represents oxygenated blood from the lungs (right radial and temporal artery blood). A difference of 15 mmHg or greater in the pre and post-ductal blood gases agrees with a R to L shunt at the ductal level. Unfortunately if the shunt is only at the PFO and not the PDA, there will not be any difference in pre and post-ductal blood gas values because the mixing of oxygenated and deoxygenated blood occurs in the left atrium (Figure 3)

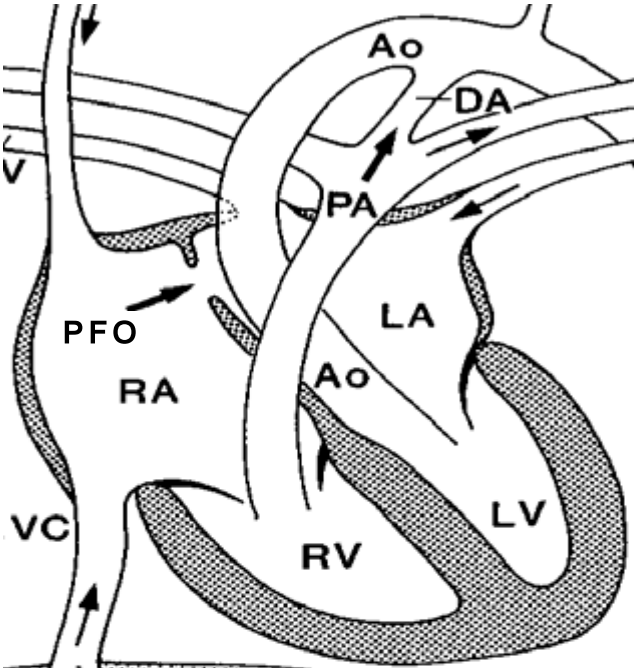


Figure 3 Potential cardiac Shunts

Management of PPHN:

- General considerations
  - An adequate circulating blood volume is necessary to maintain right ventricular filling and cardiac output; however, repeated bolus administration of crystalloid and colloid solutions does not provide additional benefit.
  - Inotropic support with Dopamine, Dobutamine, Adrenaline and/or Milrinone alone or in combination, is frequently helpful in maintaining adequate cardiac output and systemic blood pressure while avoiding excessive volume administration.
- Mechanical Ventilation

- Mechanical ventilation is usually needed to maintain adequate oxygenation, usually in conjunction with surfactant administration and/or high frequency oscillatory ventilation. Newborns with PPHN nearly always require sedation and or paralysis.
- The use of iNO with HFV creates particular problems for transport, and this should be considered, early discussion between referral and ECMOP centre should be encouraged

- Acidosis and alkalosis
  - Metabolic acidosis and respiratory acidosis require correction

Congenital Diaphragmatic Hernia (CDH)

Congenital herniation of the abdominal contents into the thoracic cavity causes serious respiratory compromise. Herniation occurs most often on the left side and represents a failure of the pleuroperitoneal canal to close completely during foetal development. CDH usually involves thoracic entry of the stomach, a large part of the intestines, and often the spleen and liver. These organs displace the heart and lungs, compromising cardiac output and pulmonary gas exchange. The lung on the affected side is hypoplastic, with varying degrees of hypoplasia on the contralateral side.

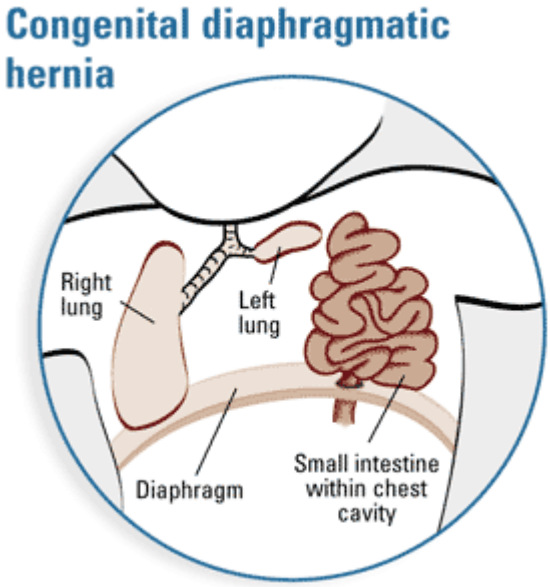


Figure 1: Left Congenital Diaphragmatic Hernia

Clinically, CDH most often presents as severe respiratory distress in a newborn infant. The physical examination is characterised by a cyanotic and dyspnoeic infant with a displaced apex beat and a scaphoid abdomen. If a significant portion of the intestines has herniated, bowel sounds may rarely be audible over the affected side of the thoracic cavity. The diagnosis is confirmed by X-ray. Pulmonary hypertension is often associated with infants who are born with CDH. It usually occurs pre-operatively, but may appear 24-36 hours after definitive surgical repair, especially if performed early. This period is often referred to as the "honeymoon period". The surgical strategy for CDH is to stabilise the patient pre-operatively, thus reducing the chance of developing pulmonary hypertension post-operatively. The aetiology of pulmonary hypertension in infants with CDH may be secondary to the hypoxaemia caused by the hypoplastic lung, but it also has been noted that the pulmonary artery branches are structurally abnormal with thickened muscular walls.

Management of CDH:

If diagnosed before birth then a combined approach to resuscitation should be adopted involving:

- Elective intubation, bag and mask ventilation should be avoided
- Passage of a large bore NGT connected to continuous suction to prevent bowel distension and further lung compression.
- Paralysis and adequate sedation should take place
- Conventional ventilation is the initial mode. A lung protective strategy is utilised with a maximum PIP of 25 cmH2O and permissive hypercapnia is allowed. HFOV (low mean pressure) should be considered if conventional ventilation is not successful, especially if CO2 is difficult to clear and because of the reduced risk of air leak.
- A meticulous attention to detail for subsequent medical care, including continuous monitoring of oxygenation, blood pressure, and perfusion. A minimal stimulation approach that reduces handling and invasive procedures, such as suctioning, is suggested.
- If PPHN exists then inhaled nitric oxide should be used.
- Adequate circulating volume is necessary to maintain right ventricular filling and cardiac output; Inotropic support may be helpful in maintaining adequate systemic blood pressure.
- In patients with severe PPHN, failing right atrium, pinching duct and poor systemic circulation a Prostin infusion should be considered to augment systemic perfusion.

After a variable period of adequate stabilisation the infant should be taken to surgery.
If the patient remains unstable, then ECLS should be considered pre-surgery with the possibility of using a perflourodecalin strategy for lung growth.

Sepsis

The incidence of culture-proven sepsis is approximately 2 per 1000 live births. The mortality rate in neonatal sepsis may be as high as 50% for infants who are not treated. Infection is a major cause of fatality during the first month of life, contributing to 13-15% of all neonatal deaths. Neonatal meningitis, a serious morbidity of neonatal sepsis, occurs in 2-4 cases per 10,000 live births and significantly contributes to the mortality rate in neonatal sepsis; it is responsible for 4% of all neonatal deaths. In the preterm infant, inflammatory mediators associated with neonatal sepsis may contribute to brain injury and poor neurodevelopment outcomes.

Neonatal sepsis may be categorized as early or late onset. Eighty-five percent of newborns with early-onset infection present within 24 hours, 5% present at 24-48 hours, and a smaller percentage of patients present between 48 hours and 6 days of life In the neonatal period the micro-organisms most commonly associated with early-onset infection include group B Streptococcus (GBS), Escherichia coli, Haemophilus influenzae, and Listeria monocytogenes. Transmission is most commonly via the transplacental route.

Late-onset sepsis syndrome occurs at 7-90 days of life and is acquired from the care giving environment. Organisms that have been implicated in causing late-onset sepsis syndrome include coagulase-negative staphylococci, Staphylococcus aureus, E coli, Klebsiella, Pseudomonas, Enterobacter, Candida, GBS, Serratia, Acinetobacter, and anaerobes. The infant's skin, respiratory tract, conjunctivae, GI tract, and umbilicus may become colonized from the environment, leading to the possibility of late-onset sepsis from invasive micro-organisms. Vectors for such colonization may include vascular or urinary catheters,

other indwelling lines, or contact from caregivers with bacterial colonization. Pneumonia is more common in early onset sepsis, whereas meningitis and bacteraemia are more common in late-onset sepsis.

Risk Factors:

- Maternal GBS status
- PROM
- Chorioamnionitis
- Low Apgar score (<6at 1 or 5 min)
- Maternal fever greater than 38°C
- Maternal urinary tract infection
- Poor prenatal care
- Poor maternal nutrition
- Maternal substance abuse
- Low birth weight
- Birth asphyxia
- Congenital anomalies
- Streptococcus or E. Coli can cause severe sepsis. The clinical picture is of respiratory distress, with hypotension, coagulopathy, renal impairment and poor oxygen delivery & utilisation. In extreme cases PPHN can develop.

Signs of Neonatal sepsis:

- Respiratory: Tachypneoa, irregular respirations, moderate retracting, apnea, cyanosis, and grunting may be observed. Neonates with intrauterine pneumonia may also be critically ill at birth and require high levels of ventilatory support. The chest radiograph may depict bilateral consolidation or pleural effusions.
- Cardiac signs: In overwhelming sepsis, an initial early phase characterized by pulmonary hypertension, decreased cardiac output, and hypoxemia may occur.
- The early phase of pulmonary hypertension is followed by further progressive decreases in cardiac output with bradycardia and systemic hypotension. The infant manifests overt shock with pallor, poor capillary perfusion, and oedema. These late signs of shock are indicative of severe compromise and are highly associated with mortality.
- Metabolic signs: Hypoglycaemia, hyperglycaemia, metabolic acidosis, and jaundice all are metabolic signs that commonly accompany neonatal sepsis syndrome. The infant has an increased glucose requirement because of sepsis. Metabolic acidosis results due to a conversion to anaerobic metabolism with the production of lactic acid.
- Renal signs: Decreased urine output, acute renal failure and increasing oedema can all result from sepsis.
- Coagulation signs: Coagulopathy, platelet abnormalities and DIC
- Neurological signs: Ventriculitis, Meningitis, multiloculated areas within the brain similar to abscesses, seizure activity, and infracted areas which can be haemorrhagic.

Treatment of neonatal sepsis

- Antibiotic - penicillin/aminoglycoside for early onset, vanc/aminoglycoside for late.
- Ventilation
- Maintain blood pressure with adequate intravascular volume and inotropes
- Maintain renal blood flow
- Correct coagulation
- Immunoglobulin infusion

Neonatal respiratory ECMO patient management

Cannulation

For most infants cannulation will be with a wire reinforced OriGen double-lumen cannula via a percutaneous route for veno-venous ECMO support. This necessitates cardiology ECHO guidance to appropriately position the cannula. The tip of the cannula should rest in the mid-right atrium with the "arterial" connector pointing anteriorly (usually towards the ear). This directs the oxygenated blood preferentially towards the tricuspid valve. Chest x-rays undertaken on a daily basis to look for position +/- migration of all V-V cannulae. Size constraints may make VV ECLS impossible. Presently, the smallest DLVV cannula available is 12F. In this situation the patient will require VA cannulation with Biomedicus cannulae which start in size from 8F. Patients most likely to require VA cannulation for vessel size include infants with diaphragmatic hernia and infants <2.5kg.

Inotropic support

Neonates cannulated for V-V ECMO will commonly be on significant inotropic support pre-cannulation. This can usually be weaned down/off within the first 24hrs hours of cannulation as the cardiac function improves with delivery of oxygenated blood from the ECMO circuit. Patients requiring ECMO support for sepsis will sometimes require a longer period of inotropic support but usually at greatly reduced levels. Those patients supported with V-A ECMO due to vessel size may become hypertensive once ECMO flows are commenced requiring inotropes to be rapidly weaned. An inotrope weaning chart should be used during this period.

Inhaled Nitric Oxide (iNO)

Most neonates requiring ECMO support will have significant pulmonary hypertension pre-cannulation and require iNO therapy. Following cannulation and good delivery of oxygen to the pulmonary bed this will usually improve fairly quickly. For most patients iNO can be weaned over a 24hr period with a careful watch on pre and post ductal saturations. Signs of ongoing PPHN, especially in patients with diaphragmatic hernia, may require more cautious weaning. ECHO may assist in determining the most appropriate weaning period for those patients. At cannulation, severe PPHN with poor right sided function, some patients may require a Prostin infusion to keep the arterial duct open and ensure adequate systemic perfusion (via the duct). As PA pressure falls and blood flow becomes left to right problems with systemic hypotension and perfusion may occur necessitating early reduction or discontinuation of the infusion. Again, ECHO may aid in recognising this.

Rest settings

It is essential that the lungs are rested during ECMO undertaken for respiratory support. This means avoiding or exacerbating ventilator induced lung injury. A 'lung rest strategy' is adopted. Typical settings would be FiO_2 of 0.3-0.4, Ti 0.5s, PIP 15-17, PEEP 5-8 and rate of 10-15bpm. CPAP via an endotracheal tube alone can also be used to avoid any inadvertent barotrauma or volutrauma from positive pressure ventilation in those patients with significant air leak. It is essential that endotracheal toileting is undertaken using minimal inspiration pressures and tidal volume with the T-piece to limit secondary lung injury at this time. A manometer can assist in monitoring inspiratory pressures.

Once there is evidence of improvement in lung compliance and weaning is commenced, typically at around 30% ECMO flows, the ventilator pressures may be gently increased to aid weaning of the blender and sweep gases. More sophisticated ventilatory recruitment strategies in tandem with physiotherapy and/or

flexible bronchoscopy may be required for those patients with diaphragmatic hernia, continued collapse or difficulty in weaning.

Paralysis and sedation

Once cannulated, neonates on ECLS can usually be successfully managed with light sedation, typically a Morphine infusion +/- a benzodiazepine. The need for continuing paralysis is usually reviewed within 24hrs of cannulation once stability and cannula position are established. Continued paralysis should be reserved for the rare ECLS patient such as those with significant cannula problems, inadequate oxygenation at maximum VV support, or a newborn with CDH that has significant intestinal distension from swallowed air. Adequate sedation is given in order to prevent excessive movement and possible cannula displacement but allow for neurological assessment, mobilisation of fluid and interaction with parents.

Physiotherapy

Physiotherapy is important for many neonatal patients early in the ECMO run. This is especially critical for those patients with MAS or congenital pneumonia. Referral should be considered as soon as the patient has stabilised post cannulation, those patients with cannula issues or bleeding should first be discussed with the consultant. Careful assessment of ET tube patency will highlight those patients requiring replacement of the ET tube.

(See physiotherapy section in patient management)

Diaphragmatic hernia, ECMO and PFC

ECMO runs for Diaphragmatic hernia tend to be longer and more complex than other neonatal runs. They can be categorised into two groups:

A. Those who fail conventional therapy but appear to have reasonable lung volumes and significant pulmonary hypertension and

B. Those that have hypoplastic lungs +/- significant pulmonary hypertension.

The first group present sometimes unexpectedly in the delivery room with suboptimal management at birth or may have a period of relative stability before a precipitate issue such as sepsis causes periods of pulmonary hypertensive crisis. Theses patients are placed on ECMO in order to manage the PPHN with an expectation of weaning prior to hernia repair. Their reasonable lung volumes make PFC unnecessary. The second group have small lung volumes but some evidence of reasonable gas exchange. They are placed on ECMO to manage PPHN and to facilitate a 'lung growth strategy'. PFC is instilled into the lungs and this is followed by early repair on ECMO. The PFC is continued after repair on ECMO to "grow" the lungs and to gently distend and recruit alveoli.

Overall survival for ECMO in CDH is about 52% (ELSO Registry). Patients with CDH may be managed on VVDL or VA ECMO. Vessel size can be small in this patient group however resulting in an increase in VA cannulation.

Data from the CDH study group shows that about 54% of those infants that go onto ECMO are repaired on ECMO and 30% following ECMO. Around 16% are never repaired and all expired.

Management during surgery

Once a date has been arranged for surgery the ECMO specialist should liaise with blood bank to ensure that sufficient blood products are available during and post surgery. As a minimum:

- 2 Prime
- 1 large unit
- 4 Paedipaks
- 4 Platelets should be available.

Prior to commencement of surgery a recent FBC, Coag, TEG and U/E should be obtained. Coagulation factors should be optimised to ensure Fib is > 2.0, and the Platelet parameter increased to 150,000. Two hrs before surgery ACT parameters should be tightened to 180-200 (Ensure flows remain high). Surgeon may reduce heparin or run Heparin free depending on clinical requirements. ACT's should be carried out every 30mins thereafter until ACT's have stabilised post surgery.

Drugs and Volume Immediately before surgery a Paedipak of blood and PPS 4.5% should be drawn up at the bed side. A large unit of PRBC should be ordered from blood bank in the cold box, checked on receipt (form is in side pocket) and only opened if required in emergency. This can be kept at bedside for upto 3 hrs and should be returned to blood bank unopened within this time frame if unused. Two doses calculated by weight of Fentanyl, Vecuronium, Atropine and Adrenaline should be drawn up with several saline flushes. Iv access should be secured during draping (Clear drapes are available).

Ventilation and Nitric moderate ventilation settings should be used and patients not currently on Nitric therapy should have a delivery unit checked and connected to ventilator circuit prior to surgery. The Anaesthetic team will determine ventilatory settings intraoperatively and liaise with ECMO specialists

ECMO settings high flows should be maintained during and immediately following surgery as ACT parameters will be tightened. The blender should be increased to 100% and patient, Pump and Venous sat obtained unless done in previous two hours. The Bridge is an area of blood stasis especially if ACT's are low therefore it should be accessible during surgery and flashed every 30mins during and after the procedure.

Surgical Procedure during surgery manipulation of the liver may sometimes result in reduced venous return resulting in an increase in –ve pressures, sometimes with a decrease in flows. This can usually be managed with a little volume and alerting the surgeon. Cardiac arrhythmias can also occur. For patients on VA ECMO these are usually self limiting and can be managed by increasing the flows if required and/or small amounts of volume. Arrhythmias for patients on VV ECMO need more aggressive management if they result in haemodynamic instability. The Anaesthetic team will assume overall responsibility and liaise with ECMO specialists

Immediately Post Operation a repeat FBC, Coag and TEG should be obtained. Chest drain losses should be closely monitored and any sudden increase/decrease discussed. ACT parameters should be kept tight for 4-6hrs and reassessed in light of chest drain losses. A chest CxR should be obtained.

Bleeding is a potential problem following surgery on ECMO. Signs of bleeding can include increasing chest drain losses, reduced perfusion, tachycardia, reducing venous sats (VA ECMO), increasing venous sats (VV ECMO), uneven appearance of the chest, mottling of skin, increasingly –ve inlet pressure, reduced flows, reduced MAP or incremental reduction in HB which is unresponsive to top up transfusion. Any suspicion of bleeding should be discussed with the surgeon and ECMO physician as a matter of urgency.

Weaning from neonatal respiratory VV ECMO

Neonatal ECMO runs are often relatively short with a mean of 5 days excluding those patients with CDH who have a mean run time of 18 days (the longest surviving run of 26 days in 2009/10). If one attempts to wean too early then it may result in further damage to the recovering lungs especially in those patients with significant air leak pre cannulation. However the incidence of complications associated with

ECMO including infection and haemorrhage increase with extended runs. Prior to weaning from V-V ECMO, ensure adequate positioning of the endotracheal tube and an acceptable chest x-ray appearance.

Weaning from V-V ECMO in the neonatal patient is usually approached differently from that of weaning from V-A ECMO. **Use the checklist in the guideline section.**

The ECMO circuit makes no contribution to cardiac output and is preload/afterload neutral as blood is drained and returns to the right heart. For this reason there is **no need to wean the flows to low levels** as this requires an increase in anticoagulation and therefore potentially a greater risk of haemorrhagic complications. **VV-ECMO should be weaned via the blender and sweep gases**. Remember that ventilation may need to be increased as ECMO gas flow/FiO2 is weaned. Once the ECMO blender is at 30%, the ventilator FiO_2 may be increased to 40%. The sweep O_2 flow should be ≤0.4 litre/min prior to capping off gas flow. There is also no need to place the patient onto the bridge and anticoagulate the patient and the circuit as in VA ECMO.

To trial the patient off VV ECMO, the membrane is capped off by disconnecting the green gas line and creating a closed circuit onto the membrane. ECMO flows continue at previous levels. Assessing suitability for decannulation involves closely monitoring the patient's gas exchange on the arterial blood gas and ECMO circuit $mvSaO_2$. If the patient demonstrates persistent respiratory compromise (rising pCO_2 and falling SaO_2/PaO_2, despite optimised respiratory support, then recommence or increase the gas flow to previous levels. It may be necessary to sigh the oxygenator.

What to do in the event of a "wet" oxygenator (see guideline 19)

- Increase sweep gas to maximum for size of oxygenator for 2-3 minutes with blender at 100%
  - Medos 0800 Max sweeo gas flow 2.0L/min
  - Medos 2400 5.0L/min
  - Medos 7000 10.0L/min
- Repeat blood gas after 3 minutes at this gas flow
- Observe circuit closely for any bubbles
- If no improvement call Perfusionist and ECLS Physician

It is best to trial off V-V ECMO by capping off the gas flow first thing in the morning to allow decannulation in the early afternoon. The surgeon should be informed of the trial off to ensure workload can be adjusted. After several hours of no gas flow to the oxygenator and demonstration of adequate respiratory support from the ventilator, the patient can be safely decannulated. This is usually done by withdrawing the double lumen cannula and ensuring adequate pressure on the insertion site. Adequate platelet levels should be present prior to decannulation to aid local clotting of the cannulation site. (See Decannulation section) A chest x-ray should be obtained post decannulation and blood bank informed so blood products can be dereserved.

It is important to ensure that the patient's cares are undertaken prior to decannulation and that there is no unnecessary movement of the head and neck following decannulation which may disturb the clotted cannulation site.

Paediatric respiratory ECMO

Introduction

Respiratory failure is a common reason for admission to PICU/HDU and usually responds well to non-invasive or invasive respiratory support. Acute respiratory distress syndrome (ARDS) in the paediatric age group is most commonly seen secondary to sepsis, pneumonia, trauma or aspiration. In a review of nine PICU's in the USA 7.6% of admissions were diagnosed with ARDS with an associated mortality of 4.3% *(Randolph AG et al. Am J Respir Crit Care Med 2003, 167:1334–1340).*

Pediatric Respiratory Runs by Diagnosis

	Total Runs	Avg Run Time	Longest Run Time	Survived	% Survived
Viral pneumonia	22	344	745	16	73%
Bacterial pneumonia	8	254	584	5	63%
Pneumocystis pneumonia	1	377	377	0	0%
Aspiration pneumonia	5	333	910	3	60%
ARDS, postop/trauma	1	519	519	0	0%
ARDS, not postop/trauma	8	201	296	6	75%
Acute resp failure, non-ARDS	7	294	492	4	57%
Other	11	377	937	6	55%

Run time in hours. Survived = survival to discharge or transfer based on number of runs

Table 1 ELSO RHSC, Yorkhill data, January 2010.

Pediatric Respiratory Runs by Diagnosis

	Total Runs	Avg Run Time	Longest Run Time	Survived	% Survived
Viral pneumonia	938	322	1372	593	63%
Bacterial pneumonia	500	275	1332	285	57%
Pneumocystis pneumonia	30	371	1144	15	50%
Aspiration pneumonia	200	268	2437	132	66%
ARDS, postop/trauma	109	250	903	68	62%
ARDS, not postop/trauma	384	301	1987	204	53%
Acute resp failure, non-ARDS	766	247	1483	389	51%
Other	1,527	209	2239	778	51%

Run time in hours. Survived = survival to discharge or transfer based on number of runs

Table 2 ELSO combined international data, January 2010.

Acute lung injury (ALI) has been defined by the Consensus Conference on ARDS as the presence of bilateral infiltrates on chest x-ray, absence of left atrial hypertension (pulmonary artery occlusion pressure < 18 mm Hg) and impaired oxygenation (PaO2/FiO2 < 300) with ARDS being diagnosed if the PaO2/FiO2 < 200
(Bernard GR et al, Intensive Care Med 1994, 20:225–232).

Severe respiratory failure in the paediatric age group is commonly managed by high frequency oscillation ventilation (HFOV) adopting a permissive hypoxia and hypercarbia lung protective strategy. The targeted use of inhaled nitric oxide to overcome ventilation perfusion mismatch or secondary pulmonary hypertension may also be of benefit though a recent Cochrane review did not back its use in this diagnosis (*The Cochrane Database of Systematic Reviews 2003, Issue 1. Art. No.: CD002787).* The place for surfactant in these patients is debatable although a recent study showed an improvement in the oxygenation index and a reduction in mortality in those given Calfactant, an exogenous surfactant, in children with acute lung injury *(Wilson et al JAMA. 2005;293:470-476).* The judicious use of PEEP, prone positioning and flexible bronchoscopy form other aspects of the

treatment algorithm for these children. Corticosteroids are commonly used in our centre starting between days 7 and 14 of ventilatory support with an initial 2mg/kg dose of Methylprednisolone followed by 0.5mg/kg every 6 hours with a long tapered wean from day 14 (see clinical guideline on CIS).

Mortality rates of 4.3% associated with paediatric ARDS are low compared to the level of 22% seen in adult intensive care *(The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000, 342:1301–1308).* This means that is difficult to undertake studies in children to delineate the survival benefit of ECMO in the paediatric ARDS population. The recent CESAR trial attempted to highlight the place for ECMO in the adult population with ARDS showing a 6 month mortality or severe neurodisability incidence of 37% in those managed with ECMO compared to those managed with conventional therapy who had a mortality or severe neurodisability incidence of 53% (Peek G et al. Lancet; 374: 1351 – 1363). The study was confounded by the fact that 17 patients referred for ECMO but supported conventionally had an associated 6 mortality or severe neurodisability of 18%. Those patient in the ECMO arm also had significantly greater numbers receiving steroids (84% vs 64%, p<0.0001) and significantly more being managed with a low volume, low pressure lung protective ventilation strategy (93% vs 70%, p<0.0001).

Reported mortality rates for children with ARDS supported with ECMO vary between 30 and 50% *(Pettignano R et al. Pediatr Crit Care Med 2003, 4:291–298. Langham MR et al. Ann Surg 2003, 237:766–772. Swaniker F et al. J Pediatr Surg 2000, 35:197–202).* ELSO have reported an overall hospital mortality of 44% in the paediatric respiratory ECMO group between 1986 and 2009 in their annual report in January 2010. For this reason ECMO may be utilised in paediatric ARDS patients with whom the lung disease is reversible and who have not been exposed to injurious ventilatory pressures for long periods of time, most ECMO centres would assess this as 10-12 days. The final decision has to be a clinical one made by the treating ECMO physician and their colleagues.

Aim of paediatric respiratory ECMO

The role of ECMO in paediatric ARDS patients is to allow lung and heart rest from injurious ventilator settings and significant inotropic support to enable the underlying process to resolve and the patient to return to a position where organ support is non-injurious. Veno-venous ECMO is the most common modality used in this setting and will often be possible even if there is modest inotrope requirement. Veno-arterial ECMO may be required if there is significant cardiovascular instability associated with the illness. Patients with ARDS often require long ECMO runs, until at least partial resolution of the lung injury. The average run length in the ELSO database for paediatric respiratory ECMO is 11 days which is in keeping with our findings. This makes the adherence to strict asepsis, optimised anticoagulation, optimal nutrition and lung rest essential in the minimisation of the secondary complications of intensive care.

Indications for paediatric respiratory ECMO

- Acute reversible lung injury
  - when there is an absence of: Long periods of injurious ventilation
    Significant neurological insult
    Uncontrolled bleeding

Paediatric respiratory ECMO patient management

Cannulation

Cannulation in these patients is commonly undertaken by a percutaneous **veno-venous** route. This necessitates cardiology echo support to appropriately position the cannula. This is even more important in the children where a double lumen cannula is utilised, especially the Avalon cannula where the tip should lie in the IVC with a lower drainage port draining inferiorly and an upper drainage port in the SVC with the ejection port being directed towards the tricuspid valve. This cannula works well to minimise recirculation, a common problem in V-V ECMO. However as the cannula is wire wound to increase its strength there is an increased risk of atrial perforation which has been found by some centres. This should always be borne in mind and chest x-rays undertaken on a daily basis to look for migration or kinking or the wire wound V-V cannulae. The wire wound cannulae minimise the recirculation therefore the need to increase the ECMO flows up to 120-150ml/kg/min to compensate for this is rarely necessary as was often required in non-wire wound cannulated patients supported on the roller pump. ECMO flows of 60% cardiac output should provide adequate support for most patients.

Veno-arterial cannulation may be required if there is significant inotropic support, often seen in those patients with septic shock with underlying ARDS or if inadequate oxygenation is possible with V-V ECMO. Percutaneous cannulation of the carotid artery and jugular vein is undertaken under echo guidance to site the cannulae effectively. The cannulae sited should be large enough to cope with a high flow ECMO strategy (up to 200ml/kg/min) to enable luxuriant oxygen delivery to the end organs and allow cessation of vasopressor support (Nor-adrenaline and vasopressin) and initiation of vasodilation with sodium nitroprusside +/- phenoxybenzamine. Inotropic support (Adrenaline, dopamine, milrinone) should be weaned rapidly in these patients to minimise myocardial work and therefore damage. As with the cardiac patient on V-A ECMO it essential to ensure that the left atrium and left ventricle do not become over distended and therefore compromise the oxygen supply to the myocardium. This is monitored with cardiac echocardiography and may necessitate an atrial septostomy being created to off-load the blood from the left heart from the left atrium to the right atrium.

Patients requiring ECMO support for respiratory pathologies are often supported on HFOV making percutaneous cannulation difficult because of the patient vibration. It is often easier to support the patient on T-piece ventilation whilst cannulation occurs. One should always ensure there is a clear view of the ETT during cannulation with $_{ET}CO2$ is being used (if not on HFOV) and that the ETT position is appropriate prior to cannulation given the fact that the head is extended for cannulation of the jugular vein.

In larger children it may be necessary to site a second drainage cannula if there is insufficient venous drainage.

Patients supported on V-V ECMO will commonly have SaO2 in the high 70's to high 80% because of mixing of venous blood with the oxygenate blood from the ECMO circuit. This will often equate to a PaO2 of 6-9kPa. As long as there is no evidence of developing end organ dysfunction this is adequate oxygen delivery.

Inotropic support

Patients cannulated for V-V ECMO will commonly be on inotropic support pre-cannulation. This will usually be able to be weaned off in the hours following cannulation as the cardiac function improves with delivery of oxygenated blood from the ECMO circuit and minimising of afterload to the right ventricle from a more gentle respiratory management approach.
Those supported with V-A ECMO should be managed on a high flow strategy (up to 200ml/kg/min) to allow a rapid wean of their inotropic support (Adrenaline, dopamine, milrinone) to minimise myocardial work and therefore damage and weaning of the vasopressors to allow a vasodilatory strategy to optimise tissue oxygenation.

Mixed venous saturation
It should be remembered that the ECMO mvSaO2 may not be the true patient SvO2 because there is often mixing of the blood between the venous deoxygenated blood returning to the right atrium and the oxygenated blood leaving the ECMO circuit. Obtaining a patient venous gas from a CVP line may assist in determining extent of mixing. It may be necessary to reduce the blood flow to minimise recirculation or use a modest amount of inotropy to aid the forward flow of blood from the right atrium to the right ventricle and the pulmonary arteries. A cardiac echo and 12 lead ECG should be obtained to guide the management of a failing right ventricle on V-V ECMO support.

A rapid rise in the ECMO SvO2 may represent significant recirculation or **cardiac tamponade.** An urgent echo should be sought and the treating intensivist and ECMO/Cardiac surgeon informed urgently.

Ventilation issues in paediatric respiratory ECMO

Rest settings
It is essential that the lungs are rested during ECMO undertaken for respiratory support. This means avoiding ventilator induced lung injury. This is undertaken by adopting lung rest settings which commonly means an FiO2 of 30%, PIP 20, PEEP 10, rate of 10bpm. CPAP via an endotracheal tube alone can also be used to avoid any inadvertent barotrauma or volutrauma from positive pressure ventilation. It is essential that any endotracheal toileting required is undertaken using minimal inspiration pressures and tidal volume with the T-piece to limit secondary lung injury at this time.

Once there is evidence of improvement in lung compliance the ventilatory pressures may be gently increased or HFOV utilised to re-recruit the lungs in tandem with physiotherapy and flexible bronchoscopy.

Physiotherapy
There is no indication for physiotherapy early in the ECMO run of patients with ARDS, the aim must be to **allow lung rest**. Physiotherapy is however essential in the management of these patients in the lead up to and following decannulation.
(See physiotherapy section in patient management)

Flexible bronchoscopy
See section in patient management

Recirculation issues
See cannulation section

Weaning from paediatric respiratory VV ECMO

As detailed above paediatric respiratory ECMO runs are often long with the longest at RHSC, Glasgow being 39 days. If one attempts to wean too early then it may result in further damage to the recovering lungs. However there are many complications associated with ECMO including infection and haemorrhage therefore the longer support is required the more likely there is to be an associated complication. The correct time to wean is one to be made using the experience of the clinicians caring for the child. There is no magic answer.

Prior to weaning from V-V ECMO ensure adequate positioning of the endo-tracheal tube and an acceptable chest x-ray appearance. A local review of 81 ECMO patients who underwent flexible bronchoscopy found that the ETT was at the carina or down the right main stem bronchus in 13% cases which would result in impaired ventilation. Therefore a low threshold for flexible bronchoscopy is adopted in these patients from a diagnostic as well as from a therapeutic angle to clear tenacious secretions.

Weaning from V-V ECMO in the paediatric patient is different to the wean from V-A ECMO as the ECMO circuit can be treated as a large shunt which merely removes and then returns blood to the right heart. For this reason there is **no need to wean the blood flows to low levels** as this requires an increase in anticoagulation and therefore potential a greater risk of haemorrhagic complications. There is also **no need to place the patient on the bridge** and anticoagulate the patient and the circuit. It is much simpler to merely **wean the gas flow and blender FiO2** then cap off the gas flow whilst continuing the ECMO flows (rpm) at previous levels whilst closely monitoring the patient's gas exchange on the arterial blood gas and ECMO circuit mvSaO2. The blender **FiO2 should be less than 40%** prior to capping off as above this the circuit is still providing significant support to the patient. If the patient demonstrates persistent respiratory compromise (rising pCO2/ETCO2 and falling SaO2/PaO2) despite optimised respiratory support then merely recommence or increase the gas flow to previous levels. It may be necessary to sigh the oxygenator (see below)

Use the VV ECMO wean checklist prior to a wean (Guideline section).

What to do in the event of a "wet" oxygenator (see guideline 19)

- Increase sweep gas to maximum for size of oxygenator for 2-3 minutes with blender at 100%
  - Medos 0800 Max sweeo gas flow 2.0L/min
  - Medos 2400 5.0L/min
  - Medos 7000 10.0L/min
- Repeat blood gas after 3 minutes at this gas flow
- Observe circuit closely for any bubbles
- If no improvement call Perfusionist and ECLS Physician

It is best to trial off V-V ECMO by capping off the gas flow first thing in the morning to allow decannulation in the early afternoon. After several hours of no gas flow to the oxygenator and demonstration of adequate respiratory support from the ventilator the patient can be safely decannulated. This is usually safely done by merely withdrawing the double lumen cannula and ensuring adequate pressure on the insertion site. Adequate platelet levels should be present prior to decannulation to aid local clotting of the cannulation site. It may also be necessary to give FFP or Cryoprecipitate to ensure clotting of the cannulation site.

It is important to ensure that the patient's cares are undertaken prior to decannulation and that there is no unnecessary movement of the head and neck following decannulation which may disturb the clotted cannulation site.

Paediatric cardiac ECMO

Introduction

This chapter will cover VA ECLS which is mainly used to rescue patients with life threatening heart disease. The majority of such patients have structural congenital heart disease. The remainder having an acquired heart disease such as cardiomyopathy, myocarditis and malignant dysrhythmias. Historically, various forms of extracorporeal support were developed around the cardiopulmonary bypass circuit, and hence the major use was in the support of the failing heart. VA ECMO supports both the heart and the lungs as an extended form of cardiopulmonary bypass. Although the predominant indication for paediatric ECLS has been neonatal respiratory support, peri-operative cardiac support is now the most common.

There are about 400 runs each year in infants and children with cardiac failure. Overall survival is between 30-50%. The overall survival of those with structural congenital heart disease is 30-35%; the indication with the lowest mortality is myocarditis/cardiomyopathy with a 60-79% survival. Whilst VV ECLS is accepted as the standard for initial ECLS support in respiratory failure, VA ECLS is almost the exclusive support for cardiac failure. VV-ECMO may indirectly improve cardiac output by improving oxygenation and ventilation (carbon dioxide clearance, a more favourable acid: base balance) and permit a consequent reduction in ventilation requirements. Collectively these reduce the pulmonary vascular resistance (afterload to the right heart) to improve right heart function, pulmonary blood flow and pre-load to the left heart. The improved substrate of the coronary blood flow may further enhance bi-ventricular function.
The basic principles of VA ECLS are identical in the treatment of cardiac and respiratory failure: gravity dependent venous drainage, oxygenation and ventilation, re-warming and return of blood to the proximal arterial side. However in patients who have structural heart disease, the characteristics of the resultant circulation can be dramatically different. This will become apparent throughout this chapter. Cardiac ECLS for the failing heart should be regarded as more than VA ECMO as it is imperative to minimize the myocardial work to promote rest and maximize any chance of recovery whilst meeting global oxygen demands. Centrifugal ventricular assist devices (VAD's) have the attractive potential of offering pure isolated ventricular support.
This chapter is not intended to be a reference text on cardiac ECLS, but rather a brief overview. It will need ongoing amendment as practice changes and experience with VAD's broadens. Published indications for VA extracorporeal support now include post-cardiotomy myocardial support, sepsis, post-cardiorespiratory arrest (E-CPR, not currently available in Yorkhill) and 'bridging to cardiac transplantation (currently not available in Yorkhill) or to recovery. A further indication is the stabilization and optimization to improve the global status of the pre-operative cardiac child.

Aim of Cardiac ECMO

- **To provide adequate tissue perfusion for vital organ function**
- **To decompress and off-load both the right and the left sides of the failing heart**
- **To maintain sinus rhythm where possible**
- **To rest the injured myocardium and expect high flow as detailed in the earlier text and indicated below (to bridge to VAD support or cardiac transplantation where appropriate)**

The aim of ECLS is to maintain global oxygen delivery. In cardiac VA ECLS strategies to rest the failing, previously injured heart, require specific attention.

The procurement of adequate coronary blood flow is essential during VA-ECMO. The heart will never recover without securing its own blood supply. The arterial cannula and thus the return of the arterialised ECLS flow are distal to the coronary ostia. Coronary flow is dependent upon primarily retrograde flow down the ascending aortic arch or the antegrade flow through the aortic valve. The antegrade flow of blood from the left ventricle will however consist of blood which has passed through the potentially poorly oxygenated and ventilated lungs and may be relatively desaturated and acidotic. Combination of antegrade and retrograde flows threatens competing flows and inconsistent delivery to the coronary ostia. Coronary flow occurs during ventricular diastolic relaxation and is dependent upon a coronary perfusion pressure, which for simplicity can be considered as the mean arterial blood pressure minus the right atrial pressure (coronary blood flow arises at the coronary ostia immediately distal to the aortic valve and ends at the coronary sinus in the right atrium). Sinus rhythm is ideal, even if this requires atrial pacing, as atrial systole against closed AV valves (right atrial systole against a close tricuspid valve) will raise the intra-atrial pressure and decrease coronary flow. Raised intra-ventricular 'end diastolic pressure' (EDP) means that in order to maintain physiological ventricular filling in diastole, atrial pressures will also be raised with similar counter-productive effects on coronary perfusion. Additionally a raised intra-ventricular end diastolic volume will stretch the coronary vessels narrowing their lumen and thus increase the resistance to flow. The heart must be decompressed and all intra-cardiac pressures minimized.

Prior to the initiation of VA ECMO, critical ill patients in haemodynamic extremis will have their tissue oxygen demands therapeutically minimised by adequate analgesia, sedation, paralysis and modest hypothermia. An initial strategy to augment a failing cardiac output is preload augmentation with volume supplementation. Volume given to the venous side will stretch the cardiomyocytes of the right atrium and induce energy dependent elastic recoil to enhance ventricular filling (Starling's Law). As tissue oxygen delivery is dependent upon the adequate movement of saturated haemoglobin, when the cardiac output is inadequate and the blood is optimally saturated, the only strategy remaining to compensate for the reduced flow is to augment the haemoglobin. A red cell transfusion may increase oxygen delivery by augmenting the pre-load and increasing oxygen carriage (although temporary low levels of 2,3 DPG may hinder oxygen release at tissue level).

Once VA ECMO has been deployed, this supplemental volume may be accommodated within the arterial side after systemic afterload reduction has been allowed by systemic vasodilation. High flow rates in excess of 200ml/kg/minute may be required to adequately circulate this volume and produce a normal cardiac output approximating 2.4litres/m^2/minute. Flow rates may be limited by the size of the venous cannula (a second venous cannula may be required), the degree of venous filling.

A balance has to be achieved between volume augmentation to achieve a satisfactory circulation (where the contribution of innate cardiac output is minimized) and total body fluid overload. If excessive intravascular volume or more commonly in the situation of total body oedema and a poor native urine output the consideration of CRRT can be interposed in the ECMO circuit as described in the guideline section. The usual practice is to discontinue inotropic support as inotropes work by raising intracellular calcium which also induces caspase activity inducing apoptosis or natural programmed cell death. The ventilation strategy should be adjusted to a lung protective mode avoiding damaging tidal volumes or high inspired oxygen concentrations. Ensuring an adequate residual lung volume with the use of PEEP to minimise atelectasis and correct the increase in pulmonary vascular resistance and right heart afterload effects is essential. Maintenance of normal lung function is very important to ensure that the fraction of pulmonary blood flow that may be presented

for coronary perfusion is oxygenated. Serial CXR's may demonstrate progressive indistinct peri-hilar shadowing suggesting increased pulmonary venous congestion of high left atrial pressures (see below) and a distended, non-rested heart. Whether maintenance of physiological tidal ventilation has any benefit is debatable.

Cardiac energy is consumed by cyclically generating the pressure required to expel a volume of blood. The stroke work index (SWI) is the work required to generate one pulse related to the patient's body surface area. The slower the heart rate, the lower the minute work and the longer the diastolic relaxation phase. Cardiac work is thus reduced by reducing the volume that is needed to be expelled (but this will reduce the effective cardiac output) or by reducing the intraventricular pressures that are needed to open the aortic valve for the left ventricle and the pulmonary valve for the right ventricle to produce a systolic pulse. A strategy of vasodilation to produce after-load reduction will decrease cardiac work. As importantly vasodilation dramatically reduces resistance to flow and thus whatever the heart is able to expel flows more freely and is propagated more efficiently. A lower blood pressure is the goal but determining the baseline target value is the difficulty. During isovolaemic contraction cardiac energy is consumed and is not available for ejection. Lowering the afterload of each ventricle will minimize this hidden work of the heart.

The key goals for Cardiac ECMO are:

- a decompressed heart (EDP and EDV)
  - 'slow' sinus rhythm
  - a normal arterio-venous oxygen extraction ratio (25-30%)
- the lowest mean arterial pressure that achieves:
- a normal acid: base balance
- normal lactate
- normal urine output
- normal arterial partial pressure of carbon dioxide.

A normal or high arterial partial pressure of carbon dioxide will optimise the cerebral blood flow when the cerebral perfusion pressure is compromised. This is crucial if the neck vessels have been cannulated as carotid cannulation impedes antegrade flow into the brain and the venous cannula obstructs the venous drainage in combination this will compromise cerebral perfusion.

In the standard VA ECMO circuit, the left atrium is not directly drained. As it is practically impossible to completely empty the right heart and prevent any pulmonary blood flow, a failing left ventricle will not be able to cope with the pulmonary venous return and is at risk of isolated distention. The chest x-ray becomes progressively congested, respiratory compliance falls and pulmonary oedema may develop to indicating poor left side decompression. The echocardiographic occurrence of 'smoking' or spontaneous contrast within the heart as the blood begins to stagnate heralds florid decompensation. A left atrial vent or a balloon atrial septostomy (BAS) to effectively create a common atria to permit venous drainage of the LA into the RA may be required.

The underlying cardiac diagnosis and its impact on ECLS physiology should be clearly understood. As detailed above the physiological principles apply equally to the structurally normal and abnormal heart, but depending upon the abnormality it is conceivable that the resulting flow characteristics when VA ECMO is added will require additional consideration. The univentricular heart, the presence of shunts (surgically created or native collaterals associated with the underlying heart defects), geometric mal-alignments and aberrations in morphology all add challenges to the management algorithm. A

consensus approach needs to be effectively communicated and coordinated. The management strategy of the initiating team should not be altered by successive staff unless clinically indicated and fully discussed.

ECLS support generates a myriad of ethical issues: It offers only temporary support until recovery. Once attached, transportation is logistically impossible using the roller pump however centrifugal circuits offer greater scope for patient transport both within and outwith the hospital environment. Emotions run high as end organ function begins to falter in the continual presence of non-pulsatile flow. Timely withdrawal of support may become the only dignified, respectful and humane option. Mobile ECLS services allow for transfer to a centre offering the hope of transplantation or the longer term support with VAD's may offer an alternative exit strategy. However the timely availability of matched donors cannot be guaranteed. There is not a paediatric transplant service available in our centre even though we have an ECLS programme.

Indications for Cardiac ECMO

The main indication for cardiac ECLS is post-operative low cardiac output syndrome (LCOS) associated with ventricular failure. This most often occurs after long and complex surgery associated with extended periods of cardiopulmonary bypass, prolonged cross clamp times and moderate or deep hypothermia. The pre-existing cardiac anatomy may make cardiac failure more likely, particularly if this has involved surgery to the coronary arteries. Most cases of cardiac failure result in severe pulmonary oedema. ECLS support has the advantage of ensuring oxygenation as well as supporting the heart.

Markers of low cardiac output:

- Increasing tachycardia
- Low mixed venous oxygen saturation (absolute value < 40%)
- Arteriovenous oxygen difference > 35%
- Rising filling pressures
- Incrementing inotrope requirement or the need for a third agent
- Toe core difference > 4°
- Sternum open or required to be reopened
- Oliguria
- Lactate increasing or failing to fall
- Persisting metabolic acidosis

Where possible we attempt to identify the potential need for ECLS pre-operatively: this patient group are characterised by a marginal preoperative ventricular function and/or a planned procedure that will radically alter the haemodynamics of the circulation. Those where the severity of LCOS is unexpected tend to require shorter runs and have a more favourable outcome.

Cardiac and pulmonary function should be deemed reversible, although this prediction is not reliably clear in the immediate post operative setting. It is contentious as to whether someone who is not considered appropriate for transplantation should be commenced on ECLS. However, ECLS as a bridge to transplantation is an established technique in centres that undertake paediatric cardiac transplantation. Late survival rates up to 43% have been reported.

There is a strong argument that the use of ECLS in the postoperative cardiac patient should be restricted to those who have had a complete repair of the underlying lesion. Several centres have

reported poor results when using ECLS after palliative procedures, most notably operation on the single ventricle.

Scenarios following cardiac surgery which may require ECLS:

- Myocardial dysfunction post-cardiomyotomy
  - Failure to separate from CPB
  - Progressive low cardiac output state
    - Arrhythmia
    - Cardiac arrest
    - Hypoxia-respiratory insufficiency
    - Pulmonary hypertension
    - Right heart failure
    - Shunt occlusion
- Elective augmentation: post-cardiomyotomy in HLHS
- Stabilisation between elective procedures
- Peri-transplant:
  - As a bridge
  - Post transplant to support RV failure induced by pulmonary hypertension
  - During acute rejection

The literature supporting emergency cardiopulmonary support is mounting. Survival rates vary in different series from 45-80%. This indicates that paediatric cardiac patients are salvageable and deserve an aggressive approach with rapid deployment of ECLS. Early restoration of cardiac output improves survival. Survival decreases with duration of CPR from time of *witnessed* arrest. We have little experience of using ECLS in the cardiac arrest setting (E-CPR). Other groups have reported variable success, with survival to discharge being reported following over 90 minutes of cardiac massage before initiation of ECLS. The potential use of ECLS in this setting is clearly dependent on the speed with which support can be initiated, though there is increasing evidence to support the statement that it is the effectiveness rather than the duration of CPR that is important, and if there is evidence of persisting neurological function then ECLS should be commenced.

A 60% survival to discharge rate has been reported by a group that could initiate crystalloid primed ECLS within 15 minutes of cardiac arrest. Other groups report a survival to discharge rate of between 11 and 64% for initiation of E-CPR up to and over 60 minutes after the cardiac arrest. As the circuit technology improves with the development of small, low-prime volume, centrifugal circuits, it is likely that commencing with a clear primed circuit will become more common. The rate limiting step for the use of E-CPR is staff availability. A clear primed circuit can be set-up and deployed rapidly.

When should Cardiac ECMO be started?

There is a lack of consistent accepted threshold criteria on which to commence cardiac ECLS. Unlike the oxygenation index in respiratory ECLS there is not a reliable predictor of mortality in cardiac ECLS. It is accepted that early initiation of ECLS, ***prior to*** a cardio-respiratory arrest offers the best chance of success. This avoids the end organ morbidity associated with prolonged periods of low cardiac output. However, ECLS is invasive and associated with life threatening complications.

Acknowledging an extremis situation which is not improving should mandate consideration and formal discussion between intensive care, cardiology and cardiothoracic surgery. Clearly the more indicators of low cardiac output state, the greater the need for a period of recovery.

Our unit's agreed criteria are in the appendix section of this chapter.

Cardiac ECMO patient management

Circuit Prime in Cardiac ECMO

The standard process for priming a circuit is the same for VA ECLS as for VV ECLS. Unless specifically requested a clear primed circuit using Plasmalyte 148 / heparin (1u/ml) is the standard prime for patients >10kg.

The circuit can be blood primed. The amount of blood which may need to be added to the prime will depend on the patient's circulating volume, haematocrit and the volume of the circuit. Whenever a clear prime circuit is on standby for a patient, blood bank should be informed so that they can ensure enough fresh patient specific blood is available. Smaller babies with lower circulating blood volumes can be haemo-diluted by initiation with a clear prime. However, this does not prevent its consideration: it may more beneficial in terms of oxygen delivery to establish an effective cardiac output, temporarily accepting the deleterious consequences of a lower haemoglobin until this can be corrected by an exchange transfusion. Children with established cyanotic heart disease develop a high haematocrit as a compensatory response to the low systemic saturations to maintain adequate oxygen carrying capacity. Starting with a higher haematocrit renders the effect of haemodilution less dramatic. Platelets and clotting factors will also be diluted by the pump prime and should be available.

Cannulation in Cardiac ECMO

There are two methods of cannulation: the transthoracic route is preferred in the immediate post operative period: advantages being rapid and accurate placement of large cannulae and sparing of the head and neck vessels; major disadvantages are the increased risk of mediastinitis and extensive haemorrhage. Neck cannulation (percutaneous or open dissection) removes these potential devastating risks but compromises cerebral blood flow during periods of low cardiac output as it impairs both perfusion and drainage of the brain. Without a recent sternotomy this may be the only approach as femoral cannulation requires narrower longer cannulae which do not usually allow sufficient flows. Echocardiography will be required to determine optimal cannulae position. The conversion of transthoracic to neck cannulation allows chest closure but runs the risk of covert thoracic tamponade (causing extrinsic compression of the atria and unreliable venous drainage). Left atrial decompression requires consideration either via a surgically placed direct LA vent if the sternum is open, or a percutaneous LA vent (catheter septostomy).

Cannula location will be influenced by the underlying cardiac anatomy, for example in patients with discontinuity of the systemic venous return (patients with a Glen circulation where the SVC is surgically attached to the pulmonary artery) may need both the SVC and IVC / common atrium drained.

Hypertension in Cardiac VA ECMO

Hypertension has been reported as the commonest cardiac complication to occur during cardiac ECLS,

reported in 38% of newborns. As previously indicated, in order to rest the failing heart, adequate decompression is required. High flows against a high systemic vascular resistance, which has resulted from the high levels of endogenous catecholamines prior to the start of VA ECMO support, will produce a high blood pressure. It is the SVR that needs to be reduced, not the ECMO flow. Merely turning down the flow will leave a residual volume within the heart which in turn will physiologically induce ineffectual contraction. If this occurs, the ventricle is not rested. Titratable short acting vasodilators such as sodium nitroprusside are preferred with the clinical goal of peripheral vasodilation to correct the 'core: peripheral' temperature gradient. Phenoxybenzamine, a long acting vasodilator paralyses the SVR abolishing the physiologically normal swings in peripheral temperature induced by changes in environmental temperature (when the child is exposed during examination and cares). From this stable baseline, vascular tone can be titrated to effect using vasopressin.

Chest exploration in transthoracic cardiac ECMO

- There is a significant risk of introducing air into the circuit at the time of chest exploration. This should therefore only be undertaken with 2 ECMO specialists present and all emergency drugs available.
- Consultant intensivist must be at bedside.
- Planned chest re-exploration only to be undertaken by Consultant Cardiac Surgeon.
- One ECLS specialist should watch patient and one watches circuit for potential air intake or other circuit issues along with perfusion staff.
- Coagulation management may need to be addressed to limit future re-explorations.

Ventilation in Cardiac ECMO

The purpose of cardiac ECLS is to restore oxygen transport that has declined because of poor pump function. A residual pulmonary blood flow is to be expected and it is important that this is oxygenated, as this will minimise the admixture of desaturated blood from the pulmonary circulation joining the fully oxygenated blood from the ECLS circuit in the aorta. Coronary blood flow may be more dependent upon the antegrade contribution thus pulmonary venous oxygenation should be maintained.
Normal respiratory ECLS rest settings may be inappropriate, but if co-morbid acute lung injury is suspected, pulmonary function can be rested with reliance upon the oxygenator. Iatrogenic lung dysfunction must be avoided, as a residual lung volume should be maintained to minimize pulmonary vascular resistance (PVR). Acknowledging the cardiopulmonary interactions and frequency of right heart injury, it is wise to minimize the high intrathoracic pressures generated by the ventilator and accept the temporary sequelae. *Note: HFOV and its constant mean airway pressure being greater than PEEP of conventional ventilation during cyclical exhalation is detrimental to the right heart. However, should this lead to re-expansion of collapsed lung units, the cumulative effect may be to reduce the PVR and paradoxically aid the right heart.* Inhaled nitric oxide causes vasodilation of the pulmonary vessels bathing the ventilated units reducing the PVR and correcting ventilation: perfusion imbalance.

Flexible bronchoscopy is often a useful adjunct in cardiac ECMO patients with a high incidence of positive findings including ETT misplacement and airway obstruction. It is often difficult to assess where the ETT radiographically ends on children supported with transthoracic V-A ECMO because of the presence of several other pieces of radiolucent material on the chest x-ray.

Myocardial stun

"A form of contractile dysfunction which follows episodes of myocardial ischaemia and persists despite restoration of coronary blood flow" (Bolli 1982)

- Classically associated with ischaemia/reperfusion episodes (e.g. aortic cross-clamping)
- Hypoxia – re-oxygenation of the cyanotic myocardium

Up to a third of patients on VA ECLS will develop myocardial stun, witnessed as a reduction or even complete loss of arterial pulse pressure soon after VA ECMO has been deployed. The causes of this phenomenon remain unclear. It is almost always completely benign. A plausible explanation is the combination of:

- The antegrade coronary blood flow is more hypoxic and acidotic because both ventilation and inotropic drive have been reduced
- Reducing the pre-load (i.e. emptying the heart) decreases cardiac filling, myocardial stretch and subsequent contraction (Starling's phenomenon)
- Retrograde coronary flow from the arterial cannula of hyperoxic blood predisposes to a reperfusion injury. (This effect could be attenuated by reducing the FiO_2 delivered through the blender.

Stun is suspected when the arterial pulse pressure falls to less than 10mmHg. Echocardiographically this is confirmed by almost complete absence of ventricular contraction (definitions vary) and a persistently closed aortic valve, in the absence of tamponade. Aortic 'pulsations' may be evident although this could be caused by oscillations of the aortic valve rather than ejection. Inotropic support has no effect on the stunned myocardium, and should be avoided. In the majority of cases, the function will improve. Adverse echocardiographic features for recovery include an increasing echo-bright myocardium, ischaemic ECG changes with the evolution of malignant dysrhythmias, spontaneous contrast swirling within the cardiac chambers and the development of valvular regurgitation.

Circuit Changes in Cardiac VA ECMO

Circuit changes should be rarely required in the centrifugal circuit.
By virtue of the fact that these patients have a failing myocardium they may be far less tolerant of circuit changes than respiratory patients. Resuscitation drugs should always be available. It is reasonable to hand ventilate the patient during these procedures and consider temporary inotropic support. With the exception of the patients with cardiomyopathy, the cardiac ECLS cohort require a reduced duration of support in comparison to respiratory ECLS, thereby offsetting the higher flows to demand less of a need for circuit changes.

Cardiac Tamponade

Thoracic tamponade can have an insidious onset and is a clinical diagnosis. ECLS staff should have a heightened awareness of this complication, especially as the conventional triad of signs (raised filling pressures, hypotension and tachycardia) may not be as recognizable whilst on cardiac ECLS. Extracardiac fluid (pleural or pericardial) may gradually accumulate and it is not until it contributes to atrial collapse and interruption with venous drainage that it becomes an urgent issue.

Emergency treatment is volume augmentation and temporary reduction in flows to achieve a reliable although reduced ECMO support. The pressure on the mediastinum may need to be released by removal of heavy dressings pending surgical re-exploration.

High suspicion of tamponade

- Blood soaked dressings visible over the mediastinum
- Acute stoppage of chest drainage
- Poor venous flow (pump "cutting out")
  - Increasing –ve inlet P
  - Falling blood flows
- Falling venous saturations
- Narrowing of arterial pulse pressure
- Rising patient PaO_2 as native ejection fails: (Patient PaO_2 = post oxygenator PaO_2)

Weaning from Cardiac V-A ECMO

Timing of the wean from VA ECLS should be tailored to the expected duration of recovery. Ventricular function post-cardiomyotomy following a protracted operation should recover within a few days, cardiomyopathy may take weeks until any reversibility is apparent. There is increasing evidence that a post-cardiomyotomy heart that has not recovered by 7-10 days is unlikely to do so. A repeated attempt to wean too early prematurely stresses the heart to reverse any benefit accrued from the period of rest and is likely to delay or even potentially prevent possible recovery. However, it is unlikely for recovery to be complete at the time of the decision to wean. Indeed it is not uncommon to decannulate myocarditis and cardiomyopathy patients with a moderate degree of ongoing dysfunction as the alternative is a prolonged bridge to transplant, with no guarantee of a heart becoming available. Continuing recovery after decannulation is also to be expected in many of these patients.

Whilst recovery post-cardiomyotomy is expected within 5 days, children with co-morbid respiratory failure after cardiac surgery may require longer. Iatrogenic lung diseases (ventilation associated pneumonia, gravity dependent atelectasis) are preventable complications. By definition there has been ventricular dysfunction in patients on cardiac ECLS, and conditions need to be optimal for the heart to take over the responsibility of tissue oxygen delivery. Finessing lung function, ventilation strategy, removing excessive total body fluid (thoracic wall oedema will make the chest less compliant, high ventilation pressures are detrimental to right heart function) and optimizing haemoglobin are more important than in the non cardiac ECLS patients. Bronchoscopy may be needed to remove obstructing plugs, confirm correct endotracheal tube placement or to diagnose an under-lying problem such as airway malacia which may only become apparent when heart and lung function is compromised. Inhaled nitric oxide may be required to minimize 'ventilation: perfusion' mismatching and to decrease pulmonary vascular resistance. It is reasonable to minimize oxygen demand by increasing sedation and reintroducing paralysis during attempts at weaning. The cardiac rate and rhythm may need to be optimized. If the patient needs to be paced then determining an optimal rate, establishing A-V synchrony (although appreciating that the addition of ventricular pacing may be detrimental) determining the optimal A-V interval and establishing thresholds all need to be optimized prior to the wean.

Signs of improving cardiac function include:

- Improved characteristics of the arterial waveform: steep up stroke, narrowing of the base, widening of the pulse pressure, re-occurrence of the dichrotic notch
- Improving SvO_2
- Discrepancy between patient PaO_2 and post oxygenator gases (as physiologically oxygenated blood ejected from the heart has a lower PaO_2 which when mixed with ECMO blood means the distal arterial PaO_2 will be lower than the post oxygenator PaO_2).
- Improving cardiac function on echocardiography

It is expected that inotropic support will be needed to aid separation, however upper limits that indicate failure are not defined. Low dose adrenaline or dopamine should be delivered to the patient and not to the circuit. Significant support should be interpreted as an indicator that the heart has not sufficiently recovered and unless circumstances mandate coming off ECMO on high inotropic support, this should be avoided. Echocardiography during weaning may demonstrate temporal changes and trends and should be considered to explore any haemodynamic change. It is impossible to be prescriptive on how to wean the cardiac patient and strategies will vary depending on whether or not there is a bridge in the circuit, the urgency to come off and response to weaning. A variable clamp can be applied across the bridge and incrementally opened to allow total circuit flows to be maintained whilst patient flows are decreased. This strategy is of particular use if there are concerns with anticoagulation and lower ACT's are being targeted. This requires a flow transducer to be located distal to the bridge. The conventional method is to reduce the total circuit flow to an 'idling' value with the bridge closed. The patient can then separated by 'clamping' **ABV**. Decannulation is considered later if the haemodynamics remain stable.

Signs that the heart is struggling:

- Increasing heart rate
- Falling SvO_2*
- Falling arterial saturations
- Increasing CVP, LAP
- Decreasing BP
- Increasing acidosis and lactate

* Note: If there is effective pulmonary venous oxygenation and an inter atrial connection (balloon atrial septostomy, surgically placed LA vent or native septal defect) which allows a left to right shunt, the SvO_2 on the venous saturation monitor may be unrepresentative of the true MvO_2.

During the weaning process if the patient has been vasodilated on the circuit then volume and/or pressors may be needed. Longer term dilators such as phenoxybenzamine are often ceased prior to the wean. It should be remembered that unless circumstances dictate otherwise, an attempted wean is a surrogate test for myocardial recovery and is being performed to assess readiness for decannulation.

The weaning process should be individualized for each patient. In the heart with significant ventricular dysfunction then a more gradual weaning process over 48-72 hours may allow the ventricle to accommodate to the increased volume and demand. Clinical criteria alone are insensitive at predicting ventricular recovery. Flows should be reduced gradually over a few hours, at a rate of 50-

100ml/min per hour towards idling flows. If this is done overnight then the response can be assessed the next morning and decannulation can occur early during the day. Daily parameters should be set to allow the ECLS specialist to wean if possible. In the majority of cases, the blender will be weaned to avoid unnecessarily high PaO_2 values. Considerable care must be taken to focus on the $PaCO_2$ as this can be dramatically reduced with the combination of ECMO and native ventilation which causes cerebral vasoconstriction and markedly reduces cerebral blood flow. The sweep gas flow may need to be reduced.

If the attempt is unsuccessful, an explanation needs to be sought. Were all conditions optimized? It may be necessary to continue with low dose inotropes and consider other adjunct therapies. Repeated weans in close succession should be avoided unless circumstances dictate otherwise, such as bleeding, sepsis or non-viability of the cannula site.

Reasons for failure to wean from Cardiac ECMO:

- Heart not recovered
- Residual / unmasked cardiac or pulmonary lesion - (cardiac catheter)
- Pulmonary hypertension
- Patient sepsis - temperature masked on circuit
- Primary respiratory problem
- Aortic cannula physically obstructing the aorta

The potential impact of any residual lesion requires careful consideration. When haemodynamics (flow, pressures and geometric arrangements) are marginal, defects tolerated by the normal heart can have a dramatic impact upon the compromised myocardium. The poor ability of a ventricle to generate a pressure cycle makes the interpretation of shunt across a VSD or residual outflow tract obstruction difficult to quantify. Valvular regurgitation will be less well tolerated. Early cardiac catheterization to exclude any residual defect amenable to surgical intervention is essential as residual cardiac lesions have been associated with adverse outcomes.

NB:

- Follow procedures for heparin infusions
- Follow procedures for flushing bridge, arterial and venous lines
- Sedation onto patient
- Inotropes onto patient
- TPN onto patient
- Avoid hypocarbia: the sweep should be reduced at the same rate as the blood flow
- Haemofiltration in parallel with the ECLS circuit will be interrupted by placing the patient on the bridge..

Single Ventricle Physiology

Controversy surrounds the use of ECLS in patients with single ventricle physiology. These patients are at particular risk of coronary insufficiency, exacerbated by run-off down the 'systemic – pulmonary' shunt causing increased pulmonary blood flow. This leads to increased pulmonary venous return back into the common atrium, compounding effective drainage, risking ventricular distention

and further hindering cardiac rest. Historically single ventricular physiology was a contraindication to ECLS. Current reported survival rates indicate 65% success with better results if ECLS is commenced in theatre. As experience grows, results continue to improve and now ECLS is used before the first stage to allow the heart and end organ function to recover.
The impact of the 'systemic – pulmonary' shunt flow is the problem. Complete occlusion is associated with severe pulmonary injury, oedema, pulmonary haemorrhage, non complaint lungs and pulmonary infarction. A patent shunt divides ECMO arterial flow depending upon the relative resistance of the pulmonary and systemic circulations. Torrential pulmonary blood flow steals from the systemic circulation and floods the lungs. This compromises coronary, cerebral and gastrointestinal flood flow producing end organ ischaemia, The disproportionate increase in pulmonary blood flow causes direct lung injury, prevents effective cardiac decompression thus jeopardizing cardiac rest and subsequent weaning from ECLS.

Two strategies have been described for the management of the single ventricle on ECLS. The shunt can be clipped to partial occlusion, although it is difficult to achieve an accurate balance and a stable situation when both the PVR distal to the shunt and the SVR are variable and influenced by many factors. Alternatively as the SVR is many orders of magnitude greater than the PVR, shunt patency is maintained whilst the SVR is manipulated with vasoactive agents. This is standard perioperative management of the univentricular circulation. Vasodilation minimizes the SVR encouraging systemic flow, whilst ventilatory strategies limit pulmonary flow. If this medical management fails, partial shunt occlusion should then be considered.

Without adequate decompression a failing ventricle will dilate and cause atrio-ventricular valvular regurgitation. During ejection retrograde flow occurs and steals from the effective systemic output. Increasing the ECLS flow in the expectation that this will improve drainage and decompress the heart is fundamentally flawed in this patient group. The only effect this intervention will have is to circulate the blood faster unless the systemic to pulmonary flow ratio is manipulated. ECLS flow rates must be sufficient to ensure adequate systemic perfusion (indicated by patient systemic venous saturations) and to compensate for that fraction of total cardiac output lost as pulmonary blood flow.

The use of centrifugal ventricular assist devices is gaining popularity in the postoperative management of these patients. Indeed in some institutions it is used in all patients with hypoplastic left hearts to augment cardiac output in the initial postoperative period. Clearly the lungs must be able to oxygenate the pulmonary blood flow for a VAD to be effective.

In order for patients with a univentricular physiology to wean from ELCS, cardiac function must have recovered enough to tolerate the obligatory volume overload on the single ventricle. The single ventricle has to accommodate and generate sufficient work to support both the systemic and pulmonary circulations. Where the left ventricle is hypoplastic, survival is reliant upon isolated right ventricular function, despite only being designed to eject into the low resistance of the pulmonary vascular bed. The greatest determinant of ventricular work is the pressure that it has to generate to eject against its afterload. Residual narrowing of the reconstructed aortic arch creates unnecessary afterload to the ventricle. It is likely that this will need to be corrected prior to successful weaning from ECLS.

Ideally these patients will have oxygen saturations of around 75% during weaning. Higher saturations imply a proportionately higher pulmonary than systemic blood flow. Under most circumstances this is the opposite of what you want to achieve. Optimum oxygen delivery to the tissues is achieved by:

1. Ensuring Hb 14-16g/dl
2. Pulmonary venous saturations >95% (not directly measured)
   a. Avoiding pulmonary oedema
   b. Avoiding atelectasis collapse consolidation
   c. Avoiding pneumothorax
   d. Cautious use of oxygen
3. Maintaining adequate cardiac output

There is limited experience with ECLS support of cavo-pulmonary circulations. With the bidirectional Glen, partial separation of the systemic venous return to divert blood directly into the lungs reduces the volume that the heart has to accommodate. The work that it has to generate is reduced as it no longer directly contributes to pulmonary blood flow. If a patient with a Glen circulation requires ECLS atrial cannulation will only drain the IVC flow. The SVC may also require draining as elevated venous pressures with a reduced arterial pressure may severely jeopardize cerebral perfusion. If a patient with a Glen circulation is cannulated via the neck the femoral vein will also need to be cannulated to achieve adequate venous drainage.

Myocarditis

On the ELSO cardiac registry survival with myocarditis is the highest of any group at almost 65%. Support should be considered if standard methods of reducing oxygen demand fail to meet supply. Inotropic support may predispose to intractable malignant arrhythmias and should be used with caution. ECLS should be considered early, especially as the outcome is so favourable. ECLS allows for recovery which is expected to be fairly rapid, and it is becoming increasingly apparent that there is significant reversibility if the heart is supported during the acute phase of the illness. These patients are particularly susceptible to left ventricular distension on ECLS and pre-emptive septostomy should be performed.

Near Infra-red Spectroscopy in Cardiac V-A ECMO

See separate section

Summary

ECLS can provide excellent short term cardiac support to allow recovery of the heart from an acute insult. The fundamental principle of cardiac VA ECLS is to provide an environment which optimises cardiac rest.

General indication for mechanical cardiac support

- Severe circulatory failure unresponsive to conventional management
- Anticipated myocardial recovery
- Continued support justified
- NO absolute contraindications to ECLS
- Technically successful surgical repair

Possible ECLS scenarios following cardiac surgery in children

- Failure to wean from cardio-pulmonary bypass
- Intractable low cardiac output
- Uncontrollable arrhythmia
- Refractory pulmonary hypertension
- Pulmonary failure

Other potential indications for 'cardiac' ECLS

- Severe pre-operative haemodynamic instability
- Acute cardiac failure (myocarditis, drug overdose, pulmonary embolism)
- 'Bridge' to transplantation or long term recovery

ECLS is contraindicated in presence of:

- Irreversible cardiopulmonary disease
- Severe neurological impairment
- Life threatening underlying disease
- Lethal congenital anomalies
- Extreme prematurity
- Grade 2 IVH
- Intraparenchymal haemorrhage
- Proven NEC
- Incurable malignancy

Relative contra-indications include:

- Severe coagulopathy
- Complex venous anatomy
- Valvular regurgitation
- Prolonged cardiac arrest or out of hospital cardiac arrest
- Advanced multi-organ failure
- Severe CNS disease
- Non transplant candidate

Guidelines for instituting an ECLS referral post Cardiac Surgery (September 2008)

Following on from the ECLS and Cardiac surgery 6 monthly reviews with the NSD it was felt that we should explore as a group the use of guidelines to assist in identifying a sub-group of cardiac surgical patients likely to benefit from ECLS therapy.
The initial meeting in December 2005 of Cardiac surgeons, Cardiologists, Intensivists" agreed to initially look to the world literature and to talk to other ECLS centres to see if guidelines or protocols were available which could be readily modified for use in the Scottish services.
A Medline search found only two relevant papers which are précised here.

Proposed entry criteria for postoperative cardiac extracorporeal membrane oxygenation after pediatric open heart surgery; Trittenwein G, Pansi Heike, Graf Bernadette et al: Artificial Organs 25:11:1999 1010-1014

This paper looked at 218 children admitted to an Austrian PICU post corrective open heart surgery in a two year period 1994 to 1995. Age ranged between 0 and 24 years with a mean age of 3.1 years. Overall mortality was 11.46%.
They identified Lactate and Central venous oxygen saturation on admission to the PICU as sensitive predictors of subsequent mortality. A ScvO2 <60% with a Lactate >7.8 mmol/l or a Lactate >18.3mmol/l with a ScvO2 > 60% were associated with an 80% mortality. They suggested that they would use this as an indication for early post operative institution of ECLS.

This paper is looking at a population from more than 10 years ago, from an institution with an overall mortality of 11.46%. No follow up paper has been published since 1999 identifying whether having instituted these criteria for ECLS an improvement was perceived in outcomes. We currently routinely measure Lactates and ScvO2s are readily available. The Lactates quoted in this paper would certainly be alarming if not rapidly correcting.

Risk factors for mortality in 137 pediatric cardiac intensive care unit patients managed with extra corporeal membrane oxygenation, Morris MC, Ittenbach RF, Godinez RI, et al: Critical Care Medicine 35:4:2004 1061-1069

This paper looked at 137 children with heart disease who were managed on ECLS in Children's Hospital Philadelphia over a 6 year period 1995- 2001.
Age ranged between 0-42 years with a mean age of 4.7 years. Overall ECLS mortality was 61% (53 survivors to hospital discharge).
89 children were started on ECLS post cardiac surgery representing 3.4% of the 2598 open heart cases in the study period (14 patients requiring VAD during this period were not included). Mortality rate for post cardiac surgery patients was 60% (NS difference from non post op cardiac patients). Significant multivariate predictors of a negative outcome from data available pre ECLS in the cardiac surgical group were age <1 month and male gender. They found no relation to failure to wean from bypass and outcome or anatomy and outcome.

This paper is from CHoP's a large North American Children's hospital with a well establish cardiac surgery programme and low mortality.
This paper concludes that "Because children with cardiac disease and circulatory failure represent a very heterogeneous group, it is imprudent to impose strict indications for or contraindications to ECLS support"

Feedback from other ECLS Centres

GOS

Alan Goldman said that they haven't pinned down parameters for going on cardiac ECLS at GOS. Failure to come off CPB would obviously be one. Their survival figures are 50-60% for ECLS started in theatre VS 28% if ECLS initiated after arrival in PICU.

He suggested that were no clear parameters but certainly lactaemia, "sagging BP unresponsive to inotropes", low SVO2, unresolving acidosis, combined with experience and "gut instinct" were what they used. They always have an infant circuit available with a clear prime which could be used to sustain an older child while a larger circuit was being prepared.

Toronto

Des Bohn replied that the criteria in Toronto are Failure to wean from CPB or cardiac arrest following corrective surgery. For pre-emptive ECLS post cardiac surgery no defined parameters but "declining cardiac output, assessed by SVO2 and Lactate measurements", not reversible with pharmacological interventions, mechanical ventilator manipulations or sternal opening would be an indication for ECLS.

Melbourne

Frank Shan replied that Melbourne had no set criteria or guidelines outwith failure to separate from CPB and low cardiac output in PICU unresponsive to conventional therapy.

Conclusion

Most major paediatric intensive care units do not have strict criteria for instituting ECLS. It is suggested that we agree criteria that should make us consider ECLS. Combining some indication of low cardiac output with Lactate and SVO2 the following combination of clinical signs/symptoms/results would trigger a discussion between the Cardiac Surgeon, Intensivist, Cardiologist and ECLS representative about the role of ECLS.

- Toe Core gap >4 , >4 hours post op
- Increasing adrenaline requirement to maintain blood pressure
- Oliguria
- Lactate > 7.8 with SvO2 less than 60%
- Lactate > 18.3 with SvO2 greater than 60%

The discussion would take cognisance of the fact that no well agreed "cast iron" criteria with sufficient sensitivity and specificity are available to correctly identify all children who would benefit from ECLS while excluding those who would not. The outcome of the discussion would be recorded in the patient's case notes.

ECMO Emergencies

Complications during an ECMO run, though rare for routine cases, should always be expected. It is a highly complex modality of support and catastrophic complications can arise unexpectedly and emergently. The success or failure of ECMO therapy is highly dependant on the prompt recognition of complications and their effective management. The ECMO specialist has a crucial role in this respect with responsibility for their recognition, prevention and management. A clear understanding of the physiology applicable to ECLS is mandatory. Familiarity with the circuit and attainment of a level of confidence can prepare specialists to handle most of the problems routinely encountered. Attendance at regular water drills and annual updates are mandatory and will prepare specialists to safely and competently manage the ECMO patient.

ECMO complications can be classified as mechanical or patient related. Mechanical complications are those involving failure of any part of the ECMO circuit or equipment. Patient complications can span the whole field of critical care medicine but are often related to the need for systemic anticoagulation and increased risk of haemorrhage. The ELSO registry with details of over 30,000 ECMO runs reports an incidence of 2.7 complications per ECMO case with an overall survival of 76% but despite dissemination of ECMO experience complication rates are increasing, partly due to the increasingly complexity of selected patients.

Neonatal Respiratory Complications	***No. Reported***	***% Reported***	***No. Survived***	***% Survived***
Cardiovascular: Inotropes on ECLS	4355	18.5%	2662	61%
Mechanical: Clots: oxygenator	4145	17.6%	2709	65%
Renal: Haemofiltration required	3,359	14.3%	1,809	54%
Infectious: Culture proven infection (see Infections)	1,443	6.1%	767	53%
Mechanical: Oxygenator failure	1,397	5.9%	739	53%

Figure 1: Complications associated with ECLS (ELSO, 2009)

The recognition of potential complications is key in preventing the escalation of minor issues into full blown emergencies. Taking a baby off ECLS, even briefly, can produce untoward effects (hypoxia, rapid acidosis, severe bradycardia, etc.).

Assessment of the ECMO circuit

A systematic assessment should be performed at the beginning of each shift and at regular time intervals according to local policy. Assessment should begin with the Cannula site, starting at the venous Cannula then following through the circuit to the arterial Cannula. Assessment of any life support system should begin with determining whether or not a life threatening situation currently exists. During ECLS the two major life threatening complications are air being pumped into the patient's arterial system or blood loss from disruption of any component.

Assessment of each component should include the following:

Venous cannula

- What Cannula is being used
- Is the Cannula in a good position via x-ray
- Is the suture secure
- Is the wound bleeding
- Are there any clots in the tubing
- Is the Cannula secured to the bed in a proper holder
- Is the venous pressure satisfactory ie < -50
- Are the pre-membrane pressures within normal limits

Arterial cannula

- What Cannula is being used
- Is the Cannula in a good position via x-ray
- Is the suture secure
- Is the wound bleeding
- Are there any clots in the tubing
- Is the Cannula secured to the bed in a proper holder
- Are the post membrane pressures within normal limits

Pump head

- Is the pump head secure in the motor
- Are there any clots in the pump head
- Are the tie straps secure
- Is the flow probe attached

Connectors

- Are they secure
- Are the tie straps secure
- Are there any clots

Oxygenator

- Is the sweep gas rate within the range for the oxygenator
- Is the blood flow rate within the range for the oxygenator
- Is the exhaust port unobstructed
- Is the sweep gas connection secure
- Is the moisture draining from the exhaust port clear
- Is there air at the top of the oxygenator
- Are there clots at the top of the oxygenator
- Is it secure in it's holder

Water heater

- Is the water bath level adequate
- Is the water circulating freely
- Is the water in the water bath clear
- Are blood flow and water flow countercurrent
- Is the patients temperature stable
- What is the water heater temperature

Environment

- Are there enough tubing clamps
- Is there a sufficient supply of blood products in blood bank
- Is there a stocked supply trolley
- Is the back up console plugged in and fully charged

To Remove Patient from Centrifugal ECMO

Never clamp venous (blue) lumen without clamping arterial (red) lumen first as this will cause cavitiation of gas in blood within device.

Remove the patient from bypass

(Arterial-Bridge-Venous)

Stop the pump by depressing the red button for 2 secs. Increase the ventilator to emergency settings. (see daily parameter sheet). Turn off all sweep gas flow to the circuit (or reduce blender to 0.21) while the patient is off bypass. (If the oxygen flow into the oxygenator is not stopped, then super-saturation with micro-bubble formation can occur.) Discontinue all infusions to the ECLS circuit. This will prevent the patient from receiving a bolus of fluid or heparin when bypass is re-started. If possible, continue to circulate the blood in the circuit through the A-V bridge at 200 ml/min. More often than not, when a patient must come off bypass, recirculating blood through the A-V bridge will not be possible. This is due to the fact that the precipitating events are usually mechanical or circuit emergencies. In this instance time becomes a very important factor. When the patient is taken off ECLS, it is important to record all parameters. The patient's vital signs should be monitored and recorded, as well as the ventilator settings. A comprehensive note should be written to explain the incident. If the patient is to remain off ECLS for longer than 15 minutes, the ACT of the patient should be checked, especially if the ACT was on the low side prior to coming off bypass. Since the patient is still metabolising the heparin in his body, it is possible that the ACT could return to normal, thereby causing clot formation to occur in the ECLS access cannulae. To prevent this, transfer the heparin infusion from the circuit to the patient. If you can circulate the pump, run a fresh heparin infusion at 50% patient rate as soon as possible.

Mechanical complications

Thrombosis

Clots in the circuit are the most common mechanical complication reported to ELSO. The development of very small clots may not be preventable and cause no significant problem. Larger clots however can lead to oxygenator failure, consumptive coagulopathy and pulmonary or systemic emboli.

Clots on the arterial limb of the circuit should be considered an emergency as this can lead to systemic embolisation

Action:

- Contact ECLS consultant and Perfusion
- Consider Increasing ACT parameters by 20
- Consider checking Heparin level, anti-Xa and anti-thrombin 3
- Consider Prostacyclin (Epoprostenol) 5ng/kg/min (Consultant only approval)
- Consider increasing flows to safeguard circuit integrity
- Consider circuit change if severe clot or consumptive coagulopathy is an issue

Cannula Problems

About 12% of patients supported with ECLS, have complications associated with the cannula, (ELSO, 2009). Cannula problems relate to both the venous and arterial cannula. The venous cannula is normally threaded through the right internal jugular vein into the right atrium. If the venous cannula is inserted either too far distal or proximal to the right atrium, this can cause obstruction of the cannula drainage holes. The venous cannula can be inserted into the Subclavian vein. Anatomic variations of the right atrium can interfere with venous return, such as an aneurismal atrial septum or redundant eustachian valve. Problems with venous cannula position will be displayed by a low or increasingly negative venous pressure, increased recirculation (VV) and a reduction in flows.

The arterial cannula is inserted into the right common carotid artery so that its top rests at the bifurcation of the right common carotid artery and aortic arch. The arterial cannula may be inserted too far into the ascending aorta, descending aorta or misdirected into the Subclavian artery. Insertion too far into the ascending aorta can cause increased afterload to left ventricular outflow and may contribute to left ventricular failure. Insertion too far down the descending aorta can compromise coronary and cerebral oxygenated blood flow. The distance from the orifice of the innominate artery to the take off of the right Subclavian artery can be very short 1-1.5cm, if the arterial cannula is pulled out to the point where the arterial infusion can selectively enter the Subclavian artery the right upper extremities can be infused with post oxygenator blood flow, while the rest of the body is hypoxic and cyanotic.

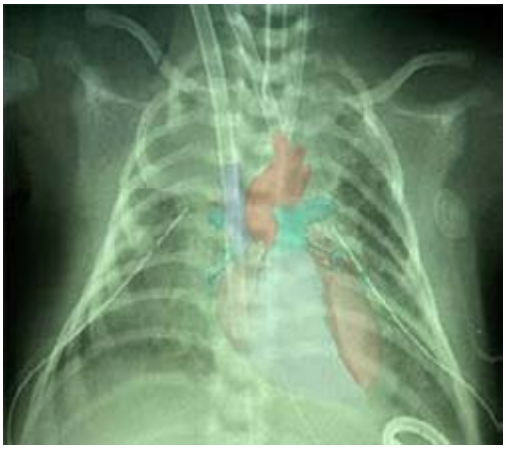


Figure 1: Good cannula position on VA ECLS

When performing single cannula VV ECLS, the procedure is the same as that for VA ECLS except that the carotid artery is not cannulated. The tip of the cannula should rest in the mid-right atrium with the arterial connector pointing towards the ear. This directs the arterialized blood preferentially to the tricuspid valve.

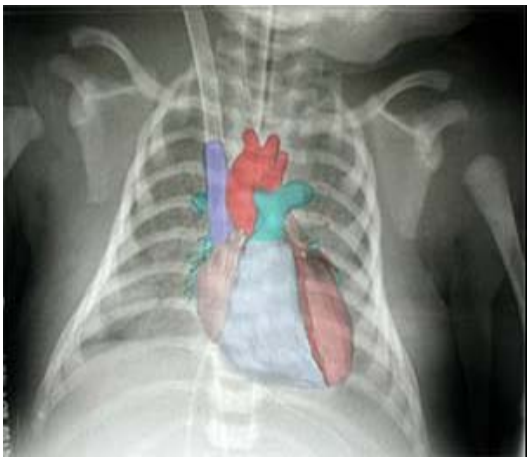


Figure 2: Correct Double lumen cannula placement

Decannulation:

In case of accidental decannulation, apply firm pressure to the site, come off bypass, notify the ECLS physician and surgeon immediately. Use Emergency Ventilator Settings. Notify the blood bank and get the backup unit of PRBC's immediately. Expect the patient to have a significant amount of blood loss. Accidental decannulation can be prevented by closely supervising all personnel who come close to the cannulae. Remember, you are the BOSS!

Flow Problems

Can be caused by:

- fall in patient's intravascular volume or by obstruction to flow into **venous (blue) cannula**

  Action:

  - Assess patients filling pressures (CVP)
  - Assess venous line pressure and venous tubing for kinks
  - Assess cannula position, reposition patient, assess need for neck roll
  - Volume 5ml/kg
  - Reduce rpm by 10% to allow filling of RA before slowly increasing rpm watching flows on pump console.
  - Consider cannula and tubing size in relation to flows demanded
  - Consider Tamponade or Pneumothorax
  - Consider CxR and/or ECHO

- or by increased afterload.

  Action:

  - Assess Arterial cannula and tubing for kinks
  - Check post- membrane pressure
  - Assess patient MAP
  - Consider sedation/paralysis
  - Consider increasing vasodilation
  - If all else is excluded consider increase in RPM if venous pressure satisfactory

Membrane Failure

Oxygenators, just like lungs can develop pulmonary oedema, ventilation/perfusion mismatch and pulmonary embolism. Clot formation can affect the membrane surface, inlet port, or outlet port. These conditions can be diagnosed by measuring pre- and post membrane pressures and pump blood gases. The resistance to blood flow produced by a membrane oxygenator is referred to as the gradient. The gradient is derived by subtracting the post membrane pressure from the pre membrane pressure. As thrombus formation increases in the oxygenator the gradient increases reflecting an increase in resistance to flow and CO2 and O2 transfer decreases. Clot formation on the oxygenator results in decreased oxygen and carbon dioxide transfer. Pre and post membrane blood gases are assessed to determine adequate gas exchange. Any acute change in the membrane oxygenation and/or ventilation would be reflected in the patient's condition. Carbon dioxide retention can occur when fluid (water or blood) accumulates in the gas phase of the oxygenator. The fluid becomes saturated with oxygen from the ventilating gas so that blood perfusing that section of the oxygenator is still oxygenated. However the gradient for carbon dioxide removal is decreased as carbon dioxide accumulates in the gas phase fluid. The rate of the oxygenator sweep gas can be increased in an attempt to improve carbon dioxide removal and to assist with removing excess water (Sighing the Oxygenator) see guideline section.

Complete clotting of the oxygenator is a clinical emergency and requires the patient to be clamped off ("ABV") and supported conventionally whilst the perfusion and surgical teams assemble to replace the circuit. The re is a risk of air entrainment at this time. This is most likely at "low-flow" situations on VA ECMO therefore the **ACT parameters should be elevated to 220-240** as detailed in the coagulation chapter **even if there is evidence of bleeding**.

Signs of a failing oxygenator include:

- Deteriorating patient gases
- Colour changes to post oxygenator blood
- Increased moisture at membrane exhaust
- Decreased post membrane Pressure (P2)
- Increased gradient despite no increase in RPM
- Increasing pre oxygenator pressure (P1)
- Consumptive coagulopathy

Action:

- Check Gas line secure
- Check gas flow meter
- Check membrane gases for ↑ Co2 or ↓ Pao2 (remember if blender < 50% pump Pa02 may be lower)
- Increase blender to 100%
- Sigh membrane for 2-3mins (0800 Oxy – gas flow of 2litres; 2400 Oxy – 5litres; 7000 Oxy – 10litres)
- Recheck pump gas at above gas/blender rates
- If no improvement contact perfusion
- Observe membrane for blood in gas phase

Air Embolism

Air in the ECLS circuit represents about 4% of complications reported to ELSO. It can range from a few bubbles in the pump head to a massive air lock which stops flow. An ECLS circuit has several potential sources of air embolism:

- When the partial pressure of oxygen in the blood is very high oxygen can easily be forced out of solution.
- The negative force generated by the centrifugal pump as blood is siphoned on the venous side of the pump will 'suck' air into the circuit even through a minute crack in a connector, a poorly closed 3 way tap or poor suture line at the site of the cannula entering the vein or atrium.
- Significant Venous airlock is usually the result of cannula dislodgment where 1 or more side holes lie outside the vessel.

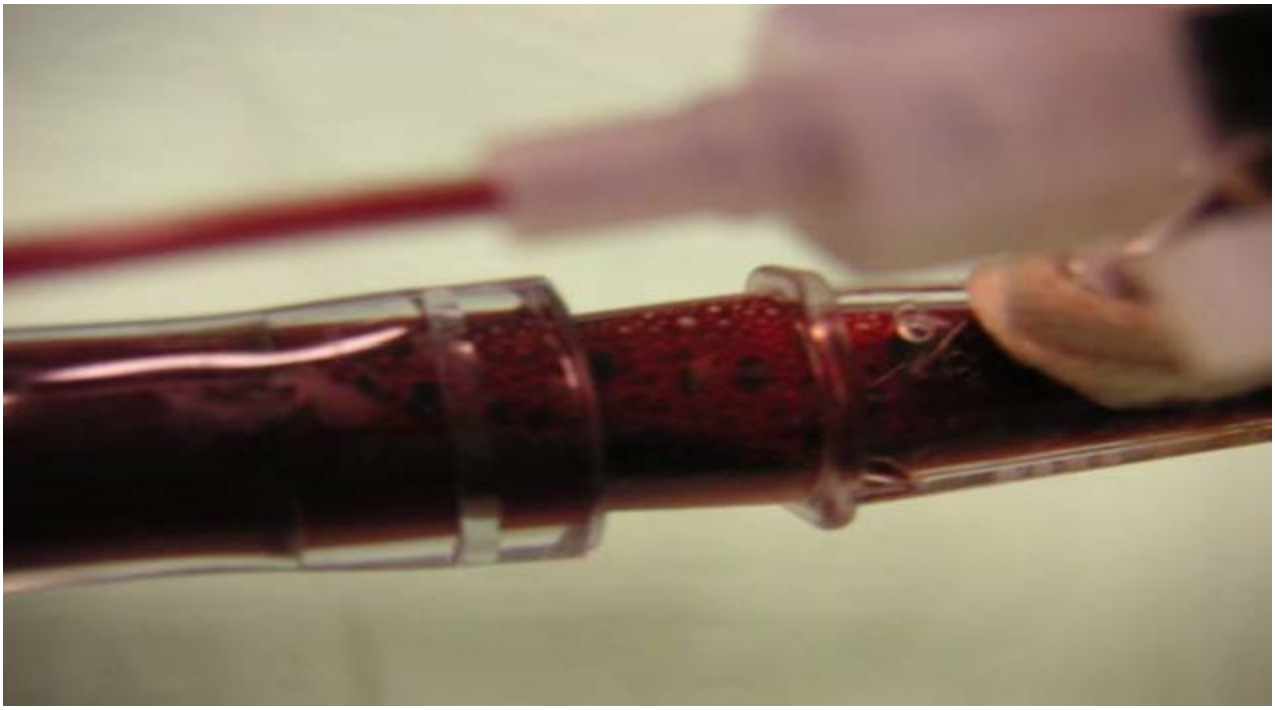


Figure 3: Air bubbles in Levitronix ECLS Circuit

Whenever air is observed in the arterial side of the circuit, it is an emergency and the patient must be immediately protected from the air.

Never clamp venous (blue) lumen without clamping arterial (red) lumen first as this may cause cavitiation of gas in blood within device.

If Air Is Observed In The Centrifugal Head **ONLY**:

- Place clamp on arterial tubing only, leaving bridge clamp in place
- Initiate emergency ventilation and inotrope settings.
- Turn the pump off, remove head from pump casing
- "Walk" air to pigtail #7 at top of oxygenator and aspirate using patient as volume reservoir
- Replace head in pump casing, set rpm to 1500 and restart pump flow by removing arterial clamp.
- Check top of membrane and remove any trapped air.
- Increase to previous RPM
- Observe circuit for source of air.
- Document total length of clamp off period in specialist evaluation

If Gross Air Identified In The Circuit:

- Take patient off ECMO immediately, clamp on arterial tubing, remove bridge clamp and clamp venous tubing. A-B-V
- Initiate emergency ventilation and inotrope settings.
- Contact on call perfusionist
- Turn the pump off, remove head from pump casing
- Prime giving set with saline and connect to venous pigtail situated immediately before pump head and open flow up fully.
- Place 50ml syringe on pigtail at top of membrane gently tap membrane and aspirate air from circuit ensuring head is kept below membrane.

- Leaving saline open, replace head in pump casing and restart pump flow, circulating through bridge.
- Check all pigtails, head and membrane.
- Place patient back on ECMO V-B-A
- Observe circuit for source of air.
- Document total length of clamp off period in specialist evaluation

When de-airing a Levitronix circuit it is imperative that you remember what ever fluid you take out of the circuit must be replaced back into the circuit otherwise you will cause air to be sucked though the oxygenator and the situation to worsen.

Trouble shooting Levitronix centrifugal pump

Pump failure

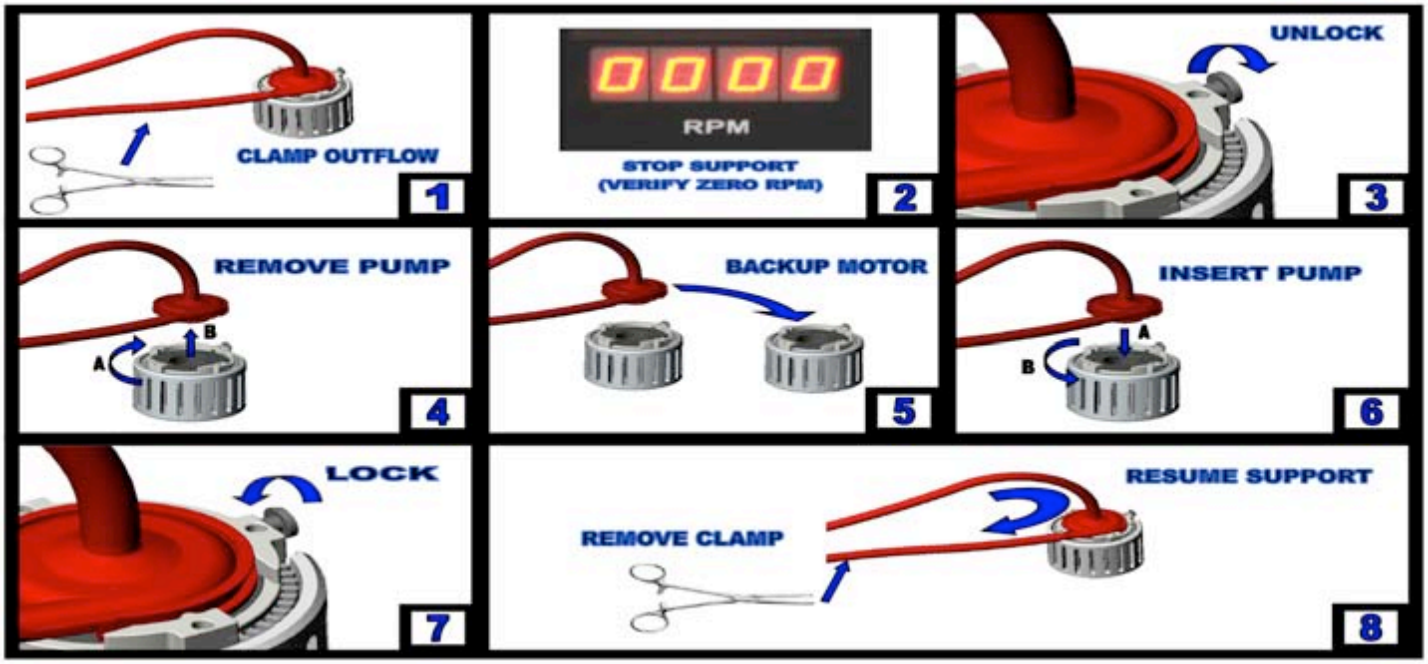
Pump failure from electrical failure or mechanical breakdown is rare. The Console is designed for operation on AC power; however, it also contains an internal rechargeable battery and charger. If a power failure causes loss of AC power or patient transport is necessary, a new fully charged internal battery will operate the Console and Pump for a minimum of 60 minutes at 5500 rpm, 3 Lpm. The switch from AC power to battery power is automatic and is accomplished without interruption of patient support.

BOTH THE LEVITRONIX CONSOLE MUST BE CONNECTED TO THE MAINS AT ALL TIMES, INCLUDING WHILST IN STORAGE.

As centrifugal pumps are unable to be hand-cranked (manually operated), then a spare

pump and console is maintained on every cart.

IF THE CONSOLE REPORTS A FAILURE, BOTH CONSOLE AND DRIVE MOTOR UNIT MUST BE CHANGED.



Pump failure action:

- Clamp Arterial tubing post pump. Depress red key on Centrimag console and hold for 2 seconds
- Turn on the backup console.
- Remove the pump head from the motor housing by loosening the locking screw. Rotate pump head clockwise and remove.
- Fit the pump head in to the backup drive motor unit, rotate counterclockwise and ensure the locking screw is in place and then tighten.
- Set the RPM on the backup console to 1500 and remove the clamp then adjust RPM to give flows at original pre emergency level. Blood flow and pre and post-oxygenator pressures can be read from original console till back-up flow probe sited.
- Fit the backup flow probe to the arterial tubing. Ensure arrow is facing in right direction
- Remove pressure monitoring cables from the back of the malfunctioning console and click into ports labelled P1 and P2 on backup console
- Access menu on backup console and ensure:
    1. Flow limit sensitivity set to normal.
    2. Pressure display - active
    3. Upper and lower flow limit alarms set:
        - Pt <10kg 50ml/min below current flow
        - Pt> 10kg 100ml/min below current flow.
    4. High and low alarm limits cannot be set within 0.3l/min of each other. High flow limits should therefore be set 0.3l/min above set low flow limits
    5. On AC power indicator illuminated.
    6. Battery level meter indicating battery fully charged
- Remove original malfunctioning console flow probe from the outlet tubing.
- Source replacement backup console and motor from perfusion.
- Re-zero P1 and P2 pressures on console (See Protocol)

Retrograde flow

Retrograde flow can occur at low RPM or during significant increases in BP and will cause acute clinical deterioration. Retrograde flow will be displayed on Levitronix console as - - - . If this is displayed immediately clamp tubing post pump head.

Action:

- Immediately clamp tubing post pump head
- Observe that flow probe is correctly placed on tubing
- Increase RPM by approximately 10%.
- Slowly release clamp observing for signs of forward flow.
- Once support has been achieved wean RPM to give desired flow rate.

Patient Complications

Patient complications can manifest as dysfunction in virtually all organ systems. ELSO reported an average of 1.44 patient complications per run. The incidence of patient complications is twice that of mechanical, but is reducing over time. Complications include surgical or cannula site bleeding, intracranial infarct or bleed, haemolysis, renal insufficiency requiring dialysis, hypertension, seizures, electrolyte abnormalities, pneumothorax, cardiac dysfunction or arrhythmias, gastrointestinal haemorrhage and infection. Despite advances in hemostatic agents such as tissue glues and sealants, bleeding remains the most devastating and difficult problem to manage.

Bleeding

Bleeding is the most common complication of ECLS, largely due to the requirement for systemic heparinisation. A small to moderate amount of bleeding is frequently seen at the neck cannulation site but can be minimized by liberal use of electrocautery at the time of cannulation. Cannula site bleeding is often related to small vessels in the wound and not the cannula insertion site its self. Neck wound bleeding can most often be managed by application of finger pressure at the site, management of the heparin infusion rate, decreasing the ACT, increasing the platelet count >150,000. If significant bleeding exists then exploration of the site maybe necessary.

Significant bleeding that is not attributable to the neck incision may represent a greater risk. A decrease in haemoglobin, tachycardia, hypotension or a rise in PaO2 on VA ECLS may suggest the presence of an acute bleed. Patients who have undergone invasive procedures are at an increased risk of bleeding. Procedures such as suprapubic bladder aspiration, paracentesis, lumbar puncture or insertion of vascular access whilst considered routine for non-ECLS patients pose significant risk to the patient on ECLS. When surgical procedures, such as diaphragmatic hernia repair is carried out on ECLS it is essential to optimise the patients clotting picture prior to surgery as well as post operatively and close attention to detail whilst managing their heparin and ACT's is required. See "bleeding bundle"

ELSO report the incidence of clinically significant Intra-Cranial Haemorrhage (ICH) as ≈7%. Factors contributing to the risk of ICH in critically ill patients include hypoxia, hypercapnia, acidosis, ischaemia, hypotension, sepsis, coagulopathy, thrombocytopenia, venous hypertension, seizures, and rapid infusion of colloid or hypertonic solutions. Additional factors during ECMO include ligation of the right common carotid artery and internal jugular vein, systemic heparinisation and systolic hypertension. Daily cranial USS's, if the fontanelle is patent, for the first 3 days followed by Mon, Wed and Fri allows early diagnosis. It also allows the ultrasonographers' to plan their weeks activity. A large bleed associated with profound clinical deterioration including flaccidity and fixed pupils will require an urgent neurological review and potential cessation of ECMO support on the grounds of futility. Smaller cerebral bleeds can

be managed with Aprotinin infusion, increased platelet parameters, tight management of pCO_2, decreased ACT's, serial cranial USS and early decannulation.

Renal Failure

Oliguria is common in ECLS especially in the first 24 to 48 hours. This may be attributable to two phenomenon. Patients will suffer some degree of capillary leak after cannulation and this may result in a decrease in renal perfusion and subsequent urine production until the leak resolves and/or normovolaemia is re-established. Studies on the renal effects of short-term bypass attribute this oliguria to perfusion of the renal glomerulus with pressures outside the normal auto regulatory range. In the presence of elevated creatinine or persistently poor response to IV diuretics, ultrasound of the kidneys, including Doppler signal analysis, is recommended to exclude major anatomic anomalies or flow abnormalities. There is now widespread use of continuous haemofiltration (CVVHDF) in patients supported on ECMO, the haemofiltration is easily added into the circuit and allows the removal of excess volume. Peritoneal dialysis or Slow Continuous Ultrafiltration (SCUF) can also be used. Albumin should be optimised prior to commencement of SCUF.

Cardio-pulmonary complications

Systemic hypertension is a potentially dangerous side effect of ECMO. A significant number of neonatal patients treated with VA ECMO develop a systolic blood pressure >90mmHg, increasing the risk of ICH. Before anti-hypertensive medication is administered to patients they should be assessed for pain, hypercarbia and hypoxia, since all can cause hypertension. It is important to assess any catastrophic haemodynamic deterioration whilst on VA ECMO carefully but factors such as cannula placement, adequacy of systemic volume status and the possibility of circuit failure need to be ruled out quickly.

Major cardiac dysfunction is generally not appreciated on VA ECMO when full flow (120ml/kg/min) can be achieved. In infants some degree of cardiac depression is common early in the ECMO run particularly in the more severely asphyxiated. Myocardial stun is defined as left ventricular shortening fraction (LVSF) decreasing by >25% after commencement of ECLS and returning to normal after 48 hours on ECMO, this occurs despite relief of hypoxia. Impaired filling of the coronary arteries and persistent subendocardial ischemia during the early high flow phases may precipitate the lower LVSF. ECMO can adequately unload the right heart but it also increases the afterload of the left ventricle. During stun VA ECMO supports the cardiac output without hindering the recovery of the myocardium. The syndrome of myocardial stun includes narrow pulse pressure and equalization of circuit and patient gases, an Echo will show decreased myocardial contractility.

If a patient suffers a cardiac arrest, develops a dysrhythmia or drops his cardiac output secondary to severe myocardial dysfunction the initial treatment choice on VA ECMO is to increase the RPM therefore increasing the flow, the cause of the acute event can then be treated. If the pump flow cannot be increased sufficiently to compensate for the fall in intrinsic cardiac output, or for patients on VV ECMO then the episode should be treated the same as a cardiac arrest in any other patient. It is important to remember that the most common cause of cardiac dysfunction in neonates is hypoxia from respiratory not cardiac failure. Extreme acid-base imbalance may be secondary to hypoxia caused by for example the tubing to the gas inlet port falling off or hypovolaemia are just a few examples of how circuit problems can precipitate such an event, the goal is always to restore normal function in the fastest and least traumatic manner as possible. If the potential problems outlined above have been eliminated then you should consider intrathoracic complications such as pericardial tamponade, tension haemothorax or pneumothorax.

CPR in VV-ECMO

- This has inherent dangers in a fully heparinised patient with large cannulae in the heart which may become dislodged or entrain air.
- One Nurse watches circuit for air entrainment and looks after pump (ensure emergency box to manage gross air is at hand).
- Do not clamp off ECMO unless air entrained during CPR.
- Switch on back-up pump in case of unanticipated pump failure.
- Consider pacing.
- Inform cardiac / surgical consultant if CPR on-going or resolved +/- perfusion.
- Be aware that significant increase in afterload secondary to high dose adrenaline will increase the resistance that the pump has to work against therefore rpm may need to be increased significantly until this has worn off.

Defibrillation

- This can be safely done on ECMO
- The patient's haemodynamic stability should be carefully monitored during this time.
- Ensure rpm and flow of ECMO return to baseline
- Back-up pump may be required in the event of unanticipated pump failure

Tamponade/Haemothorax/Pneumothorax

Pericardial tamponade and tension haemothorax and/or pneumothorax show a similar pathophysiology of increasing intrapericardial pressure and decreasing venous return. With decreased venous return to the heart, pulmonary blood flow is decreased, cardiac output is decreased and peripheral perfusion is decreased. Peripheral perfusion is initially maintained by the non-pulsatile flow of the VA ECLS circuit. A PaO2 measurement will increase while the patient will have decreased peripheral perfusion with a decreased pulse pressure and decreased SvO2. The decreased SvO2 in VA ECMO confirms the decreased oxygen delivery of the circuit and further deterioration of the patient.

A. VA-ECMO

Recognise tamponade:

- Increased HR
- Increased CVP
- Increased Pao2
- Decreased Svo2
- Decreasing perfusion
- Decreased pulse pressure
- Late sign decreasing BP
- Inlet venous pressure reading more negative

Manage tamponade:

- Maintain flows, may need volume
- Call for help early
    - ECHO, Surgeon, ECMO Physician
    - Sedate & paralyse
- Check drain losses
- Mx of chest drains / swabs / dressings, open and closed chest
- Correct coagulopathy
- Discuss Aprotinin

- Discuss other Mx re bleeding patient on ECMO
    - Platelets / FFP / Cryo / Red heparin / Novo 7 / surgical intervention
- May require pericardial drain or thoracic exploration +/- repair of atrial trauma

B. VV-ECMO

Recognise tamponade:

- Increased HR
- Increased CVP
- Decreased Pao2
- Increased Svo2
- Decreasing perfusion
- Decreased pulse pressure
- Late sign, decreasing BP
- No problem with ECMO drainage

Manage tamponade

- Support cardiac output, volume, inotropes, emergency drugs
- Give all volume to patient
- Call for help early
- Emergency ECHO
- Drain tamponade with angiocath under ultrasound guidance
- Discuss Aprotinin
- Discuss other Mx re bleeding patient on ECMO
    - Platelets / FFP / Cryo / Red heparin / Novo 7 / surgical intervention

Patients with a tension pneumothorax will have a similar pathophysiology of increasing Intracardiac pressure and decreasing venous return. Diagnosis is best confirmed by a cold light or chest x-ray. Where haemodynamic instability is absent pneumothorax can be managed by a period of CPAP, acute instability will necessitate emergency drainage which should be undertaken with caution due to the risk of bleeding. **Chest drains should only be sited using imaging by the surgical team with the use of electrocautery to minimise bleeding complications.**

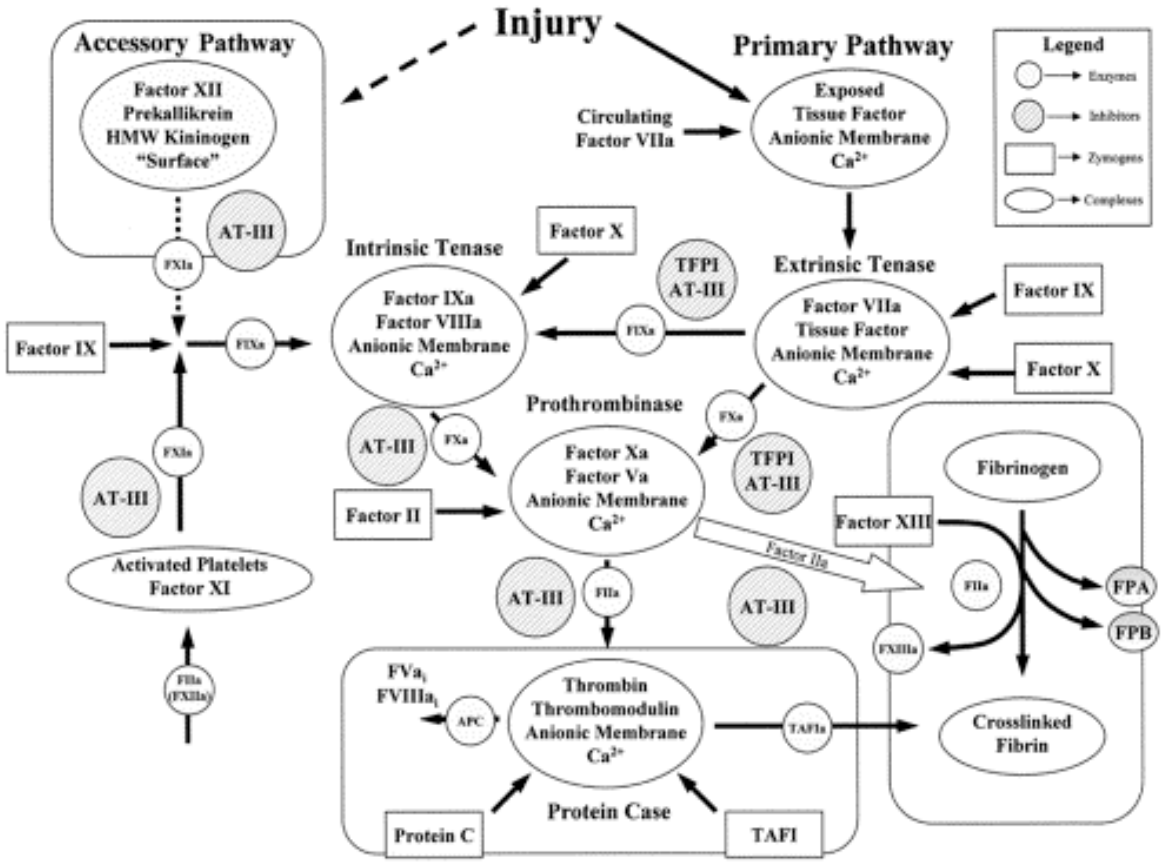
Sepsis

See Microbiology section

Coagulation

Anticoagulation is an essential component of extracorporeal membrane oxygenation (ECLS) management due to the thrombogenic effects of circuit components. Close monitoring, most commonly by the activated clotting time (ACT), is paramount to minimise the risk of haemorrhage.

Never site or remove any line or drain without discussing with ECMO physician because of risk of haemorrhage whilst patient supported on ECMO.



Overview of haemostasis (Wintrobe's Clinical Hematology)

Physiology of Coagulation

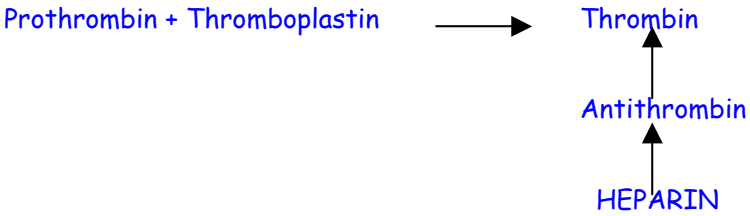
Plasma contains prothrombin, while thromboplastin is liberated from injured tissues and degenerated blood platelets. The clotting process starts by the calcium-dependent reaction between these two hormones to produce thrombin:

Prothrombin + Thromboplastin + Ca^{++} = Thrombin

Fibrinogen is a plasma protein upon which thrombin acts to form insoluble fibrin:

Thrombin + Fibrinogen = Fibrin

Heparin is produced by the mast cells of the liver and basophil leukocytes. It inhibits coagulation by preventing conversion of prothrombin to thrombin, by acting on antithrombin III (AT-III), the natural intrinsic anti-coagulation factor.



Heparin is destroyed in tissues except the lung and liver, and is excreted by the kidneys. Commercial heparin is extracted from porcine intestine or beef lung and may vary in its activity.

Blood Surface Interactions

ECLS involves exposing a majority of the blood volume to a large artificial surface each minute. Even with the unusual flow patterns that the circuit produces and the effect of the pump, the damage to blood cell components is small. Platelets show the greatest effect of prolonged surface interaction leading to continuous platelet aggregate formation and decreasing blood platelet count.

Within seconds after going on ECLS, there is a molecular layer of protein composed of fibrinogen, albumin, alpha globulin, gamma globulin, Hageman factor (factor XII), and others, which becomes adherent to the prosthetic surface. Hageman factor reacting with a foreign surface would turn on the clotting cascade if not for the presence of heparin. If the circuit is pretreated with a single protein such as albumin, the binding of other proteins such as Hageman factor can by decreased.

Once all the active sites on the prosthetic surface are covered by a protein monolayer, the flowing blood "sees" a protein-water-electrolyte layer. Further adherence of protein does not occur, and this layer does not increase in size.

The nature of the proteins that are adsorbed on the surface affects the other blood products, particularly platelets which adhere to fibrinogen. A single platelet will adhere first and after platelet distortion, with release of platelet activating agents such as serotonin and adenosine diphosphate, comes further attachment of other platelets resulting in a platelet aggregate. These aggregates can be washed off into the patient. Even in the presence of heparin, fibrinogen is converted to fibrin, so that the final result is a smooth fibrin-platelet white thrombus which is firmly adhered to the surface and results in smoothing of the flow patterns at such areas as where connectors and tubing come into contact. The mechanical damage caused by the pump probably enhances platelet aggregation. These platelet aggregates do not lodge in capillary beds, but are taken up by the liver and spleen. There appears to be recirculation of these platelets, and in fact, if allowed, the drop in platelets would level off at 30,000-40,000 $x10^6$/L. Platelet counts are maintained at between 100,000-200,000 $x10^6$/L depending on the individual patients case. Infants with bleeding tendencies should have their platelet count kept about 120,000 $x10^6$/L.

Heparin

Heparin is administered continuously in the ECLS circuit to avoid clotting anywhere inside the tubing surface. Heparin will prolong the clotting time of whole blood by blocking the clot formation at various sites in the normal coagulation system, notably that of prothrombin to thrombin and fibrinogen to fibrin.

By inhibiting the final clot, blood can freely circulate within the ECLS circuit. Excessive heparin administration can inhibit the clotting cascade such that the patient may haemorrhage. The site of haemorrhage will vary with the individual patient but may include pulmonary, gastric, cerebral, cannulation site and any other surgical sites.

It is just as important to maintain the clotting time at a level that will avoid thrombosis formation in any portion of the ECLS circuit. Heparin is non-fibrinolytic and therefore will not lyse any clots that form in the ECLS circuit.

Management of Heparin

The normal accepted neonatal range for an ACT using the Haemochron Elite is 130-190 seconds. This value may be greater if the patient has a coagulopathy, a heparin infusion, or is septic. (**Note:** normal values for sick infants have not been established). This baseline ACT may be elevated due to heparin in the arterial line. In order to obtain the most accurate ACT, draw back 2-3 ml's of blood before obtaining your ACT sample if sampling from patient.

Cannulation bolus

During cannulation, after the initial incision is made and the blood vessels are isolated by the surgical team, the bolus of heparin is given to rapidly achieve a systemically anti-coagulated state. (ACT's >350 sec).

Depending on the patient's estimated risk for bleeding, the heparin bolus for cannulation is 50 units/kg.

An ACT may be obtained when the venous (2nd) catheter is placed while going on VA ECLS (time scale usually too short with DLVV ECLS). If the patient is not on bypass within 20 minutes of the initial bolus, an ACT must be drawn and an additional 25-50 units/Kg heparin can be given if the ACT is <300 sec.

Neonatal & Paediatric respiratory cases

Once on bypass, a maintenance heparin infusion of 50 units/kg/hr is started if drain losses are <5ml/kg/hr (see Protocol Procedure section). The maintenance infusion will vary from patient to patient and from hour to hour on the same patient, to maintain the ACT within the acceptable range to prevent clotting. Inconsistency in ACT measurement technique can alter the ACT results and inconsistent results should be double checked. (See ACT measuring protocol).

The heparin infusion is begun when the patient is connected to the ECLS circuit. If the ACT is less than 300 seconds, the heparin infusion is started at 2 ml/hr (50 iu/kg/hr). This may appear to be a fast infusion rate for such a high ACT, but you must remember that the blood in the ECLS circuit has very little heparin in it, consequently the ACT will fall rapidly once the heparinised patient and non-heparinised circuit blood mix. With this mixing, at a heparin infusion rate of 2 ml/hr the patient's ACT will usually stabilise very close to the set parameter.

Standard ACT ranges for centrifugal ECMO or VAD circuit:

- ***Low risk of bleeding***
  - ***ACT's 200-220 sec***
    - ***When Pedivas circuit & flow <300 ml/min: run ACT's 210-230 sec***
    - ***When Centrimag circuit & flows < 750ml/min run ACT's 210-230 sec***
- ***High risk or active bleeding***
  - ***ACT's 180-200 sec***

Paediatric cardiac cases

A common reason for ECLS post-cardiac surgery is if the patient fails to successfully wean from cardio-pulmonary bypass. In this instance the patient will be fully anti-coagulated already with ACT's at very high levels (250-400 secs) therefore a bolus of heparin is not required. The patient will be cannulated in cardiac theatre via the transthoracic route and then taken to PICU. Bleeding in these patients can be a significant problem and for this reason the Heparin may be partially reversed with Protamine by the Anaesthetist if required. It would be standard practice to only start the heparin infusion on these patients once the bleeding reduces to less than 5ml/kg/hr. It may be appropriate to start the infusion at 25iu/kg/hr. This will be decided by the treating Intensivist in conjunction with Perfusion and Cardiothoracic Surgical colleagues. It will often be necessary in these patients to ensure the platelet levels are kept higher, up to 150, if there is ongoing bleeding.

If the patient is cannulated in PICU following cardiac surgery then a Heparin bolus of 50iu/kg should be given but again the heparin infusion only started when the drain losses are less than 5ml/kg/hr. An infusion rate of 25iu/kg/hr of heparin is appropriate. This may be undertaken via the transthoracic or neck routes.

Active bleeding

If active bleeding see "Bleeding patient".

Patient management

As soon as the patient is placed on ECLS, it is important to be sure that any non-ECLS personnel (nurses who are assigned to the baby) understand that the baby is systemically heparinised. No intramuscular medications, venepunctures or heelstick blood work may be done. Nasal suctioning should be avoided. Blood sampling should if possible be taken from patient's arterial line or if checking "circuit bloods" then take from ECMO circuit. Medication administration should be **undertaken through peripheral or central venous access if possible**. (Exceptions: oral medications are given via the OG tube; medications that are inactivated in heparin are given via a pre-existing peripheral IV or UAC.)

If the patient's blood is exposed to the untreated ECLS surface for a period of time, fibrin formation may occur. The time required for the patient to metabolize the initial dose of heparin is variable. For this reason, an ACT should be done within minutes of going on bypass and then every half hour until the ACTs are stable. Abrupt changes in the heparin rate will produce a 'roller coaster' effect on the ACT's and should be avoided.

NOTE: Any emergency requiring coming off the circuit requires close observation of ACT's from the patient. The catheters are in the patient with stagnant blood. ACT's should be increased to 220-240

seconds during this period.

Without a severe coagulopathy, a stable series of ACTs can usually be obtained without radical adjustments in heparin infusion rates. However, as the platelet count drops (due to platelet adherence in the oxygenator), the effects of the heparin maintenance dose may diminish. Platelets should be administered when the platelet count drops below 100,000 x106/L. If the patient is bleeding, platelets are transfused when the level falls below the set parameter. After giving platelets, the heparin requirement will usually increase. If the patient has been on ECLS long enough to have already received blood products, it is best to evaluate the previous response.

Heparin dosing during blood product administration

The ACT should be checked more frequently during and immediately after blood product administration (at least every half hour). The amount of the increase in the heparin infusion will vary with the individual and the previous requirements of that infant. A fair amount of judgment and experience is helpful. If the ACT should fall below 180 seconds, or drops rapidly, a bolus of heparin (10-20 Units/kg) may be necessary along with an increase in infusion rate. Again, judgment is necessary to avoid the roller coaster effect. The previous hourly heparin requirements, the coagulation studies, the rate of fall of the ACT's, last blood transfusion, etc., will affect the dose of heparin that will be adequate, but not excessive. Serial ACT's until stable are the best index of adequate heparin dosage. It is rare that the heparin requirement changes by more than 30 units/kg in one hour. Remember that once the platelet transfusion is complete, the heparin requirement may suddenly drop to the previous rate.

Administration of red blood cells or fresh frozen plasma may have a transient effect on the ACT's but will not usually be as dramatic as platelet administration. When platelets are given increase the Heparin infusion by 2.5 - 5iu/kg/hr to allow for the increased clotting risk unless the patient is actively bleeding.

Aberrant ACT recordings

ACT's will often be high at the start of the ECMO run following the heparin bolus. If for some reason after the first 4-6 hours following cannulation the ACT is greater than 300 seconds, the heparin infusion should be continued at a very low rate - 2.5U/kg/hr (0.l ml/hr) - until a normal value is achieved. If the heparin infusion is stopped altogether, minor clotting may occur. Check an ACT at least every 30 minutes.

Another factor to consider when assessing heparin requirement is the urine output. Heparin is excreted by the kidneys. A baby with poor renal function will most likely have a decreased heparin requirement (< 50 U/kg/hr). Conversely, if the patient has a diuresis due to frusemide or improved renal perfusion, the heparin requirements may rapidly increase.

Abnormal coagulation studies, particularly the presence of fibrin degradation products (FDP's), will affect the heparin requirements. The ACT result relies on a solid fibrin clot forming over time. If there are FDP's and therefore a poorly solidified clot, the machine will have a difficult time determining the end point of clotting. Any ACT over 400 seconds should be removed from the machine and checked for validity. If a heparin overdose is suspected, the specialist must call the ECLS physician.

If the ECLS patient is at great risk for haemorrhage Aprotinin may be used. Aprotinin (Trasylol) is an

antifibrinolytic that acts through inhibition of plasmin and both plasma and tissue kallikrein. Improved haemostasis has been demonstrated with its use in patients on cardiopulmonary bypass (CPB). It is only available on a named patient basis.

If the patient requires surgery on ECLS, as with the CDH patient, Aprotinin may be used. Fibrinogen levels should also be kept at >2.0 g/L. This can be accomplished through transfusions of cryoprecipitate. Platelets can also be increased to > 150,000

Routine coagulation investigations

- Hourly ACT
- 8hrly APTT, Fibrinogen, Platelets
- Daily anti-thombin, Anti-Xa and D-Dimers
- ROTEM if bleeding concerns

- If ACT >260 repeat ACT in 15-30mins and consider dropping heparin ivi by 5iu/kg/hr
- If concern re bleeding repeat ROTEM, aPTT, Fibrinogen, FBC

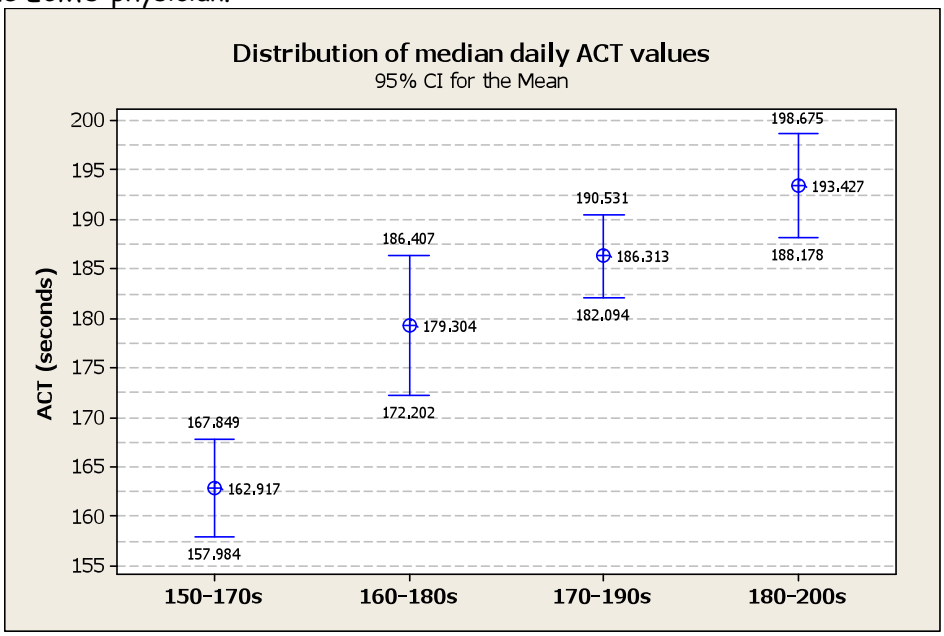
Activated clotting time (ACT)

Near patient monitoring of the anticoagulation of ECMO patients is undertaken using the ACT at RHSC, Glasgow.

Guidance on undertaking an ACT is shown in the guideline section towards the rear of the manual.

At the initiation of ECMO it is essential to check an ACT at least every 30 minutes until there is stability and only then can the ACT be checked every hour.

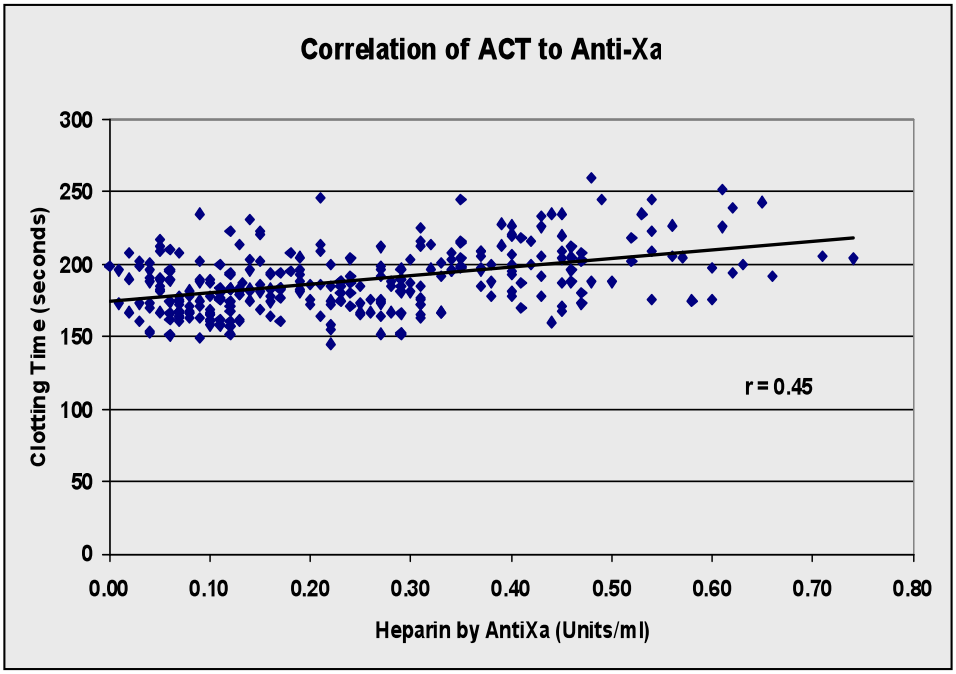
Recent local work has shown that the ACT's obtained by the specialists (using the old ACT machine which reads 40 secs lower than the current Haemochron Elite) at RHSC, Glasgow correlates exceedingly well with prescribed target range (see table below). It should be noted that the median level tends towards the lower target range and this should be borne in mind when the daily orders are written by the ECMO physician.



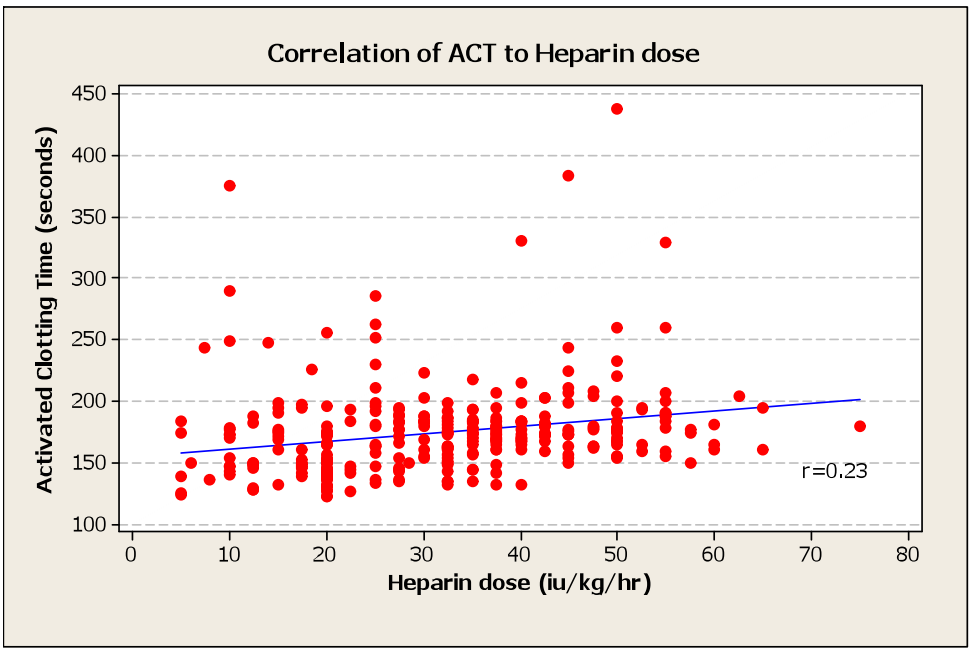
Anticoagulation practices during paediatric extracorporeal membrane oxygenation: a single institution's experience ***Veer Shah, Gillian Wyllie, Mark Davidson***

As one of the most common complications of ECMO is haemorrhage it is essential that high ACT's are acted on appropriately. Further recent local studies have shown that over a one year period there were 109 episodes where an ACT of greater than 220 was recorded outwith the cannulation and decannulation periods. In only 11% of the cases was an ACT rechecked prior to the next hour. In 18.5% cases the heparin level was not adjusted. It is essential that if an ACT of greater than 260 is recorded that unless there is clinical concern or it is round the time of cannulation when the ACT is still falling following the initial heparin bolus that a follow-up ACT is checked 15 to 30 minutes after the high level. The heparin infusion should also be reduced by 5iu/kg/hr again unless there is clinical concern or it is round the time of cannulation when the ACT is still falling following the initial heparin bolus. If there is active bleeding it may be appropriate to reduce the heparin further than by 5iu/kg/hr and this should be discussed with the ECMO physician.

The ACT allows the trend analysis of the anticoagulation effect of heparin. It assesses the intrinsic and common pathways of coagulation. It should be remembered that the ACT is highly sensitive to many factors including platelet and coagulation factor levels, level of diuresis, hypothermia, protamine administration (reverses heparin), anti-thrombin levels. It has a poor correlation to the gold standard of heparin management, the factor anti-Xa level (see graph below).



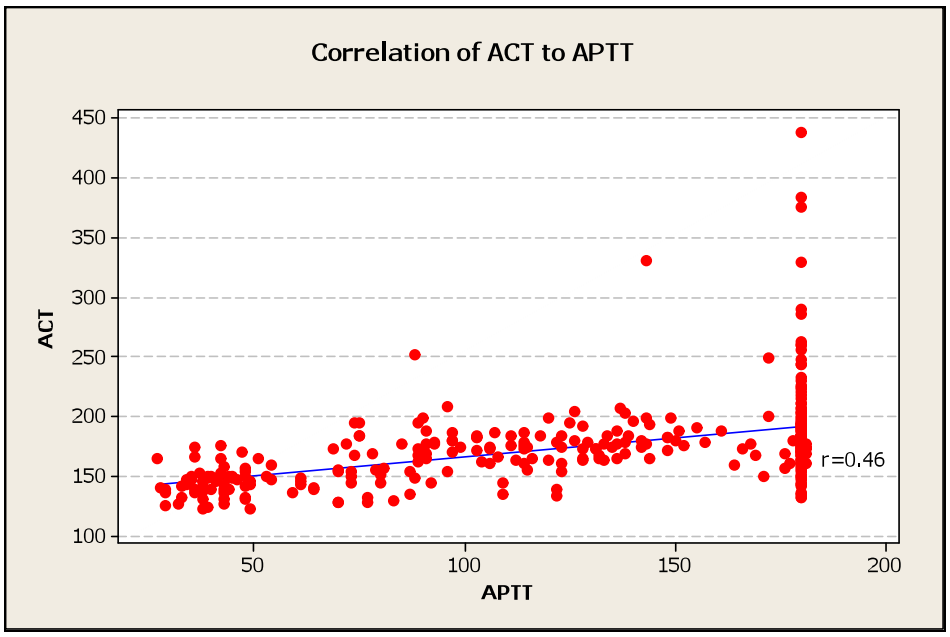
Heparin monitoring during paediatric ECMO
Walker A, Kasem K, Allen G, Rafferty I, Davis C, Davidson MG, Chalmers EA



Anticoagulation practices during paediatric extracorporeal membrane oxygenation: a single institution's experience ***Veer Shah, Gillian Wyllie, Mark Davidson***

Activated artial thromboplastin time (aPTT)

The aPTT is used by some centres to guide anti-coagulation, more commonly on those managed with longer term cardiac support such as the Berlin Heart or similar long term ventricular assist devices. It is checked in the routine battery of tests in our ECMO patients and can be useful if the ACT is not in keeping with the picture seen clinically. There is a poor correlation (r=0.23) between ACT and aPTT as seen in the graph below, from local work. Direct heparin management should be driven by the ACT trend.



Anticoagulation practices during paediatric extracorporeal membrane oxygenation: a single institution's experience ***Veer Shah, Gillian Wyllie, Mark Davidson***

Thromboelastometry (ROTEM)

The management of acute bleeding in the patient receiving ECLS can be a major challenge. This is highlighted in the post operative cardiac patient, where it can be difficult to distinguish between a surgical bleeding point and deranged coagulation. In the cardiac subpopulation where cannulation has occurred through a sternotomy it is desirable to minimise re-exploration whenever possible to reduce the risk of infection and perpetuating generalised ooze by recurrent exploration, whilst avoiding treating a surgical bleeding point conservatively.

The clinical significance of routine laboratory tests, PT APTT, platelet count, near patient testing ACT and the less often requested Heparin assay is debatable, as they don't assess the clotting process holistically. There can be an associated delay between specimen retrieval and result availability. Consequently blind therapy with blood products occurs with inappropriate overuse of blood products and the potential delay in addressing a surgical lesion as the cause of bleeding. There is a cost and safety implication with the incorrect use of blood products.

Thromboelastography/ thromboelastometry can theoretically address some of these issues by looking at the clotting process holistically, in real time, from formation of the initial fibrin strands through to clot lysis. Of note the limitations of the test have to be realised. It is insensitive to the effects of platelet inhibitors ie Aspirin and Clopidogrel, and specifically it is unable to detect Von Willebrand Factor deficiency. A normal TEG does not exclude the effects of Warfarin or Low Molecular weight heparin.

We use the ROTEM system. The pin is rotated, as clot forms this rotational movement becomes increasingly restricted, as such movement is a function of clot firmness. Movement is detected optically and the thromboelastogram and its numerical parameters are calculated by an integrated computer (Fig. 1).

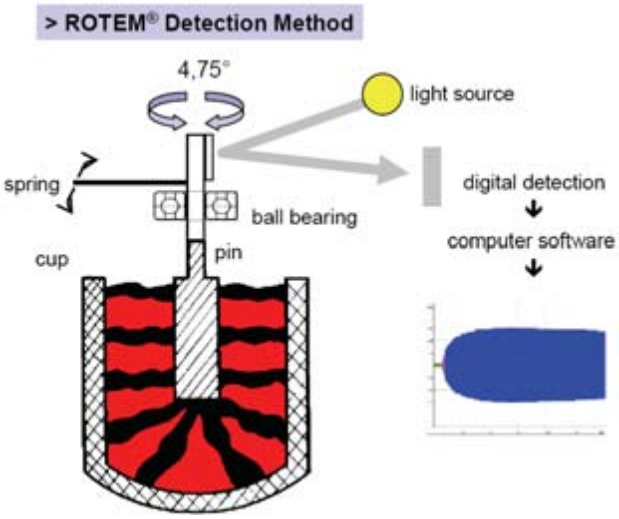


Fig. 1

The derived parameters are the clotting time, clot formation time, maximum clot firmness and maximum lysis. Their graphical derivation is shown in Fig.2

Work we have undertaken on the ECMO patients who have had ROTEM's undertaken shows that the amplitude of the clot at 10 mins or A10 correlates very well with the Maximum Clot Firmness (MCF). This means that clinically meaningful result interpretation can be gained approximately 15 minutes after taking the sample and targeted blood products can be ordered or a discussion with the surgeon about going back to chase a bleeding point can be undertaken.

CT (clotting time): time from start of the measurement until initiation of clotting → **initiation of clotting, thrombin formation, start of clot polymerisation**

CFT (clot formation time): time from initiation of clotting until a clot firmness of 20 mm is detected → **fibrin polymerisation, stabilisation of the clot with thrombocytes and FXIII**

MCF (maximum clot firmness): firmness of the clot → **increasing stabilisation of the clot by the polymerised fibrin, thrombocytes as well as FXIII**

ML (maximum lysis): reduction of clot firmness after MCF in relation to MCF → **stability of the clot (ML< 15%) or fibrinolysis (ML > 15% within 1h)**

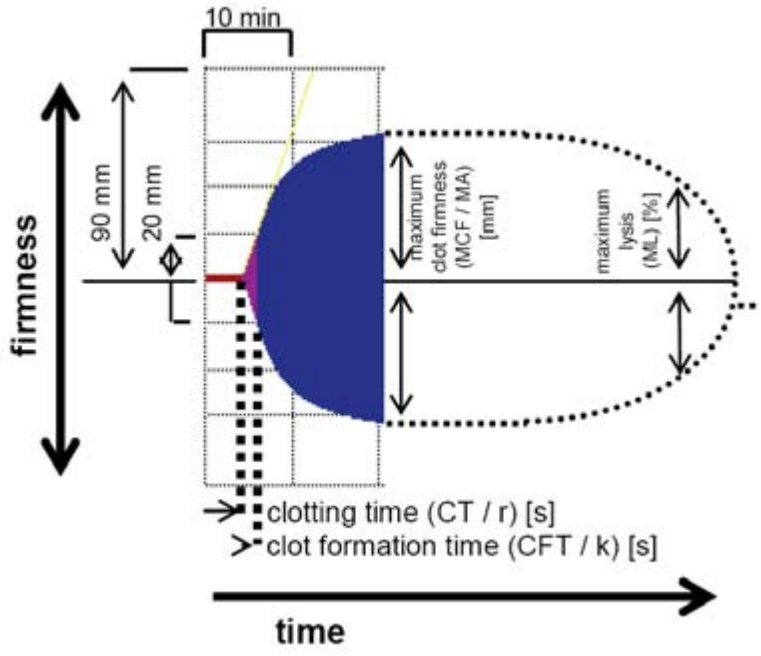


Fig.2

In addition activators or inhibitors can be added to focus on different aspects of haemostasis.

- **EXTEM**; tissue factor is added allowing assessment of clot formation. Factors I,II,V,VII,X, platelets and fibrinolysis.
- **INTEM**; coagulation is activated by the contact phase (similar to ACT aPTT), thus assessing deficiencies in the intrinsic system and the presence of Heparin.
  Fcators:I,II,V,VIII,IX,X,XI,XII, platelets, fibrinolysis.
- **FIBTEM**; platelets are blocked , so clot formation is dependant on fibrin formation and polymeristaion
- **APTEM**; platelets are blocked and the fibrinolytic process is inhibited, thus comparing FIBTEM and APTEM allows assessment of fibrinolysis, and APTEM will suggest whether Fibrinogen will help
- **HEPTEM** is the same as INTEM, but heaprinase is added , negating the effects of heparin, thus permitting analysis analysis in heparinised patients.

Whilst there are normal values, theses have not been validated in paediatrics. To start with we will not be using the data from the TEG to guide therapy. Rather we wuill be collecting data in conjunction with ACT's a, Heparin Assays, P, Aptt, Fibrinogen and Antithrombin levels.

The algorithm in Fig. 3 shows how coagulation activation, clot formation and fibrinolysis are assessed from EXTEM and INTEM.

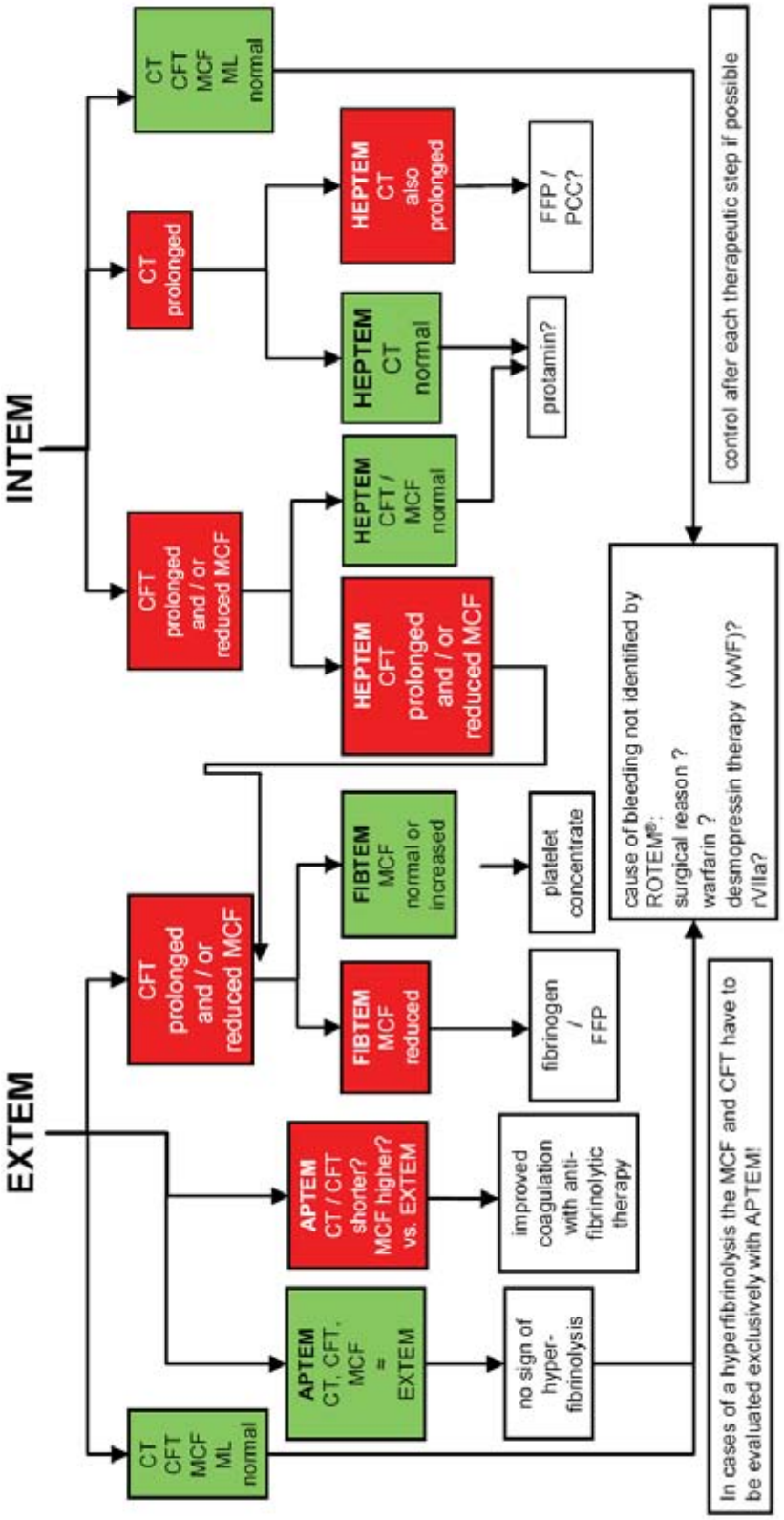

INTEM
EXTEM
CT CFT MCF ML normal
CT prolonged
CFT prolonged and / or reduced MCF
HEPTEM CT also prolonged
HEPTEM CT normal
HEPTEM CFT / MCF normal
HEPTEM CFT prolonged and / or reduced MCF
FFP / PCC?
protamin?
control after each therapeutic step if possible
CFT prolonged and / or reduced MCF
FIBTEM MCF normal or increased
FIBTEM MCF reduced
platelet concentrate
fibrinogen / FFP
APTEM CT / CFT shorter? MCF higher? vs. EXTEM
APTEM CT, CFT, MCF ≈ EXTEM
improved coagulation with anti-fibrinolytic therapy
no sign of hyper-fibrinolysis
CT CFT MCF ML normal
cause of bleeding not identified by ROTEM®:
surgical reason ?
warfarin ?
desmopressin therapy (vWF)?
rVIIa?
In cases of a hyperfibrinolysis the MCF and CFT have to be evaluated exclusively with APTEM!

Fig. 3

Aprotinin (Trasylol)

What is Aprotinin?

Aprotinin is a naturally occurring polypeptide consisting of 58 amino acids. It is extracted from bovine lung.

How does Aprotinin work?

Aprotinin inhibits a wide range of serine proteases but in particular trypsin, plasmin and kallikrein. There are two mechanisms that have been suggested for how Aprotinin reduces blood loss:

- Stabilisation of fibrin clots – by inhibition of proteases involved in coagulation and fibrinolysis such as kallikrein and tissue plasmin.
- Preservation of platelet function – possibly by inhibiting the activation of platelets by artificial surfaces.

What are the pharmokinetics of Aprotinin?

It is inactivated after oral administration and therefore needs to be given intravenously as a continuous infusion. It is cleared rapidly after intravenous administration and accumulates in the kidney for metabolism. It is metabolised in the kidney lysosomes to small peptides and amino acids and excreted in the urine.

What are the potential benefits of Aprotinin?

Efficacy has been monitored by assessing the cumulative blood loss in chest drains or by monitoring the number of units of blood transfused. Studies have shown a marked reduction in blood loss for patients.

Who may need Aprotinin?

- Post-operative patients who may have a high risk of bleeding (occasionally in cardiac patients with tenuous coronary circulation, it may be omitted)
- Patients with active bleeding
- Patients with high risk of bleeding – usually high risk neonates

What is the dose of Aprotinin?

Aprotinin is given as a continuous infusion of 1ml/kg/hr (10,000 IU/kg/hr) after an initial loading dose of 1ml/kg given over 20 minutes. This is a relatively large volume load for patients. The dose may be reduced if/when the bleeding risk recedes later in the run.

Novo7 (Recombinant factor VIIa/rFVIIa)

Guideline for use (see also in guideline section)

Recombinant FVIIa (rFVIIa) has proven efficacy and is licensed for use to control or prevent bleeding in haemophilia patients with inhibitors who are unresponsive to conventional Factor VIII or IX replacement, and also patients with congenital FVII deficiency or Glanzmann's Thrombasthenia.

Increasing evidence from case reports and case series indicate its potential usefulness in non haemophilia patients with intractable bleeding, or even as prophylaxis prior to high blood-loss surgery. rFVIIa is at present, unlicensed for such indications and is expensive. Nevertheless there may be a place for considering its use to control bleeding in certain situations when other measures have failed.

A few case reports describe the successful use of rFVIIa to achieve haemostasis in severe refractory bleeding after cardiac surgery in adult and paediatric patients. There is no published data on rFVIIa administration in ECLS patients although several UK centres have used it in a small number of patients (see chapter 38,"ECLS Red Book"). The mechanism of action of rFVIIa probably relates to a supra-physiological surge of thrombin generation. The main concern with this type of procoagulant therapy in ECLS patients is the concomitant risk of thrombus formation. A primed circuit should be available as emergency back up in case of significant circuit clotting.

Prior to considering treatment with rFVIIa it is important that every effort is made to correct deficiencies of platelets and coagulation factors by administering appropriate platelets, plasma and cryoprecipitate.

How to obtain rFVIIa

All patients being considered for rFVIIa treatment must be discussed with the on call haematology consultant.

Dosing and administration

An initial dose of 95 mcg/kg rFVIIa by slow IV bolus may be repeated after 1-2 hours if there is still significant blood loss. Further doses of 95 mcg/kg can be considered if bleeding decreases but does not stop. These should be administered at 2-3 hourly intervals. rFVIIa may be administered peripherally or via a central venous line.

rFVIIa is dissolved in the accompanying solvent before use (see Guideline). If the complete vial is not used the remaining solution may be kept for up to 6 hours in the fridge and used for subsequent doses. As there is a risk of bacterial contamination the vial must be prepared aseptically.

Efficacy

The primary measure of haemostatic efficacy is the clinical assessment of bleeding rate (i.e. stopped, decreased or unchanged). Clinical benefit is often seen within 10-20 minutes of rFVIIa administration. A reduction in blood and blood products use is a reasonable secondary measure of haemostatic efficacy.

Monitoring

The following clinical variables should be monitored in any patient receiving rFVIIa:

The rate of blood loss e.g. volume in drains, rate of drop in Hb or rate of packed cell requirements.

Laboratory investigations

Prior to administration of rFVIIa:

- FBC
- Coagulation screen (APTT, fibrinogen and D-dimer)
- TEG/ROTEM

The efficacy of rFVIIa relies on the presence, *in vivo*, of at least modest coagulation factor levels. Therefore appropriate amounts of FFP/platelets and cryoprecipitate should already have been administered.

20 Mins following administration of rFVIIa:

- FBC
- Coagulation screen (APTT, fibrinogen and D-dimer)
- TEG
- ACT

The PT will usually have shortened dramatically, however this may be affected by the concomitant administration of anticoagulant medication. rFVIIa has a short half-life and, therefore, the effects on the coagulation screen may not be long lasting and further coagulation monitoring will be required appropriate to the clinical situation.

Record of rFVIIa administration

As with all blood/coagulation products, details of dose and batch number should be recorded in the case notes. Details of other blood and blood products administered, results of FBC and coagulation investigations as well as a comment on apparent clinical efficacy of the rFVIIa should be recorded in the case notes or clinical information system.

Off label use of rFVIIa is the subject of an ongoing national audit. An audit questionnaire will be sent following release of rFVIIa for unlicensed use and should be completed and returned to haematology.

ECLS precautions

As there is a potential risk that the ECLS circuit may clot after rFVIIa administration a spare circuit, primed to plasmalyte should be available. Most patients will not require perfusion to be notified or present on site, as long as a plasmalyte primed circuit is available. Exceptions to this are patients given Novo7:

- Who are running heparin free
- Who are being managed with an ACT consistently <180

Anti-thrombin

Anti-thrombin (AT) is a naturally occurring serine protease inhibitor in the blood which is the primary inhibitor of thrombin. It also inactivates other coagulation factors such as factors Xa, IXa and XIa (see figure 1). AT irreversibly binds with thrombin to form stable thrombin-antithrombin complexes (TAT). This is a process greatly potentiated by heparin, and is the mechanism through which UFH exerts its anticoagulant effect. Heparin requires near normal levels of AT in order to function optimally. Normal serum levels of AT vary with age as shown in table 1. These levels were measured in healthy children undergoing elective blood tests and not in the cohort of patients seen in PICU. A study of coagulation markers of patients requiring PICU [10] showed lower levels of AT than the accepted reference ranges in table 2, however there was no separation of those supported with ECMO or post-cardiopulmonary bypass from those not in these groups making more detailed analysis difficult.

Consumption of AT during ECMO may lead to low levels of AT which can increase the risks of thromboembolism. The administration of AT in pediatric ECMO has been proposed. However a recent paper in which a sub-group of 15 PICU patients were supplemented with a dose of AT, did not show any significant effect on Anti-Xa or Endogenous Thrombin Potential (ETP) levels despite significant increases in serum AT levels.

A recent study suggested anticoagulating the ECMO circuit with high levels (>100%) of AT given by infusion and using low level UFH infusions to target an ACT no less than 150 seconds. AT dosing was guided using the following formula: (desired – actual AT level) x Body Weight in kg. A further formula in use is that from the Royal Children's Hospital Melbourne which describe: [(desired – actual AT level) x Body Weight in kg]/2.2. Different calculations have been used in adult patients given AT.

Work has also been undertaken in adult patients looking at those patients supported with continuous renal replacement therapy which supports the use of AT infusions to reduce circuit clotting and failure.

We undertook a local review of the AT levels in our patients in PICU to delineate a normal range for our patient cohort and from this and published evidence have produced this evidence based guideline for the use of anti-thrombin in PICU.

	Day 1	**Day 5**	**Day 30**	**Day 90**	**Day 180**	**Adult**
AT (U/ml)	0.63 ± 0.12	0.67 ± 0.13	0.78 ± 0.15	0.97 ± 0.12	1.04 ± 0.10	1.05 ±0.13
PT (s)	13.0 ± 1.43	12.4 ± 1.46	11.8 ± 1.25	11.9 ± 1.15	12.3 ± 0.79	12.4 ± 0.78
APTT (s)	42.9 ± 5.80	42.6 ± 8.62	40.4 ± 7.42	37.1 ± 6.52	35.5 ± 3.71	33.5 ± 3.44
Fibrinogen (g/L)	2.83 ± 0.58	3.12 ± 0.75	2.70 ± 0.54	2.43 ± 0.68	2.51 ± 068	2.78 ± 0.61

Table 1 Accepted coagulation marker reference ranges according to age
Andrew M et al. Maturation of the hemostatic system during childhood.Blood, Oct 1992; 80 (8): 1998-2005

	Larger population (n=19)	**ECMO population (n=8)**
Median age (days), range	61, (17-2275)	61, (24-187)
Median AT (%), range	52%, (25-109%)	51% (32-78%)
Mean AT (%) ± SD	54.9% ± 20.0	52.3% ± 10.7

Table 2 Median age and AT measurements for the larger population and the sub-population on ECMO
Shah V et al, RHSC, Glasgow data 2009

Guideline for use of Anti-thrombin

- High heparin dose on ECLS (as per treating intensivist or >60iu/kg/hr)
  - Check AT level – if <50% **and** heparin requirements >60iu/kg/hr:
    - Order AT through HISS by "Blood bank / Blood Product Request / Other blood Product Request and enter ATIII giving dose, date and time"
    - Prescribe AT (KYBERNIN) on CIS (Found in "Haemostasis slow iv bolus" menu)
    - Administer AT by slow bolus over 20minutes using following formula:

      (100 – Actual patient AT %) x body weight in kg
    - Take the following blood samples prior to and 1 hour after AT administration: AT, Anti-Xa, Rotem (If outwith normal working hours AT and anti-Xa will be stored in the lab and analysed the following day).
    - For Anti-Xa level type in "Hep"

  - **Precautions:**
    - Do not give if bleeding complications present
    - Expect to reduce heparin dose – check ACT every 30mins until stable
    - Watch for increased anticoagulation with potential for hemorrhagic complications as AT will potentiate heparin effect

Fibrinogen Concentrate (Riastap)

Fibrinogen (factor I) is a soluble plasma glycoprotein which circulates in plasma as a precursor of fibrin. Fibrinogen is an important element in the clotting cascade as it impacts on the structure and stability of the clot formation. Normalization of plasma fibrinogen levels may be associated with satisfactory haemostasis and reduced bleeding. ECMO patients are at an increased risk of bleeding due to the effects of Heparin and the bonding of plasma proteins onto non-endothelial cell surfaces. Fibrinogen is one of the main proteins absorbed by the artificial surfaces of the ECMO circuit. Patients managed on ECMO will have a prescribed Fibrinogen parameter of > 200mg/dl. To attain and maintain this level soon after cannulation most patients will require 1 or 2 units of Cryoprecipitate, for a Neonate, and up to ~ 6 units for an adult. Levels are then usually well maintained during the run, except in certain patient groups.

ECMO patients that may be particularly at risk of increased consumption of Fibrinogen are those with active bleeding, sepsis, DIC and clots either within the ECMO circuit or body. If administration of Cryoprecipitate is not giving an adequate increment of Fibrinogen levels, or excessive volume load or accessibility of cryoprecipitate supplies becomes an issue, then Human Fibrinogen Concentrate may be given.

Human Fibrinogen Concentrate (RiaSTAP) has proven efficacy and is licensed for use in the control or prevention of bleeding in congenital fibrinogen deficiency. Increasing evidence from case reports and case series also indicate its potential usefulness in non-congenital fibrinogen deficiency patients with intractable bleeding, or even as prophylaxis prior to high blood-loss surgery. Human Fibrinogen Concentrate is at present unlicensed for such indications and is expensive (~£400). Nevertheless there may be a place for considering its use to optimise Fibrinogen levels in ECMO patients especially for the indications above.

The mechanism of action of RiaSTAP® serves as a physiological substrate of thrombin (factor IIa), which converts soluble fibrinogen to insoluble fibrin. Under the influence of factor XIIIa, fibrin strands are cross-linked to provide strength and stability to the blood clot—fulfilling an essential need for clot formation in patients with Fibrinogen deficiency.

How to obtain Human Fibrinogen Concentrate

The use of Human Fibrinogen Concentrate treatment must be approved by both the on-call Consultant Paediatric Intensivist (if PICU patient) or Neonatologist (if NICU patient) and the on-call Paediatric Haematologist. Vials can be obtained from blood bank on a named patient basis. Two doses should be ordered initially.

Pre- Fibrinogen Concentrate management

There is very limited experience of the use of Human Fibrinogen Concentrate in infants and children. The most serious adverse reactions that have been reported in adult subjects who received RiaSTAP® are thromboembolic episodes, including myocardial infarction and pulmonary embolism, and allergic-anaphylactic reactions. The most common adverse reactions observed are allergic reactions, including chills, fever, nausea, and vomiting. Patients should be monitored for early signs of allergic or hypersensitivity reactions, and if necessary, discontinue administration. The risk that Fibrinogen Concentrate may clot the Oxygenator of the ECLS circuit or cause thrombotic incidents appears to be reasonably low in our patient group but it is important that an accurate level of Fibrinogen and coagulation status is determined prior to administration of Human Fibrinogen Concentrate , therefore:

- Check ACT
- Full Coagulation screen "/ECLS1" order set on HISS
- ROTEM (inc FibTEM assay).

Thromboelastography/ thromboelastometry looks at the clotting process holistically, in real time, from formation of the initial fibrin strands through to clot lysis. The derived parameters are the clotting time (CT), clot formation time (CFT), maximum clot firmness (MCF) and maximum lysis (ML). In **FIBTEM**; platelets are blocked, so clot formation is dependant on fibrin formation and polymeristaion. Discrepancies will often be seen between the FIBTEM and the laboratory level of Fibrinogen as FIBTEM is more sensitive to clot polymerisation disorders. Normal parameters for children have not yet been determined but the adult parameters are a MCF of 9-25mm. Levels of < 9mm are associated with an increased risk of bleeding.

Human Fibrinogen Concentrate, Pasteurized (Riastap) Preparation

Riastap is a sterile, preservative free, lyophilized fibrinogen concentrate in a single-use vial. The labelled amount of Riastap is 1 g of fibrinogen with the actual potency for each lot indicated on the vial label and carton. Riastap is reconstituted with 50ml Sterile Water for Injection (20 mg/mL) and is administered intravenously. Each vial contains between 900 to 1300 mg fibrinogen and 400 to 700 mg human albumin. Riastap is stable for 24 hours after reconstitution when stored at 20-25°C and should be administered within this time period. Partially used vials should be discarded after 4 hours.

Human Fibrinogen Concentrate, Pasteurized (Riastap) dosing and administration

There are very few case studies which determine the recommended dosage of Riastap in children, although a shorter half life and faster clearance than in adults has been observed. There is some evidence from our limited clinical experience that the dosage recommendation for congenital fibrinogen deficiency is inadequate for our patient group. Therefore an initial dose of 80 mg/kg Fibrinogen Concentrate should be given by slow IV bolus (3-5 mins) to the patient and **not** pre-oxygenator, the injection rate should not exceed 5 mL per minute. A repeat Coagulation screen and ROTEM should then be obtained within 2 hours to recheck levels of Fibrinogen and access need for further doses. Further doses may be repeated if there is still evidence of hypofibrinogenemia as

determined by Clauss Fibrinogen and ROTEM assay. Further doses of Riastap can be incremented by 20% if an insufficient response is obtained from the initial dose.

Post Human Fibrinogen Concentrate Monitoring

The following clinical variables should be monitored in any patient receiving Human Fibrinogen Concentrate:

- Monitor for signs of allergic reactions
- Check ECLS circuit after 30mins for evidence of new clot formation
- Monitor inlet, Pre/Post Membrane pressures
- Repeat Coagulation and ROTEM within 2hrs of dose

Record of Human Fibrinogen Concentrate

As with all blood/coagulation products, details of dose and batch number should be recorded in the case notes.

Patient weight	Calculated dose per formula	Actual dose given	Time given	Pre Fib level	Post Fib level
Pt no 1 - 2.5kgs	100mgs 40mg/kg	10mls/200mgs 80mgs/kg	18:00	1.32	1.43
	84mgs 33.5mgs/kg	10mls/200mgs 80mgs/kg	23:00	1.43	1.5
	107mgs 42.9mg/kg	15mls/300mgs 120mgs/kg	18:10	1.27	1.79
	138 55.3mgs/kg	20mls/400mgs 160mgs/kg	08:00	1.06	1.9
Pt no 2 - 3.5kgs	33mgs 9.4mgs/kg	12.5mls/250mgs 71.5mgs/kg	17:00	1.84	2.63
Pt no 3 - 3.73kgs	31mgs 8.4mgs/kg	14.8mls/296 80mgs/kg	11:30	1.80	2.61
		14.8mls/296 80mgs/kg	22:15	1.86	2.96

Review of RioSTAP dosing and increment of Fibrinogen in three ECMO patients in RHSC

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The bleeding patient

Bleeding is a significant complication in cardiac ECLS patients, especially in those who are post cardiopulmonary bypass with an open chest.

Local control of bleeding is extremely important. Meticulous haemostasis is essential during cannulation and all surgical procedures. It is anticipated that there will be moderate bleeding (5-7ml/kg/hr) in post-operative Cardiac ECLS patients and re-exploration of the chest cavity may be required to evacuate retained blood and exclude a surgical cause.

The current strategy is to treat with factor replacement (especially cryoprecipitate to achieve high fibrinogen levels) and platelet supplementation. ROTEM monitoring has allowed further tailoring of replacement more scientifically. Anti-fibrinolytics prevent clot resorption. Aprotinin (also an adjunct of platelet stimulation and aggregation) used to be a universal therapy but is now limited to 'named patient' use in view of its nephrotoxicity. Bed-side testing uses the Activated Clotting Time (ACT) which cannot be used in isolation to guide heparinisation. Ionised calcium is an important co-factor for coagulation especially in patients receiving high volumes of citrated blood products. When assessing anti-coagulation it is important not to focus on the ACT in isolation as this may be significantly misleading. Therefore we may need to assess a wider profile including the platelet count, anti-thrombin levels, Anti-Xa, APTT, Fibrinogen and ROTEM.

It is to be expected that this patient cohort will demand greater blood product supplementation. Cardio-pulmonary bypass, modified ultrafiltration and cooling all predispose to coagulopathy. Potential sites include the ECLS cannulation sites, the sternal edges, suture lines and generalised ooze. Bleeding is the most common complication reported on the ELSO registry in cardiac ECLS. Bleeding leading to tamponade and circuit failure is not an unexpected complication.

Large volume replacement transfusion is an independent risk factor for mortality even when accounting for the critical illness. Disseminated intravascular coagulopathy caused by factor depletion and microvascular endothelial injury triggering the clotting cascade add to the complexity. Transfusion related acute lung injury is an apparent clinical manifestation of the damage caused by multiple blood product transfusions. Low levels of 2,3 DPG impede oxygen dissociation at tissue level thus elevated saturations offer false reassurance. In addition hypocalcaemia and hyperkalaemia contribute to a coagulopathic state.

The incremental strategy for bleeding ("Bleeding Bundle"):

1. Aggressive correction of coagulopathy
- Discuss urgently with ECLS physician/surgeon
- Fibrinogen >2 using cryoprecipitate
- Platelets > 120,000 up to 150,000
- Correct PT using FFP +/- Vitamin K
- Reduce ACT parameters to allow lower heparin dosing
- Check APTT, TEG
- Maintain high ionised calcium levels

2. Consider aprotinin (1ml/kg/hr)

3. Call ECLS physician & surgeon if blood loss is excessive
- >10ml/kg/hr despite optimising above
- Discuss with ECLS physician and surgeon regarding re-exploration of chest

4. If bleeding remains excessive after re-exploration or 2nd exploration within 12-24 hours
- Novo7 (recombinant Factor VIIa) can also be used at a dose of 95mcg/kg (see Novo7 guideline in appendix or medicine guideline on CIS) at the discretion of the ECLS physician. A clear primed circuit should always be available and surgical and perfusion staff should be aware of the first administration because of the risk of clotting within the circuit, especially if there is evidence of existing significant thrombus. It should always be **given to the patient** and **not** pre-oxygenator.
- Consider conversion to neck cannulation

5. Ongoing correction of clotting screen

Extreme circumstances (bleeding >30ml/kg/hr) despite all of the above.
- Run ECLS circuit without heparin
- Prostacycline 5ng/kg/min may be used as an anticoagulant for 6 hours. Note: Risk of hypotension. This should be undertaken during the day if possible.
- ? decannulation
- Consider VAD if cardiac patient & appropriate

Persistent bleeding (> 10ml/kg/hour) needs additional measures. Heavy swabs and large volume blood/clot in the chest can compromise circuit flows and the surgical repair. Repeated re-exploration increases the risk of mediastinal infection and may paradoxically perpetuate the problem. Targeting a prothrombotic state by pro-coagulant therapy or reducing anticoagulation, risks clot formation in the circuit. If strategies to intensify the clotting process are adopted then high flows should be maintained on the circuit to decrease the risk of intra-circuit clot formation. rFVIIa may be transfused, but our unit policy dictates that this is under consultant authorisation with a replacement circuit available and the perfusionists aware. Heparin free circuits have been used in many centres if exsanguination becomes life threatening. Persistent refractory haemorrhage is potentially fatal. The last resort may be premature decannulation.

Extended Coagulation tests

Patient heparin management should be guided by ACT readings.

The role of the extended coagulation screen is to allow fine tuning of the heparin management if the ACT results are not in keeping with the clinical situation of the patient. The duty Consultant should guide the management of heparin by means other than ACT results.

Anti-thrombin

- Anti-thrombin or AT is a plasma protease inhibitor it inactivates its physiological target enzymes, Thrombin, Factor Xa and Factor IXa. Acquired antithrombin deficiency may result from a range of disorders such as liver dysfunction, sepsis, prematurity, kidney disease with protein loss, or as a result of interventions such as major surgery or cardiopulmonary bypass

- Maintenance of an adequate level of AT activity, which is at least 70% that of a normal functional level is essential to ensure effective inhibition of blood coagulation proteases. AT deficiency, occurs when functional antithrombin levels are reduced to below 50% of normal. When levels are less than 50% and heparin requirements are rising we give AT supplementation as per the guideline.

Heparin Assay

- The heparin assay or anti-Xa (aXa) level is used to determine the concentration of heparin in the body. The blood sample is added to a mixture of anti-thrombin and the enzyme Factor Xa. The enzyme Factor Xa in the heparin assay test is used in the formation of thrombin. Thrombin is involved in the clotting process. If the person has heparin in his or her blood, it will bind to anti-thrombin, and not to Factor Xa. The amount of unattached Factor Xa left as a residue in the blood sample will be measured, and this is directly proportional to the person's heparin concentration.

- The therapeutic level should fall between 0.3 and 0.7. If the ACT does not correlate with the clinical picture of the patient then this is a useful guide to heparin levels in the patient.

aPTT

- Activated partial thromboplastin time testing is routinely used to monitor heparin therapy. Heparin works by increasing the activity of anti-thrombin to inhibit activated factors II, IX, X, XI, and XII. The sample is mixed with calcium and the time (in seconds) it takes for a clot to form is the result.

- Normal ranges for a non Heparinised patient are between 25-39 seconds for the formation of a clot. Heparin therapy will prolong an aPTT.

- APTT does not correlate effectively to ACT levels but generally should be at least > 2.5 times normal. If the ACT does not correlate with the clinical picture of the patient then this is a useful guide to heparin levels in the patient.

D-dimer

- **D-dimer** is a fibrin degradation product (or FDP), a small protein fragment present in the blood after a blood clot is degraded by fibrinolysis.

- D-dimer testing is of clinical use where there is a suspicion of clot formation or DIC.

- A positive D-dimer result may indicate the presence of an abnormally high level of fibrin degradation products. It suggests that there may be significant blood clot (thrombus) formation and breakdown in the body or circuit, but it does not specify the location or cause.

- Elevated levels may be also be seen in conditions in which fibrin is formed and then broken down, such as recent surgery, trauma, haematoma and infection.

- Normal levels are < 0.5 but its use is most valuable in ECMO as a trending tool, a raising level may suggest inadequate anticoagulation and clot formation within the circuit.

Renal support and ECMO

Renal dysfunction is common in patients referred for ECLS. The aetiology is often attributed to the cardiorespiratory haemodynamic extremis, pre-ECLS, which results in global hypoxia, ischaemia or a combination of both. Consequently, renal perfusion will have been compromised. These infants present with a combination of oliguria / anuria, fluid overload, hyperkalaemia, acidosis and an elevated urea & creatinine.
Prior to ECLS, one of the initial strategies to augment a failing cardiac output is volume expansion. Patients who remain fluid overloaded for an indeterminate length of time have been repeatedly shown to have a higher mortality rate, even when controlling for the detrimental impact of multi-system organ failure.
Commencement of ECLS may not simultaneously resolve the acute renal failure. The intra-vascular volume, and the maintenance of ECLS flow, may still require fluid supplementation, especially in the presence of a systemic inflammatory response and capillary leak. However, it is to be anticipated that the improvement of renal blood flow commensurate with ECLS, will eventually lead to recovery of renal function. Renal replacement therapy may be required to bridge this gap, and to allow for the forced urine output, driven by aggressive diuretic therapy, to be suspended.

Continuous renal replacement therapy (CRRT)

- Peritoneal dialysis
- Slow Continuous Ultrafiltration (SCUF)
- Continuous Veno-venous Haemofiltration (CVVHF)

During ECLS we use SCUF by using the volumetric pumps and CVVHF is provided by the Aquarius.

- **Peritoneal dialysis**

Peritoneal dialysis is commonly undertaken in the post-operative cardiac patient. It can be done safely in patients on ECMO. It should be undertaken as per standard unit guidelines.

Peritoneal dialysis catheters should <u>not</u> be sited percutaneously in patients on ECMO because of the heparinisation required

- **Slow Continuous Ultrafiltration (SCUF)**

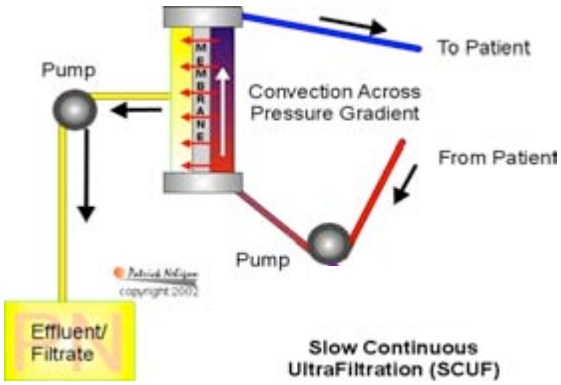
"Drain off ultrafiltrate"

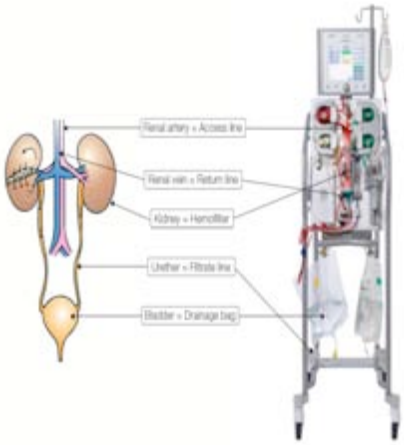
This strategy is mainly used for gentle continuous fluid removal with minimal solute clearance. It can be used to allow volumes of parental nutrition to be accommodated as the solute load of the TPN can be titrated. If larger volume losses as required (>5mls/kg/hr), bicarbonate loss can result in a metabolic acidosis.

See SCUF protocol and **remember the 3 way stop-cock at the venous end must always be opened and all clamps removed before the stop-cock at the "arterial" end is opened.**

A volumetric pump is used to draw ultrafiltrate from the filter. This method is **inaccurate** so it is essential that ultrafiltrate is measured hourly and the pump adjusted if necessary.
Leakage of ultrafiltrate from the filter and circuit can be a problem. To make this easier to identify a green theatre drape should be placed on the floor under the filter.
If the level of ultrafiltrate diminishes and the "air in line" detector on the volumetric pump

begins to alarm this usually means the filter is clotting off. As it takes an hour to prime another filter circuit this should be done as soon as problems begin to arise to limit the time the patient is off SCUF. If this are no problems, filters should be electively changed at 72hours





- **CVVHF**

Please refer to CVVHF guidelines on CIS

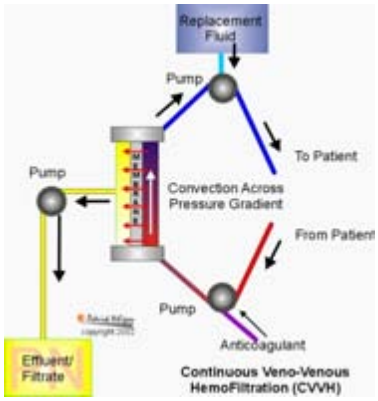
Haemofiltration can be difficult to adequately achieve when the filter circuit is in parallel with the ECLS circuit that is when it is interposed from the arterial limb back to the venous limb. This connection creates a 're-circulation' shunt (blood flows around the ECMO circuit, across the haemofiltration circuit to re-join the ECMO circuit back at the venous side). Haemofiltration in this position filters the blood circulating around the ECLS circuit, rendering less efficient patient clearance. Also return flow to the patient is lost down the haemofiltration limb, 'stealing' artificial cardiac output away from the patient.

The most accurate and effective method of haemofiltration is to establish a independent circuit. However, this requires additional venous access which may be problematic if attempted once ECLS has been commenced in view of the patient having to be fully anti-coagulated.

"Convective" Clearance

CVVHF works via Convection. This is defined as the transfer of solutes in a stream of solvent across a semi-permeable membrane by ultrafiltration mediated by a hydrostatic force. It most closely mimics physiologic glomerular filtration. Water is forced across a semi-permeable membrane, drawing with it solutes by convection. A large volume of fluid is removed to achieve adequate clearances and therefore must be replaced. In Yorkhill, this is fluid is replaced pre-filter and the composition of the replacement fluid should resemble the extra cellular fluid, which can be considered as plasma but without the protein fraction which are generally too large to pass through the pores of the semi-permeable membrane. This is done using the Aquarius and is considered the more efficient, reliable and consistent method of RRT.

The CVVH prescription will be written daily by the PICU consultant.



It must be emphasized that when either method is used either in series or in parallel with the ECLS circuit, the fluid balance calculations may not be accurate. The reason for this inaccuracy is poorly understood. Artificial fluid balance calculations should not replace regular clinical assessment.

Special considerations:

- **Access Pressure to Aquarius**

Gate clamping the arterial limb may be necessary to limit arterial pressures to Aquarius circuit. This can successfully be done using the gate clamps provided (see CVVH guideline).
This can also be undertaken by placing an Lectrocath line as per CVVH guideline.

- **Ensure de-airing**

The most important phase of attaching a CVVH circuit is the de-airing. It is essential to follow the guideline as air entry to the circuit can significantly compromise the patient. The easiest way to de-air the return limb of the CVVH circuit is to do this through a syringe attached to the return 3 way tap to flush the air from the venous side.

- **ACT**

Careful observation of the ACT's is essential when a new haemofilter circuit is attached. The ACT will rise immediately after a new filter has been attached despite the use of minimal heparin and thorough flushing of the circuit during priming.

- **Shunt**

Depending on its size and position in the circuit, a haemofilter may "shunt" 10-20% of the artificial flow away from the patient. If this affects the patient's haemodynamic status, their SvO2 will fall. Consequently, this may necessitate a compensatory increase in the ECLS flow rates if the ECLS circuit will allow.

- **Hypokalaemia**

Potassium is cleared very quickly. Initial treatment should be Potassium-free if blood level is >4mmol/L.
If serum K falls below 3.5mmol/L then add 4mmol KCL to each litre of dialysate fluid (i.e. 20mmol KCL to 5 litre bag of dialysate).Thorough mixing of the dialysate solution is essential – **invert the bag 10 times**.

- **Hypophosphataemia**

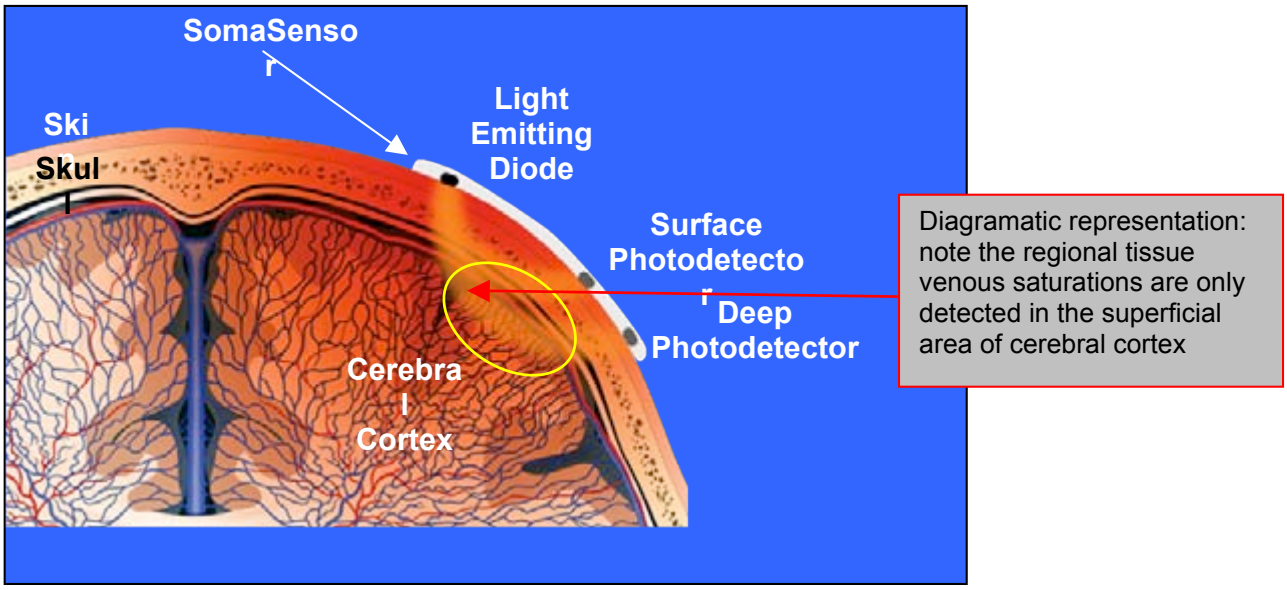
Phosphate level should be monitored closely. A separate infusion of Sodium Phosphate may be required.

Near Infrared Spectroscopy (NIRS) or the 'INVOS' System and ECLS

The NIRS equipment provides information regarding *regional tissue perfusion* depending upon the site of the probe. Currently we use systemic venous saturations to inform us about *total body* perfusion.

The INVOS system is designed to non-invasively, directly and continuously measure regional tissue haemoglobin (venous) oxygen saturations (%rSO_2 value), to indicate the adequacy of the balance between *regional* tissue oxygen delivery and extraction. Its interpretation is analogous to the systemic venous saturations (%SvO_2), indicating the balance between *systemic* oxygen delivery and extraction. The %rSO_2 reflects the amount of haemoglobin still saturated after passing through the local tissue bed. *A low* number (greater than 40% below the arterial saturations) indicates either *inadequate tissue perfusion* (the tissue has extracted an increased amount of oxygen from the haemoglobin as it slowly passes through the tissue bed) or excessive tissue oxygen consumption. The *trend* in value is more significant that the actual number.

A skin probe is placed onto the skin. To assess the cranial saturations, this will be across the forehead. Near-infrared light is emitted from one end of the probe, passes through the superficial tissues, where it is either scattered or absorbed. The amount of absorption correlates to the degree of saturation of haemoglobin. The detector in the skin probe senses the light that has not been absorbed, measured as relative deoxy-haemoglobin, and converts this data into a number which indicates the regional venous saturations (%rSO_2).



NIRS Monitoring on ECMO in PICU

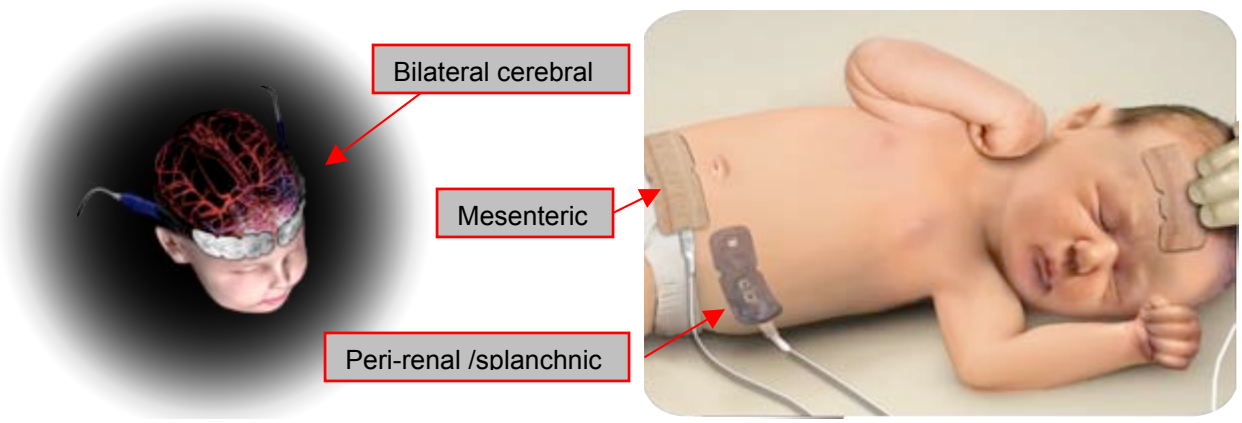
We initially plan to simultaneously use bilateral cerebral probes and a splanchnic probe in the PICU patient group. This is especially important whenever neck cannulation is considered as blood flow to and from the brain will be impeded by the cannulae. Simultaneous comparison of the regional %rSO_2 value of both sides of the brain will reveal the impact of the cannulae on local cerebral blood flow.

In the physiological normal circulation, the cranial saturations are lower than the systemic venous saturations, whereas the splanchnic saturations are higher. This is reflective of the combination of both a higher cardiac output delivered to the kidneys and a lower renal oxygen extraction, compared to the brain. When the venous return from the upper body (cranium) and lower body (splanchnic) meet they unite to form the systemic venous oxygen saturation. Acceptable parameters: SvO2 75%, cerebral %rSO2 65, splanchnic %rSO_2 85

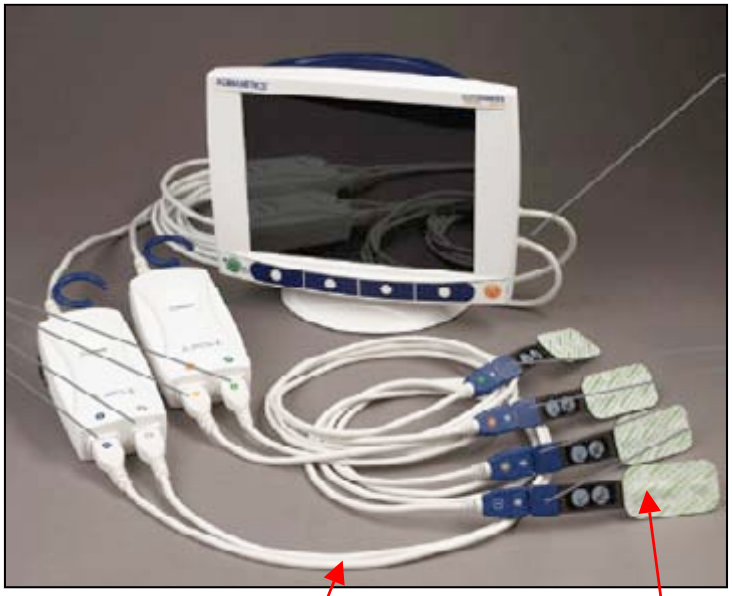
In the ECLS circulation, an atrial septostomy is considered to decompress the left atrium. Depending upon the efficiency of gas exchange within the lungs, partial / fully saturated pulmonary venous blood will flow (with variable characteristics) from the left atrium to the right atrium, *falsely elevating* the *circuit* systemic venous saturation to an unknown extent. In view of this uncontrollable, unquantifiable contamination of left atrial blood into the right atrium to mix and become the circuit 'venous' oxygen saturation, neither the absolute value or trend can be used as a reliable marker for adequacy of global oxygen delivery. *The systemic venous saturation may be falsely reassuring and potentially dangerous.* Regional venous saturation monitoring becomes crucial.

Sensor placement PICU, RHSC

Cerebral – bilateral and not to cross the midline
Peri-renal (splanchnic) – either flank
Mesenteric – below umbilicus



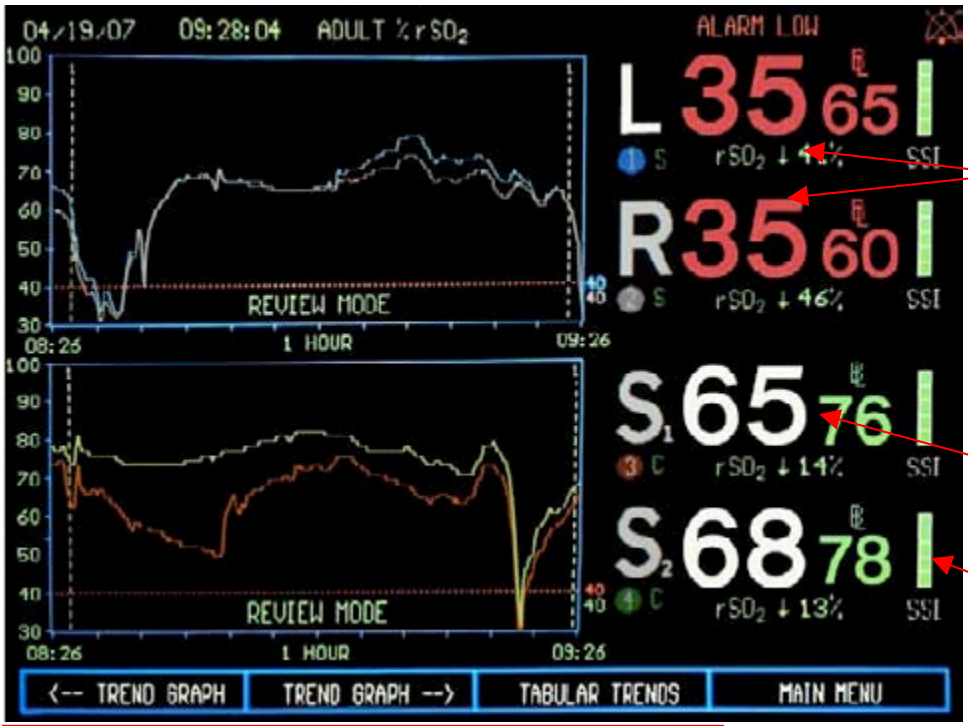
We may use 3 sensors: 1 or 2 cerebral & 1x splanchnic/mesenteric – the splanchnic sensor can be placed over either flank / renal bed.



Disposable sensors

We will use 3+ channels: Modules, channels and Sockets are clearly marked.
The modules with channels 1 (blue) and 2 (grey) connect into the port labelled '1,2':
The module with channels 3 (orange) and 4 (green) connect into the port labelled '3,4' as shown.
Conventionally

- Connect channel 1 (blue) to RIGHT cerebral sensor; channel 2 (grey) to the LEFT cerebral sensor
- Connect channel 3 (orange) (and 4 (green) if requested) to the splanchnic sensors



Regional cerebral %rSO_2

Note: splanchnic %rSO_2 higher than cerebral %rSO_2

Regional splanchnic %rSO_2

Signal strength indicator

Time line can be varied – here, 1 hour data is displayed

The INVOS system is new. **Refer to the user guide on CIS for further set-up information.**
If the %rSO_2 values are worrying, check *arterial* and *patient* mixed venous blood gases and inform a senior clinician.

- Check the Signal Strength Indicator (SSI). This should display 5 green coloured bars to confirm optimal detection. If contact is lost or excessive tissue oedema, the SSI drops and the %rSO_2 is unreliable
- The *trend* is more significant than the actual value
- High values may indicate dead tissue

Absolute cranial %RSO_2 < 40% = significant neurological ischaemia / hypoxia

Clinical indications for NIRS

The role for NIRS is yet to be fully developed in RHSC, and therefore we recommend widespread use in order to familiarise ourselves with the technology and identify a patient cohort where its use may prove most beneficial.

- Potentially any patient with haemodynamic instability, especially the neonate with a 'balanced' circulation
- Any patient supported on ECLS (especially when using neck cannulation) as the circuit systemic venous saturation cannot be used to indicate the adequacy of global tissue oxygenation.

Clinical scenarios

1. ⇩cranial %rSO_2 and ⇩splanchnic %rSO_2 = global oxygen delivery inadequate = check arterial and patient venous blood gases.

   - Check cannula position by echocardiography
   - Patient may have ⇧ oxygen consumption: (investigate aetiology)
     - analgesia, sedation, cooling, paralysis
   - Patient may have ⇩ oxygen delivery:
     - Red cell transfusion
     - Check oxygenator – exclude clots within circuit / exclude wet membrane
     - *Consider* ventilation strategies to improve oxygenation (recruitment manoeuvres, ⇧PEEP, ⇧PIP, ⇧ FiO_2) use with care in patients with parallel circulations.
     - ⇧ cardiac output (⇧ ECLS flow: ⇧ native CI: consider vasodilation)

2. ⇩cranial %rSO_2 (risk of neurological injury) = to exclude embolic event / intra-cranial haemorrhage = consider need for neurological imaging

   - Patient may have ⇧ oxygen consumption:
     - Awake, pain , seizure, hyperthermia
   - Patient may have ⇩ oxygen delivery (local ischaemia):
     - ⇩$PaCO_2$ (consider reducing sweep gas flow)
     - Check cannula position

3. ⇩splanchnic %rSO_2 (risk of NEC)

- ⇧ oxygen consumption:
    - Consider excessive feeding/Local inflammation / gut infection
    - Consider stopping feeds, abdominal X-ray, NEC a'biotic strategy, surgical review
- ⇩ oxygen delivery:
    - Check cannula position
    - Local effect of vasoconstriction (high dose vasopressin)
    - as a secondary physiological compensation in order to maintain cerebral blood flow (may need global tissue oxygenation improving = see 1 above)
      If both are low *and* the ratio is reversed (⇩ cerebral, but ⇩⇩ splanchnic = life threatening disaster)
    - Consider splanchnic infarction

The NIRS equipment provides information regarding **regional tissue perfusion** depending upon the site of the probe. Currently we use systemic venous saturations to inform us about *total body* perfusion.

The INVOS system is designed to non-invasively, directly and continuously measure regional tissue haemoglobin (venous) oxygen saturations (%RSO_2 value), to indicate the adequacy of *regional* tissue perfusion. Its interpretation is analogous to the systemic venous saturations (SvO_2) indicating total body perfusion. The %RSO_2 reflects the amount of haemoglobin still saturated after passing through the local tissue bed. *A low* number (greater than 40% below the arterial saturations) indicates either *inadequate tissue perfusion* (the tissue has extracted an increased amount of oxygen from the haemoglobin as it slowly passes through the tissue bed) or excessive tissue oxygen consumption. The **trend** in value is more significant that the actual number.

A skin probe is placed onto the skin. To assess the cranial saturations, this will be across the forehead. Near-infrared light is emitted from one end of the probe, passes through the superficial tissues, where it is either scattered or absorbed. The amount of absorption correlates to the degree of saturation of haemoglobin. The detector in the skin probe senses the light that has not been absorbed and converts this data into a number which indicates the regional venous saturations (%RSO_2).

It is recommended to use 2 probes simultaneously: one across the forehead to access the venous saturations in the brain and the other across the flank to access the splanchnic venous saturations. Ideally, the cranial saturations should be only marginally lower than the systemic venous saturations (the saturation of haemoglobin in the venous limb of the ECLS circuit) and the splanchnic saturations should read 10-15% higher, reflective of the higher cardiac output delivered to the kidneys.

- Cranial RSO_2 ≈ (just less than) SvO_2
- Splanchnic RSO_2 ≈ SvO_2 + 10-15%

Ideally, the **Signal Strength Indicator** (SSI) should show 5 coloured bars to confirm optimal detection.

NIRS Monitoring on Neonatal ECMO in NICU

Indications
All neonates being considered for ECMO or cannulated for neonatal ECMO (VV or VA). Ideally the probes should be in situ prior to cannulation.

Equipment
The NIRS monitor in use in 2B/NICU is the Invos Somanetics 2000. This monitor has the potential to display 4 channels of information from 4 different tissue beds. A standard approach would be to monitor cerebral saturations in conjunction with a renal or mesenteric probe (somatic measures).

Figure 1

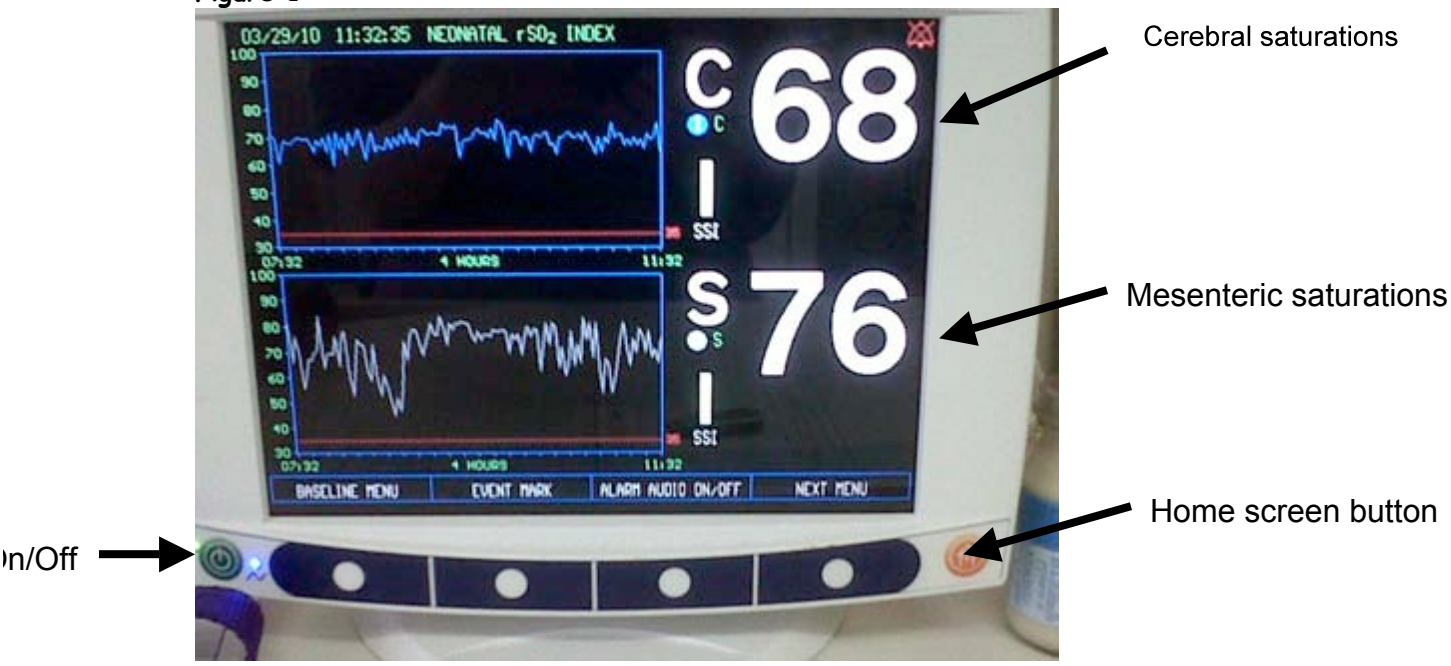
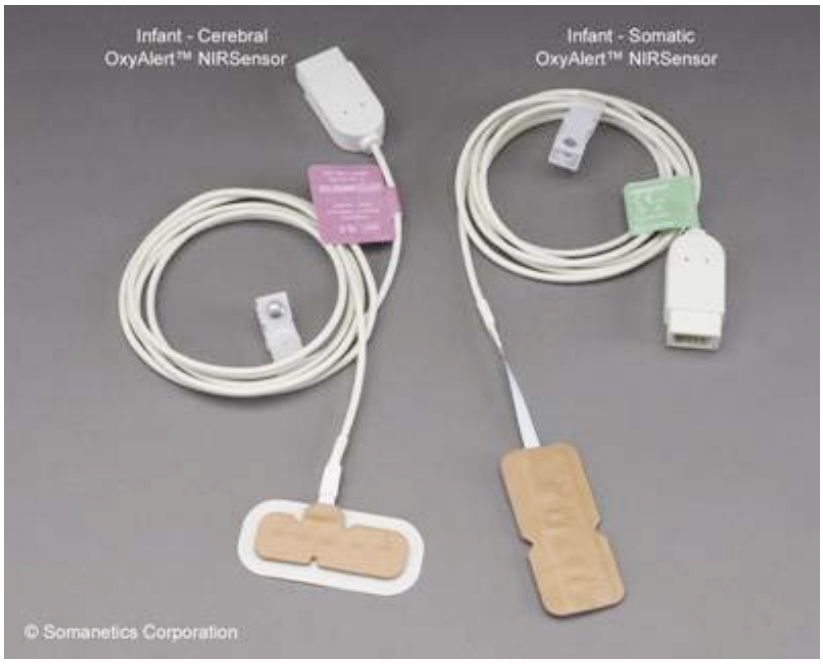


Figure 1 demonstrates the Invos Somanetics monitor and typical screen outputs. The large figures represent the regional oxygen saturation and the graphs demonstrate the change in saturation over a period of time. In this situation the upper readings refer to cerebral saturations and the lower readings are the somatic saturations. The red line represents a danger line when regional saturations are < 40 % - a point at which tissue oxygenation is considered to be dangerously low. The bars marked SSI are indicators of the quality of pick up – the coloured bars should be full. If not check that there is good probe contact and exclude extraneous light sources. A hat over the cerebral probe may be useful.

The monitor is simple to operate with a green on/off button at the bottom right of the screen. Simply switch on, connect the probes and press the function key below the "new patient". No other clinical data is required. Labelling of probes and the time scale of the charted information can be adjusted by scrolling through the menus and selecting choices using the touch function keys along the bottom of the screen.

Probes and probe position:

Figure 2



There are two different neonatal probes available for use with the Somanetics monitor (figure 2). The Cerebral OxyAlert sensor for use over the scalp (cerebral saturations) and the Somatic OxyAlert sensor (for use on the body). They have different orientation of leads for ease of use (see below) but have also been validated for different areas and are not interchangeable.

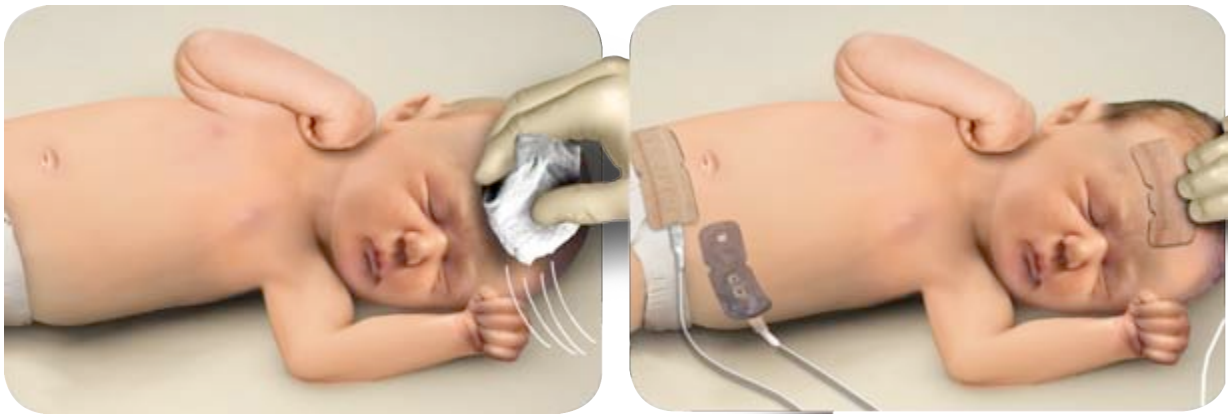


Figure 3

Figure 3 demonstrates suggested probe positions; cerebral and somatic. In this case the somatic probes are renal or mesenteric. Note from Figure 1 the cerebral readings are usually very steady whilst the renal and mesenteric readings fluctuate considerably reflecting the much greater influence of the sympathetic nervous system on these regions.
The supra umbilical area is generally best avoided because of the impact of hepatic blood flow.

Changing probes

Manufacturers recommend changing after 24 hours. However our experience in term babies with normal skin is that they can remain in situ for 48 -72 hours before reviewing skin integrity.
Probes are designed to be disposed after a single use.

Interpreting NIRS readings.

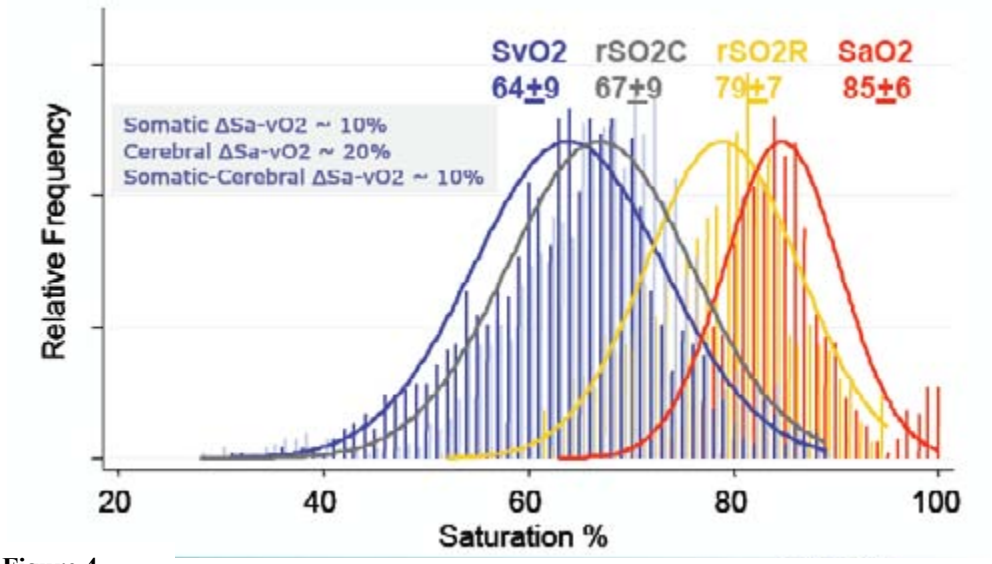


Figure 4

Figure 4 presents data from neonates with congenital heart disease but demonstrates the typical relationship of cerebral and renal regional saturations compared to SvO2 and pulse oximetery readings. SvO2 and cerebral saturations are closely related – a finding that has been confirmed on neonates on VV ECMO (1). Typically renal saturations are higher than cerebral saturations but this may not be a consistent finding on ECMO patients.

Although NIRS is proving to be a useful clinical tool variation in sampling means that absolute values cannot be deduced from measurements. Therefore it is an adjunct to monitoring. Interpretation is based on the following points:

- Trends in values are key
- Cerebral saturations generally should be 60-80%
- Renal saturations generally should be > cerebral saturations 5-10%
- Multi-site readings for comparison are important (cerebral and at least 1 somatic probe)
- An increasing gap between pulse oximetery and regional saturations suggests poor end organ perfusion.
- **Critical values that should cause concern are**
  - absolute value < 40%
  - fall of > 20% from initial baseline
  - fall in absolute value with loss in regional differences
  - widening gap between pulse oximetery and regional perfusion (pSaO2 – rSo2 > 20% is abnormal)

Clinical scenarios on neonatal VV ECMO:

NIRS measurements can highlight poor organ oxygenation are therefore useful in highlighting poor cardiac output states and anaemia. However there are some specific scenarios common to neonates on ECMO where NIRS may be particularly useful.

- **Falling NIRS values:**
  - Recirculation on VV ECMO

Significant recirculation on VV ECMO is suggested by a rise in SVO_2 without a matching improvement in pulse oximetery or a gradual fall in $PSaO_2$.
Regional saturations will fall in this situation if recirculation is significant.

  - Pulmonary hypertension

VV ECMO will deliver adequate oxygenation in severe pulmonary hypertension but systemic blood flow may be severely compromised in the presence of a restrictive PFO or closing PDA. Low NIRS measurements, often coupled with low SVO_2 but adequate arterial saturations may indicate the need for cardiac Echo and management of the arterial duct.

  - Cardiac tamponade

Regional saturations can be critical in giving early warning in this situation. As tamponade develops SvO_2 will be high due to recirculation and $PSaO_2$ will be maintained. However organ blood flow will be dramatically reduced due to poor cardiac output causing a rapid drop in regional saturations. This diagnosis should always be considered in this context. Figure 5 shows the NIRS screen of a baby developing tamponade who had "normal" SVO_2 and $PSaO_2$ readings. The sharp upward deflections at the end of the trace occurred immediately after blood was aspirated from the pericardium.

Figure 5

- **High NIRS values:**

Generally high NIRS values are indicative of good organ perfusion and oxygenation but extremely high NIRS values can indicate abnormal organ blood flow and/ or decreased organ metabolic activity which typically follows significant hypoxic ischaemic insult. Rises in cerebral NIRS 24 hours after a hypoxic ischaemic insult are significantly associated with poor neurological outcomes (3). Therefore persistently high cerebral NIRS values may indicate a pre existing brain insult or significant intracranial haemorrhage on ECMO.

References

1.Rais Bahrami K, Rivera O, Short BL. Validation of a non invasive neonatal optical cerebral oximeter in veno-venous ECMO patients with a cephalad catheter. *J Perinatol.* 2006 Oct;26(10):628-35.

2.Lemmers P, Toet M, Van Bel F. Impact of PDA and subsequent therapy with Indomethacin on cerebral oxygenation on Preterm Infants. *Pediatrics* 2008;121:142-147

3.Toet M , Lemmers P, Van Schelven L, Van Bel, F. Cerebral Oxygenation and Electrical Activity after Birth Asphyxia: Their relation to outcome. *Pediatrics* 2006;117:333-339

Neurological issues in ECMO

Imaging

Undertake daily Cranial USS if anterior fontanelle open for 1st three days of run then Monday, Wednesday, Friday unless clinical concern.

ECMO patients are transportable however moving a patient on ECMO support to CT for example takes significant planning (see "Intra-hospital transport procedure"). Bedside cranial ultrasound is possible in those with a patent anterior fontanelle. An EEG is difficult to interpret in view of the sedative medication, scalp oedema and interference from the bed-side equipment. Continuous cerebral function monitoring (CFAM) can be used effectively as an early warning of seizure activity, especially in the paralysed patient. Retinal photography may prove to be a beneficial adjunct. To allow for clinical examination of cerebral function, paralysis needs to be discontinued and sedation lightened, but without jeopardizing cannulae safety. Continual paralysis obscures neurological signs, risks critical care poly-neuromyopathy and is associated with extravascular tissue oedema.

Cranial USS should be undertaken on a daily basis for the 1st three days of the "run" and then on a Monday, Wednesday and Friday electively unless there is a clinical need for one to be undertaken at other times.

A call should be placed to the duty radiographer to highlight when a patient is being placed on ECMO so they can plan this. We should not make repeated calls to USS to "chase" this up unless there an urgent clinical need as it merely slows down the radiographer's activity.

CT scanning can be undertaken if there is concern as to a patient's neurological status (see "Intra-hospital transport procedure").

Continuous EEG Monitoring (CSA) in PICU

The Continuous EEG or CSA (Continuous Spectral Activity) is not an alternative to the traditional EEG, which provides detailed information about localised pathological electrical brain activity over a specific short period of time. Instead, the Continuous EEG(CSA) provides long-term, continuous, global feedback to help you guide therapy for the brain conditions encountered in the ICU.

What can it show?

- Separate hemispheric events
- Cerebral swelling
- Vasospasm
- Fitting
- Prognostic waves
- Sedation levels

It is a useful adjunct to assess the degree of sedation which may be difficult early on in the ECMO run with unpredictable clearance of sedatives.The concurrent use of muscle relaxants makes this task even more difficult. Catastrophic cerebral events whilst on ECMO are sadly not rare and may be suggested by an unfavourable CSA with potential vasospasm around the site of a bleed. The major limiting factor of Continous EEG monitoring is that-in contrast to an EEG-it provides a 4 channel recording which limits its capability to detect a problem as it covers a very limited area of the scalp and thus underlying brain. If seizure is clinically suspected then a formal EEG would be best with subsequent mapping and appropriate placement of CSA electrodes for ongoing monitoring purposes.

A PICU Guideline Document will be available for reference in the near future.

Continuous EEG monitoring in NICU

Continuous EEG monitoring in NICU is performed using the *Olympic CFM 6000* (cerebral function monitor). The principle is very similar to PICU in that it is used as an adjunct to other forms of monitoring but not as a replacement to a traditional EEG. Within NICU the CFM is used primarily to identify suppressed brain electrical activity and / or seizures and hence to guide clinical decisions regarding cooling and / or anticonvulsant therapy.

Therapeutic hypothermia

Current evidence indicates that induced hypothermia (cooling) to a rectal temperature of 33-34°C improves survival and neurological outcome in neonates with moderate or severe perinatal asphyxial encephalopathy (Azzopardi et al, Moderate hypothermia to treat perinatal asphyxial encephalopathy; N Engl J Med 2009). Babies referred for ECMO support are also known to be at risk of adverse neurological outcome, often as a result of hypoxic brain injury, however the role of cooling in this context is unclear. The *Neonatal ECMO Study of Temperature (NEST; www.npeu.ox.ac.uk/nest*) has recently completed recruitment and surviving children will be developmentally assessed at two years old. Hopefully this will provide further clarification however final results will not be available until 2012. In the interim a decision to cool for neuroprotection whilst on ECMO will be made on an individualised basis.

Microbiology

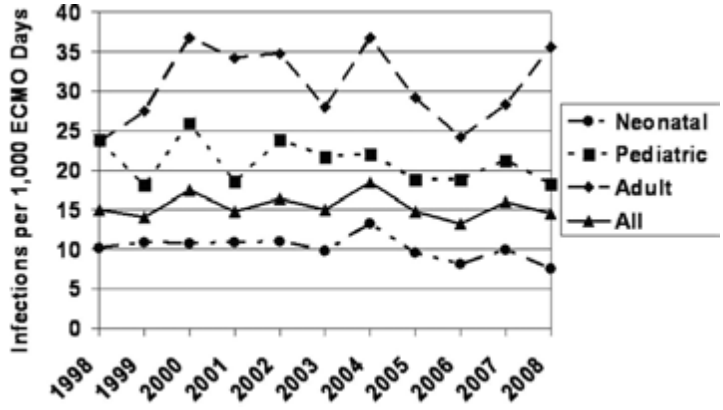
Overview

Bloodstream infections are a significant complication of ECMO. Placement of intravascular catheters into large vessels is an invasive procedure and the ECMO circuit has multiple portals of entry to the circulation, which renders a patient on ECMO more susceptible to infection. Odetalo et al reported that patients who required intravascular access devices for ECMO had a ten-fold increased risk of developing a BSI. In addition, patients on ECMO often have deficiencies in their immune system. Sepsis in patients on ECMO has been reported in many studies to increase mortality. Meyer et al also reported that septic neonates on ECMO were at increased risk of other complications, including seizures, gastrointestinal bleeding, renal dysfunction and metabolic derangement.

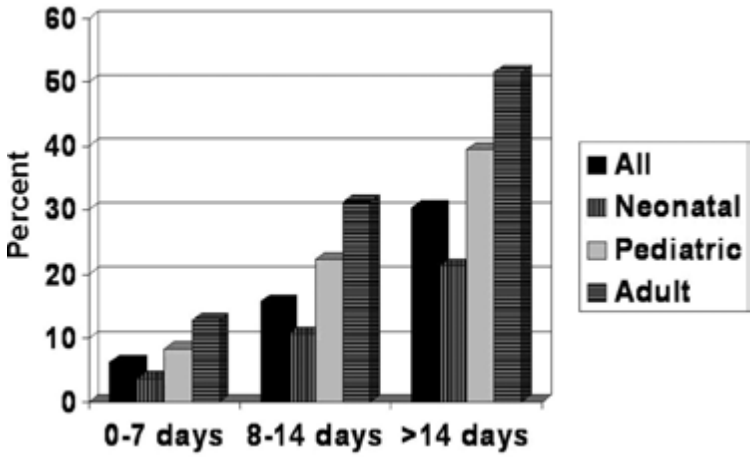
Sepsis can be difficult to identify in patients on ECMO, and therefore difficult to initiate appropriate antimicrobial therapy promptly. Temperature instability is uncommon on ECMO as an active heat exchanger maintains the patient's core temperature. Thrombocytopenia is an unreliable indicator as it often occurs secondary to platelet aggregation and white cell derangement may occur on ECMO. Diagnosis of sepsis continues to be a challenge and some centres use daily surveillance cultures and/or prophylactic antibiotics during ECMO support.

A prospective observational study has been ongoing looking at all patients who required ECMO support in either the paediatric intensive care unit (PICU) or the neonatal intensive care unit (NICU) from July 2006. The incidence has reduced from 55 to 16 BSI's/1000 ECMO days. There were no BSI's in the VV ECMO patients or those in the neonatal unit.

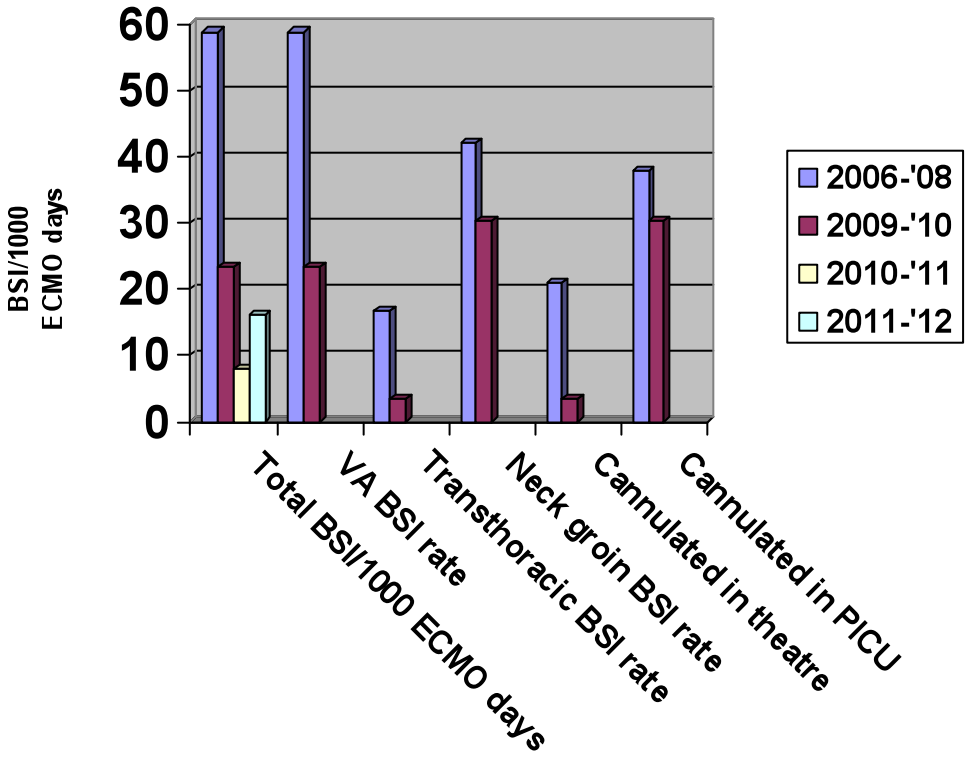
A recent retrospective review of the ELSO database found similar levels of infection (15.4 BSI's / 1000 ECMO days) in 20,741 neoanatal, paediatric and adult patients receiving ECMO between 1998 and 2008 (Pediatric Critical Care Medicine 2011; 12). They also showed an increasing risk of infection the longer the ECMO run went on. (see figures below)



Trend of BSI/1000 ECMO days over time
Bizzaro et al PCCM 2010;12



Prevelance of ECMO infections by duration of ECMO run
Bizzaro et al PCCM 2010;12



RHSC Blood stream infection during ECMO data

Infection prevention

There is no evidence to support prophylactic antibiotics in these patients. However a tight surveillance system is needed as well as heightened awareness of the possibilities of infection given the increased risks of hospital acquired infection in patients supported with ECMO.

Ideal practice is to give the first dose of antibiotics at least 30 minutes prior to cannulation to ensure reasonable serum levels. It is routine practice to give 24Hrs of Cefuroxime following cannulation. Antibiotics should be stopped at 24Hrs unless there is clinical concern regarding infection.

If patients are already on antibiotics covering presumed sepsis these should be continued but the extent of antimicrobial cover reviewed and rationalised in light of positive cultures.

The most common organism isolated in blood cultures in our institution is coagulase negative staphylococci (48%) however we have managed to avoid any BSI's with this bacteria in the last year. Our reporting of coagulase negative staphylococcus can cause some difficulties when comparing results with other centres as some centres will consider this a contaminant and thus not report it as a BSI, which may cause the BSI incidence reported by ELSO to be artificially lowered[8]. Coagulase negative staphylococcus can cause sepsis and has thus been included in our figures. 80% of the organisms causing BSI in our cohort are sensitive to vancomycin or gentamicin, which would consolidate our current approach as first line treatment for suspected sepsis. Two patients in the last 7 years developed candida parapsilosis. Thus increased awareness of fungal pathogens is important and thus there should be a low threshold for starting antifungal cover in cases of sepsis.

Strict hand cleanliness guidance should be followed by all staff approaching bedspace.

Needle-less port systems, such as Bionector, have been shown in conjunction with other infection control measures to reduce the incidence of BSI's. Bionectors allow regular access to a circuit whilst preserving a relatively closed system. The advantage over a traditional pigtail sealed with a cap is that the membrane over the Bionector can be easily cleaned before activation and therefore is more likely to remain a sterile point of access. To that end, all access points on the circuit will have Bionectors.

When accessing invasive lines it is essential that adequate sterility is observed and effective hand washing and the use of sterile gloves should be employed at all times. Cleaning the Bionector port to ensure maximum sterility is very straight forward BUT slightly more time consuming than before. It is recognised that time is not always on our side but vigilance regarding this procedure is worth it. A reduction in blood stream infections on ECMO which has a known morbidity and mortality and is associated with an increased length of stay can lead to an overall decrease in workload and improved patient care.

Bactericidal killing is a time related process. Firstly the port needs to be cleaned with an alcohol swab (soon to become a Chloraprep swab) for 30 seconds followed by a further 30 seconds allowing the port to dry. This part, whilst tedious, is very important. It is the evaporation of the alcohol during this stage that causes the most destruction of bacteria and any attempt to access the port prior to the completion of this process could introduce infection into the circuit. Once the Bionector has been used, any blood should be wiped away from the surface as this could attract bacteria and encourage their proliferation.

Bionectors can be activated 150 times and therefore as per unit policy will be changed twice a week. When placing Bionector on the circuit it should be attached to a 2 ml Luer Lock syringe and flushed through to remove any air from the dead space. It can then be directly attached onto the circuit

To minimise infection risks then infusions should be run by the circuit or central lines and boluses given peripherally where possible.

Infection surveillance
There is no evidence to support the routine undertaking of blood cultures

If increase in inflammatory markers or pyrexial or clinical concern of sepsis:

- WBC, White cell differential, CRP
- Blood cultures should be taken by standardised aseptic method
- Blood C&S all venous & arterial sites (do *not* undertake venepuncture on heparinised patients – ECLS Consultant decision only)
- Blood C&S from ECMO circuit
- Urine C&S if catheterised
- Blind broncho-alveolar lavage (BAL) if no blood per ETT or bronchoscopic BAL

Surgical antibiotic prophylaxis

- Cefuroxime 30mg/kg tds for 24Hrs only

1st line antibiotics for presumed secondary sepsis

- Vancomycin
- Gentamicin
- Prophylactic Fluconazole 6mg/kg once daily (ensure neonatal dosage correct)
- Add Tazocin if suspect Pseudomonas

2nd line antibiotics

- Review in light of current microbiology

Rationalise antibiotics after 48Hrs in light of cultures
Ensure that microbiological data is updated daily in CIS datasheet.

Enteral Nutrition in ECMO

It is widely accepted that enteral nutrition (EN) is the preferred method of feeding in critical illness due to its' physiological advantages of maintaining gut integrity and reducing infectious complications compared with parenteral nutrition (PN). There are numerous studies which suggest that early implementation of nutritional support (within 24hours) is associated with improved outcomes, shorter length of stay, decreased infection rates and enhanced immune function. Providing nutritional care in critically ill children poses a number of challenges resulting in failure to deliver estimated energy requirements. Failure to provide nutritional intake equal to or exceeding the basal metabolic rate (BMR) is associated with higher mortality and morbidity rates.

If the patient is clinically stable and not in the "high risk abdomen group" (see below) nutritional support may be initiated within 24 hours as per standard guideline (see Critical Care Nutrition website or PICU guidelines website). Those in the High Risk Group should be nutritionally supported as per the "HRA" nutrition guideline (see Critical Care Nutrition website or PICU guidelines website).

High Risk abdomen Group

Is the patient < 12months of age and have any of the following "HRA" triggers?

PICU Consultant to identify patients requiring this guideline:

Cardiac Group:

- HLHS/Crit AS/IAA/Coarctation
- Cardiopulmonary bypass time > 180mins
- Aortic cross clamp time > 90mins
- Deep hypothermic circulatory arrest
- ECLS (ECMO or VAD)
- Nil enterally pre- cardiac surgery
- Introduction & /or escalating use of vasoconstrictors

All Patients:

- Confirmed & treated NEC in last 4 weeks
- < 30/40 gestation (+ < 6 wks corrected gestational age)
- Initiation of VA ECMO or VAD
- Presence of UAC
- Clinical concern at Consultant level
- 2 consecutive $\triangle$A-VO_2 >35%
- SvO_2 or $ScVo_2$ < 45%

Most patients supported with ECMO will tolerate EN and this will be the preferred route of nutritional support where possible. Gastric feeding should be attempted in the first instance but if there is evidence of gastroparesis then post-pyloric feeding should be initiated by passing an oro-jejunal feeding tube (please refer to Guideline on Blind Bedside Jejunal Tube Insertion).

If there are no concerns regarding the patient's abdomen then EN should be initiated using the Guideline for Introducing and Establishing EN in PICU.

If there are concerns over gut function, such as suspected or confirmed necrotising enterocolitis (NEC), long cardiopulmonary bypass run or low cardiac output state then it may be appropriate to take a more conservative approach to commencing enteral nutrition by keeping the patient nil enteral or feeding at trophic volumes and initiating PN as the main mode of nutritional support. This plan should be made by the treating Intensivist. A "high risk abdomen" feeding guideline has been produced to support and guide the management of nutrition in these patients.

Our work has shown that we start enteral feeds within 6 hours in patients on VV-ECMO and a median of 21 hrs in those on VA-ECMO. There were no instances of necrotising enterocolitis in our group. Median energy intake was only 30.5kcal/kg however with the main limit being feed volumes. Thi sled to only 45%

of the patients achieving their basal metabolic rate. This is something to continually try to optimise during an ECMO run. Future audits will assess this further.

Ordering EN in PICU
Standard infant formulas are available within PICU store room and the stock levels of these are monitored at ward level. Specialist infant formulas and Paediatric feeds are requested from the Special Feeds Unit (SFU) via the unit dietician. If such a feed is required out with dietetic working hours or at a weekend/public holiday these are located in the SFU freezer/store room.

Administration of EN in PICU
The majority of patients on ECMO will tolerate gastric feeding, delivered via an oro-gastric (OG) tube (unless Consultant Intensivist has requested a naso-gastric tube).

Most PICU patients will be fluid restricted which can make meeting estimated nutritional requirements challenging. Generally speaking, most infants will require 80-100ml/kg of standard infant formula to meet their minimum nutritional needs. Patients over the age of 1 year should meet their minimum energy requirements with 50-70ml/kg of enteral feed.

All patients should be commenced on continuous feeding initially, and if there are no concerns over feed tolerance, this can change to bolus feeding.

If there are concerns relating to gut function or if the patient is being fed via an oro-jejunal tube (ie post-pyloric) then the patient should remain on continuous feeding.

Monitoring of EN in PICU
Measurement of gastric residual volumes (GRVs) is used routinely to assess enteral feed tolerance in critical care units. There is wide debate over the use of measuring GRVs to assess feed tolerance as they have never been validated as a reliable measure of aspiration risk of enteral feed tolerance. Current opinion suggests that a GRV >5ml/kg can be considered to be an indicator of delayed gastric emptying.

Please refer to the "Guideline for Introducing and Establishing Enteral Nutrition in PICU" for guidance relating to the management of GRVs.

If there are signs of abdominal distension, vomiting, faeculant or frankly bilious or bloody aspirates then please inform PICU medical staff for advice.

Existing daily monitoring of U+Es, phosphate, calcium and magnesium should be sufficient to determine if additional supplementation of electrolytes is required. It is not common practice to add electrolytes to enteral feeds in our unit.

Enteral nutrition in NICU
This should be undertaken in conjunction with the ECLS physician or surgeon caring for the child

Parenteral nutrition in ECMO

Most patients supported with ECMO will tolerate enteral nutrition and this is the optimal route for nutrition delivery. If gastric feeding is not successful post-pyloric feeding can be established with passing an oro-jejunal tube (see guideline). **NB do not pass nasal feeding tubes on ECMO patients unless under Consultant request and supervision**.

If there is concern over the patients gut, for example suspected or confirmed necrotising enterocolitus (NEC), low cardiac output state, long cardio-pulmonary bypass run it may be appropriate to initiate parenteral nutrition (PN). These patients will fall into the "High Risk Abdomen" Group.

PN for ECMO patients is ordered and monitored in the same way as for other PICU/NICU patients. The main difference is the route of administration.

Ordering PN
PN is ordered jointly by medical staff and the ward pharmacist, with advice on nutritional requirements given by the dietitian. PN must be ordered by 11.30am Monday to Friday. Weekend bags will be prepared on a Friday.
If a patient on PICU needs started on PN outside pharmacy opening hours, contact on-call pharmacist for advice.
Neonatal bags are available from NICU and Clinimix N9 bags are stored in the emergency cupboard.

Administration of PN
Most PICU/NICU patients are fluid restricted to some extent and PN is ordered in a concentrated form to maximise calorie content which must then be given centrally. If no central access is available, PN can be given via the ECMO circuit itself. Some units avoid this due to concerns about membrane blockage but this has not been our experience at RHSC.

There is currently no evidence regarding nutrient removal via the ECMO circuit, so we do not know if the patient receives all the intended nutrition, but in the absence of alternative routes of administration the circuit may be used. If PN is being given into the circuit in a patient on V-A ECMO it should be given pre-oxygenator to minimise the risk of air embolus.

Aqueous PN is given as a continuous 24 hour infusion but lipid is normally given over 20hours (off from 4am until 8am) to improve tolerance.

Monitoring
ECMO patients are likely to have a degree of renal and hepatic compromise. This can affect the amount of protein, glucose, lipid and electrolytes that the patient may tolerate.

Existing routine daily monitoring under the HISS code *"/ECLS1"* (FBC, aPTT, U+Es, LFTs, magnesium) is sufficient to detect most PN related problems.
Triglycerides must be monitored daily at first until lipid tolerance established, then twice weekly thereafter. It is routine in PICU to stop the lipid infusion for 4 hours prior to checking triglyceride levels. This is not routinely undertaken in NICU.
If liver impairment is significant, GGT may need to be checked.

If PN continues long term (more than a month), then bone density should ideally be monitored but in practice this is rarely done. Vitamin and mineral screening may be carried out on the advice of the GI team. See the back of the PN prescription for more details.

Compatibilty with other infusions

Ideally PN should be given via a dedicated line. Information is available on CIS about compatibility with commonly used drugs (Use information tag on prescription page). Contact pharmacy for advice if a drug is not listed.

Physiotherapy and ECMO

During ECMO

Indications
Chest physiotherapy should be considered for all patients with retention of secretions, with or without focal collapse or consolidation. Referral should be made by medical staff via HISS with a follow-up page (Pg 8137) during normal working hours or a telephone call via switchboard to the on-call physiotherapist out of hours. Every effort should be made to make the PICU physiotherapists aware of likely referrals during the normal working day.

Respiratory
Accurate physiotherapy assessment of the chest may be difficult when the patient is first placed an ECMO if ventilation is reduced making auscultation difficult and chest x-ray frequently shows a total white-out if the patient is on V-V ECMO as they are often placed on rest settings on the ventilator and this therefore may not be secretion related.

Joint/Neurology
Joint range of movement should be monitored within the restrictions of lines and cannulae and if a prolonged period of immobility is anticipated the patient should be referred to the Orthotics department for pressure relieving ankle foot othoses (PRAFO's) – this requires a HISS referral to Orthotics from medical staff and should be marked as urgent.

Contraindications
Open chest
Tension pneumothorax
Severe pulmonary haemorrhage
Significant bleeding from cannula site
Significant pulmonary hypertension

Timing
The overall treatment plan as well as the plan for the day should be discussed with the ECMO Specialists caring for the child and the treating Intensivist. This will allow optimal care to be co-ordinated and allow cluster care when tolerated.

Inhaled medication should be co-ordinated with chest physiotherapy –

- Bronchodilators – 10-15 mins pre-physio if necessary and benefit shown.
- Hypertonic saline – just before physiotherapy (assess for bronchoconstriction).
- DNase – (nebulised or instilled) at least 1 hour pre-physio.
- Inhaled antibiotics or steroids – post-physio.

ACT should be checked and if over 240 sec ECMO specialist nursing staff and the responsible Intensivist should be consulted regarding the acceptable parameters for the individual patient.

Stability of patient must be discussed with nursing staff prior to undertaking physiotherapy assessment and input.

The number of treatment sessions will be determined daily by the physiotherapist based on response to treatment and perceived benefit/risk.

Method

Effective positioning should be attempted as patient stability and cannula position allows – this is important for both VQ matching and mobilisation of secretions but would be precluded if cannulae are precarious.

Manual hyperinflation is carried out with care to avoid volutrauma and barotrauma.

Bronchial lavage may help remove secretions and where particulary tenacious, e.g. MAS, alveolar proteinosis, etc., larger volumes of saline than may be commonly used when treating a conventionally ventilated patient may safely be instilled into the ETT since the circuit is supporting gas exchange. Often physiotherapy input in tandem with flexible bronchoscopy can work well to aid secretion clearance in difficult cases.

Hands on techniques (autogenic drainage, vibrations, etc.) will be utilised with care to avoid trauma whilst cardiovascular stability and circuit flow are monitored throughout.

ET and OP suction are carried out with care but *NP suction should be avoided.*

After ECMO

As patients improve, age-appropriate developmental, mobilisation and strengthening programmes should be instituted. Since patients who have been very debilitated by their illness may require long-term rehabilitation, this may require communication with the physiotherapists at the referring hospital.

Flexible bronchoscopy during ECMO

Flexible fibre-optic bronchoscopy (FFB) has been used in paediatric medicine for over 25 years following the pioneering work of Bob Wood. Several papers have highlighted the high safety profile and effectiveness of this investigation when used in paediatrics.
We undertook a review of the first 200 FFB's undertaken in PICU between October 1992 and 2003. 80 of these FFB's were undertaken on 44 patients receiving ECMO support. This represents 53% of the 83 patients that received ECMO support in PICU during this period. 12 of the 80 (15%) FFB's showed an endo-tracheal tube that was abutting or below the level of the carina and 3 (3.75%) showed near complete obstruction of the endo-tracheal tube with haemorrhagic clot. These patients were re-intubated. 13 patients had non-pulsatile compression of the airways and 3 had pulsatile compression. Evidence of pulmonary haemorrhage was noted in 12 patients on ECMO at the beginning of the procedure and therefore not related to the FFB. 43 FFB's (53.8%) undertaken on patients on ECMO were reported as normal. A broncho-alveolar lavage (BAL) was undertaken in 52 of the 80 FFB's undertaken on patients supported with ECMO. 28 were positive with 21 being a pure growth (40.4% of all BAL's in ECMO patients). All patients tolerated the procedure well and no pulmonary bleeding was noted in relation to the FFB. No change was made to anti-coagulation in relation to the FFB taking place.
Flexible bronchoscopy is now seen as a routine test in patients on ECMO with a high rate of positive findings. The bronchoscopy report should be filled in and the event logged on CIS in PICU for all patients.

Indication

- Confirm correct ETT placement
- Assess airway anatomy or obstruction
- Endobronchial toileting / lavage
- Suspicion of discrete lobar ventilator associated pneumonia

Contra-indication

- Active bleeding per-ETT

Drug prescribing in ECMO

ECMO can affect drug handling in a numbers of ways:

- Direct effect of ECMO circuit e.g. drug binding to tubing or oxygenator
- Haemodilution and volume expansion increases volume of distribution (more pronounced at initiation of ECMO or if bleeding issues requiring multiple transfusions of fluid boluses)
- Impaired renal function

Effects on individual drugs are hard to predict due to lack of evidence and there is wide inter and intra-patient variability. In some drugs e.g. sedation or heparin, the effects are easy to measure and make adjustments for, but in others such as gentamicin the effects will not be known until serum drug levels are reported or toxic effects occur.

Changing the ECMO circuit may affect drug handling in a previously stable ECMO patient.

Antibiotic monitoring
Refer to the critical care prescribing and monitoring policies for advice on gentamicin and vancomycin in ECMO in NICU & PICU.

Anti-coagulation
Administration and monitoring of Heparin, Recombinant Factor VIIa (Novo-7) and antithrombin are covered in the anti-coagulation section.

Sedation
Sedation requirements are usually much higher if patients are supported on ECMO.

Decannulation
Remember that drug dosing must be reassessed once a patient is decannulated or if renal support is initiated or ceased.

If in doubt, contact PICU/NICU pharmacist for advice (or on-call pharmacist via switchboard out of hours).

Laboratory investigations

- **Admission: Trakcare code ECLS1**

UE	FBC	
LFT	Coagulation	
Mg	ACT	
CRP	D-Dimers	
Blood Culture	Anti-Xa (Heparin assay)	ROTEM (Extem/Intem/Fibtem/Heptem)
	Anti-thrombin	

CXR & Cr USS currently need to be ordered seperately

- **0700Hrs: Trakcare code ECLS1**

UE	FBC	CXR
LFT	Coagulation	Cr USS (every day for 1st 3 days then
Mg	ACT	on a Monday, Wednesday, Friday
CRP	Anti-thrombin	unless clinical concern)
ABGas	Anti-Xa (Heparin assay)	
	D-Dimers	ROTEM (Extem/Intem/Fibtem/Heptem)

Venous gas to calibrate mvSaO2 line
CXR & Cr USS currently need to be ordered seperately

- **1300Hrs: Trakcare code ECLS2**

UE	FBC
AB Gas	Coag
	ACT

- **2100Hrs: Trakcare code ECLS3**

UE	FBC
LFT	Coagulation
Mg	ACT
AB Gas	

- **Hourly:** ACT

Remember: pre and post-membrane gases only need to be undertaken daily or if you have concern regarding membrane function.

Decannulation from ECMO

Low threshold for one off doses of Vancomycin +/- Gentamicin 2hrs pre-decannulation

Open neck/groin ECMO decannulation

See Weaning & decannulation checklist in guideline section

Personnel Required:

Paediatric or Cardiovascular surgeons
ECLS Physician
Theatre Staff
ECLS Specialists

Medications Required:

Vecuronium
Atropine
Fentanyl
Volume
Back-up PRBC's available

Preparation for decannulation is much like going on ECLS. The same physician staff and technical staff (except perfusionist) must be available and a team brief should take place to review plans/access/medical therapies etc. The emergency blood back-up unit should be brought to the ward and a syringe of blood (10 ml/kg.) should be available for transfusion and/or emergencies during the procedure (Not in percutaneous decannulation). The infant is placed in Trendelenburg position with a roll placed under the shoulders. Vecuronium is the chosen paralytic agent because it is an short acting neuromuscular blocking agent and last about 30-45 minutes. The infant must be paralysed during the removal of the venous catheter to ensure that no air is pulled into the catheter with a deep inspiration. A dose of 0.l mg/kg of vecuronium is used and may need to be repeated. Anaesthetic doses of fentanyl should again be used for this procedure (10-20 mcg/kg). Atropine should be available in case a bradycardia results from vagal stimulation.

Note re NICU: The neonate's ventilator settings should be turned up after being paralysed to an IPPV 40-50/min, FiO2 30-40%, PIP 15-20 mmHg (depending on chest movement), and Ti 0.5 sec. A pulse oximeter should be used to determine the amount of ventilator support needed. If lung compliance is normal the infant should not need more than that listed above. They should be allowed to breathe on their own as soon as possible following decannulation and a compensated respiratory acidosis should be aimed for on blood gas analysis. Most neonatal respiratory ECMO cases will extubate within 1-2 days of decannulation.

As the venous catheter is removed, the physician must supply an inspiratory hold on the ventilator until this catheter is out. Release of the inspiratory hold too early may result in air embolus.

Surgical procedure for ECMO decannulation

- **Positioning of patient:**

A shoulder roll is placed to fully hyperextend the patient's neck. The head is then turned to the left. A diathermy pad should be placed at this time if open decannulation.

- **Anaesthesia:**

The patient is sedated with Fentanyl (10-20 mcg/kg) and paralysed with vecuronium (0.1 mg/kg). These drugs are given by the ECLS Physician. The neck wound is anaesthetised with 0.5% lignocaine with 1:200,000 adrenaline.

- **Prep & Drape:**

A wide prep of the patient's chest and neck is done with Betadine solution. The right ear should be prepped and draped into the field. Towels are used to cover the infant as well as the entire bed. Steri-drape secures the towels to the skin of the neck and all but the right side of the surgical field. This portion is left open so that the cannulae can be moved out of the way of the field after removal.

- **Procedure:**

The incision in the neck is anaesthetised as previously described. The vicryl suture is cut and removed and the neck wound opened. Haemostatic packing is removed exposing the cannulae and vessels. The cannula that is most readily accessible should be removed first. This is usually the venous cannula as the vein lies more anteriorly in the neck. When the vein is isolated it is encircled with a 2-0 silk tie which is used for traction and haemostatic control. The cannula is freed from surrounding tissues. There may be an impressive degree of fibrosis between vein and surrounding tissue but a clean and complete dissection should be performed. The patient must be taken completely off ECLS (A-B-V).

Next a Satinsky clamp is placed around the vein and cannula and stabilised. The silk ties securing the cannula into the vein should then be cut with a #11 blade, the two proximal ties should be cut where they cross the bootie. As the catheter is withdrawn there is usually a blood loss of 5-10 ml before the Satinsky clamp can be secured. An inspiratory hold must be placed on the ventilator at the time the catheter is withdrawn. Also, it is useful if pressure is exerted on the liver by the assistant. These manoeuvres help prevent air embolism. The 2-0 silk is then tied. A second 2-0 silk tie is placed as a more distal ligature. The sutures can then be cut and the Satinsky clamp removed.

The common carotid artery and its cannula are then dissected free from surrounding tissues. The artery may be adherent to surrounding tissues and the dissection must be performed carefully. One must be certain that the Vagus nerve is freed from the posterior portion of the artery before proceeding with removing the cannula. The artery is then encircled with a 2-0 silk ligature as well as the Satinsky clamp. The three silk ties holding the cannula are then cut with a #11 blade. The cannula is removed then the Satinsky clamp is secured and 2-0 silk ligature tied. A second more proximal silk ligature is placed and secured both of these are cut and the Satinsky clamp removed. The wound is now irrigated with saline. Search for haemostasis is made and any obvious bleeders are cauterised. Wound closure is performed using of 4-0 or 5-0 undyed vicrly. The silk sutures holding the cannula behind the right ear are then removed.

A suitable dressing is applied.

- **Percutaneous Cannulae**

The same preparation is required for removal of percutaneously inserted cannulae. However, theatre staff are not required as the cannula can just be withdrawn and removed while applying pressure to the site. It is important to have some head-down and to get an inspiratory hold as in the open technique to prevent air embolism. Usually, a single suture is necessary. After the suture is inserted, a little head-up greatly facilitates haemostasis. The cannula tip should be sent for microbiology. Be patient!

- **Disposal of the ECLS circuit**

The used circuit is disconnected from the ECLS trolley and pump. Ensure the pressure lines are depressurised prior to removing from the transducers otherwise damage can occur to the transducer membranes. The open ends of the tubing are clamped with the blue clamps from the pressure lines to reduce leakage of blood. Ensure that all taps are off and no metal clamps are left attached to the system. Discard the circuit in the large "sharps" bin and apply an appropriate tag.

The trolley must be cleaned, along with all equipment. Moist towels should be used with a mild detergent. Alcohol solutions or Milton should **NEVER** be used with ECLS equipment. The water heater hoses are turned

off and the water (Silver Nitrate tablets are added during servicing to prevent bacterial growth) is left insitu.

Complete the check list and leave with circuit.

Return the ECLS trolley and consoles to the ECLS equipment room on the 2nd floor and **ensure** all equipment (especially both consoles) needing to remain on charge is plugged in. This is the responsibility of the ECLS Specialist, **not** the Porter.

Transthoracic ECMO decannulation

See Weaning & decannulation checklist in guideline section

Personnel Required:

Cardiovascular surgeons
ECLS Physician
Theatre Staff
ECLS Specialists

Medications Required:

Vecuronium
Atropine
Fentanyl
Volume
Adrenaline 1:100,00
Back-up PRBC's available

Patient preparation:

Pacing wires attached to pacing box (spare battery available) and accessible to ECLS physician
Volume line accessible to physician
Ventilation optimised
Paralyse and sedate patient
Anaesthetic T-piece accessible to ECLS physician
Ventilator tubing secure on ETT
ETCO2 working
SaO2 tone on

Procedure:

Team brief prior to initiation of procedure

Once patient is decannulated the chest wall is commonly left open but sealed with a GoreTex membrane to allow access to potential bleeding sites and minimise stress to ventricular function. It should be the aim to formally close the chest within 24-48 hrs of decannulation to minimise the risk of secondary infection.

Remember to undertake a chest X-ray once patient is stable.

Maintain paralysis for 12-24 hrs following decannulation to minimise oxygen demand.

Mobile ECLS

We now have the capacity to undertake mobile ECLS with the team consisting of a Retrieval Nurse Practitioner or ECLS Coordinator, trained Perfusionist, Consultant Paediatric Intensivist and if cannulation is required a surgeon.

There is a folder based in PICU, behind the nurse's station, as well as on the ECMO trolley which is in the retrieval store, which details roles, responsibilities and checklists for retrieval and referrals.

There are also SOP's with regard to all the equipment on the trolley.

All referrals should be documented on the transport forms.

Ventricular Assist Device chapter

Ventricular assist device (VAD) support has the aim of supporting either the left or right ventricle that has decompensated following cardiopulmonary bypass or cardiomyopathy/myocarditis when there is no significant lung pathology. The aim for this support is for it to be a "bridge to recovery" or bridge to transplant at a transplant centre.

VAD offers significant potential advantages over ECMO in that it requires less heparinisation as there is no oxygenator and therefore less thrombosis risk, there is minimal haemolysis and less exposure to plastic and therefore less inflammatory response to the circuit. Given the small volume of the circuit it also gives the opportunity to prime to saline rather than blood.

The over-arching aim is to minimise sedation, respiratory support and optimise enteral nutrition in this group. Extubation should be aimed for in those children with a predicted run of more than 7 days and good respiratory function, but only when enteral intake is well tolerated so that medications such as sedation and vasoactive agents can be switched to the enteral route. This is made possible by a tunnelled transthoracic cannulation route with immediate skin and sternal closure.

The aim of this guideline is to act as an "aide memoire". It is not set out as a bible on how and when to utilise centrifugal VAD technology.

We would like to thank all those who have contributed to these guidelines (knowingly and unknowingly).

Contents

Indications / Contra-indications for VAD

Triggers for multidisciplinary meeting to discuss potential use of VAD:

- Uni-ventricular failure post-bypass (non-HLHS patient)
- Primarily uni-ventricular failure in setting of end-stage heart failure as a portable
- rescue therapy to enable access to destination therapy or long term bridge
- Uni-ventricular failure secondary to cardiomyopathy/myocarditis if neck cannulation
- for ECMO is discounted
- Wean from VA-ECMO in setting of uni-ventricular failure

This MDT should include Paediatric Intensivist, Cardiologist, Cardiothoracic surgeon +/- Cardiac Anaesthetist

Contra-indications for VAD:

- Bi-ventricular failure
- Significant lung disease – O2 requirement >50% or high pressure/tidal volume
- ventilation
- Atrial or Ventricular Septal Defect
- Severe pulmonary hypertension if LVAD support (PA pressures >50% systemic)
- Severe Aortic regurgitation if LVAD / Severe Pulmonary valve regurgitation if RVAD

Cannulation considerations

Intra-operative interventions:
- Nasal endotracheal intubation – uncut tube
- Consider use of Edwards mvSaO2 line (triple lumen) or tunnelled double lumen CVL
- Cannula in groin (to enable wiring of haemofiltration line if needed)
- Arterial access
- Nasogastric tube
- Urinary Catheter
- Near Infra-red Spectroscopy (NIRS)

Basic monitoring:
- ECG
- Invasive BP
- CVP
- Continuous mvSaO2 monitor
- NIRS
- $_{ET}CO2$

Cannulation:
Most cannulations will take place in cardiac theatre after problems disengaging from cardio-pulmonary bypass successfully. Patients will normally be fully heparinised from bypass hence no heparin bolus will be required.

- Cannulation is always transthoracic
- Antibiotic prophylaxis
  - Cefuroxime immediately pre & post cannulation then dose for 24Hrs only
  - Take blood culture from central line post-cannulation
- Appropriate cannula selection to allow up to 200ml/kg flow
- If weaning from bypass patient suitability for VAD support may be confirmed as follows
  - LVAD : clamp right atrial drainage cannula and run on L atrial drainage to Aortic cannula flow, observing CVP, cardiac function, echo and ventilator parameters. Patient may need full ventilatory support and some right heart inotropic and or lusitriopic support. Nitric Oxide should not be needed to redice pulmonary pressures as this would indicate a requirement for VA-ECMO support rather than VAD.
  - RVAD : Site Pulmonary outflow cannula and then clamp left atrial drainage cannula and aortic cannula and observe cardiac function over 20-30 minutes on full ventilation with inotropic +/- dilation support for left heart.
- Complete ECLS briefing as per support tool
  - Heparin bolus 50iu/kg prior to cannulation if not on bypass
  - Initiate heparin infusion @ 25iu/kg/hr once drain losses <5mls/kg/hr (see coagulation section)
- At initiation of support Increase revs to 1500-1700rpm
  Unclamp “Blue” venous limb

Unclamp “Red” return limb
Slowly increase RPM till desired flow achieved

- Echo re cannula position
- Aim to close sternum & skin
- Wean inotropic support as in cardiovascular section
- Ventilate to normal CO2/O2 as per respiratory section

Circuit prime

- Patients > 10kg may be placed onto VAD support with a clear prime.
  - Heparin 1iu/ml Plasmalyte A
  - Standard blood prime should be used otherwise (see blood prime checklist)

VAD specific patient management issues

Daily VAD orders
These should be written in CIS on a daily basis by the Duty ECLS Intensivist or senior PICU trainee who is ECLS trained. These should be updated if the orders are adjusted.

Chest re-exploration
- The need for re-exploration should be minimised if possible by having sternal & skin closure.
- There is a significant risk of introducing air into the circuit at the time of chest exploration. This should therefore only be undertaken with perfusion staff present and all emergency drugs available.
- Consultant intensivist must be at bedside.
- Re-exploration only to be undertaken by Consultant Cardiac Surgeon.
- One ECLS specialist should watch patient and one watches circuit for potential air intake or other circuit issues along with perfusion staff.
- RPM should be reduced to approx 50-75% set levels (ensure above minimum flows) to minimise risk of air embolism. The patient's haemodynamic stability should be carefully monitored during this time.
- Once re-exploration completed there should be skin closure or a goretex membrane should be placed over wound and then rpm can be raised.
- Coagulation management may need to be addressed to limit future re-explorations.

Weaning
- Consider use of Levosimendan 48Hrs prior to attempted wean of VAD flows.
- Consider flexible bronchoscopy to ensure no residual airways issues that may complicate wean from VAD support.
- Wean over 24Hrs with introduction of Milrinone +/- low dose adrenaline. A loading dose of Milrinone 30-50mcg/kg may be considered.
- Complete VAD wean checklist.
- Consider stopping Phenoxybenzamine if prescribed 24Hrs prior to planned wean and decannulation.
- Paralyse and keep well sedated for period of wean and for 24Hrs after decannulation as directed by ECLS physician
- Ensure normothermia with regular anti-pyretics +/- active cooling to minimise cardiac work.
- Wean flows by 5-10% every hour to minimum flow ratings as described (Pedivas 200ml/min & Centrimag 500ml/min) or as prescribed by ECLS physician. **Remember to increase ACT by 20 as approach minimum flow** to minimise risk of clotting in VAD circuit.
- As RPM wean watch for retrograde flows especially if patient becomes hypertensive.
- Paired gases & Echo at minimum flows to confirm success or otherwise of wean.
- Once wean is successfully completed decannulated asap or increase support if decannulation is to take place the following morning.
- Aim to decannulate as first case of day or by 1200Hrs at latest.

Decannulation

- Should be undertaken as soon as is practical and patient has demonstrated a safe wean to minimal settings.
- This should be undertaken ideally in the morning of a normal working day.
- Ensure repeat echocardiogram immediately prior to decannulation to confirm function appropriate for removal of VAD support.
- Decannulation may take place in either the PICU (most usual) or theatre. This will be decided by the Intensivist/Cardiac surgeon.
- Ensure well sedated and paralysed prior to and for 24Hrs after decannulation
- Chest X-ray after decannulation when patient stable

Moving the patient on VAD

- Complete VAD patient transfer sheet (see guidelines section)

• Minimum staff group:	ECLS Physician or Cardiac Anaesthetist ECLS Specialist x 2 Perfusionist Nurse runner
• Battery life of Levitronix	60 mins at 5500 rpm, 3 Lpm
• Equipment:	Portable monitor (fully powered) Oxygen cylinder (full) Appropriate bed for lift Resuscitation drugs Volume (50ml/kg) ACT LR cuvettes / Elite Internal transfer bag Pacemaker Transport ventilator Back-up pump and console
• Prior to move:	ECLS specialist to assess destination of transfer and Route and ensure awareness of UPS socket positions Remove unnecessary equipment Secure Cannulae Place patient on transport ventilator Place pump head on pole at patients head Place Console on bed with patient Ensure back up console fully charged
• At destination:	Plug primary and back-up consoles in to UPS mains socket Conduct full circuit and patient check

Cardiovascular Management of VAD patients

Hypertension

This is relatively common in all ECLS patients.

- Ensure sedation and pain relief adequate
- Exclude possibility of seizures
- Review use of catecholamine's
- Short acting vasodilators
  - Sodium Nitroprusside 0.5-4mcg/kg/min
  - Esmolol may be used
- Longer acting vasodilators
  - Milrinone 0.3-0.75mcg/kg/min +load
  - Labetolol may be used
  - Phenoxybenzamine 1mg/kg load over 4 hr then 0.5mg/kg tds
    Avoid Phenoxybenzamine for 24Hrs pre-wean and decannulation

Hypotension

Causes include the following:

- Inadequate VAD flows
  Slowly increase rpm
- Patient underfilled:
  May need volume (5ml/kg or less aliquots) and transient dropping of flows by ~10% to allow re-filling of left or right atrium
- Poor venous or arterial cannula position:
  echo
- Too much systemic vasodilatation:
  Wean SNP / stop Phenoxybenzamine and consider vasopressin to counteract alpha effects of phenoxybenzamine
- Inadequate respiratory support with CO2 rise
  Ensure good chest movement
  Check gas, suction ETT, optimise respiratory support
- Sepsis: Surveillance cultures, FBC, White cell diff and CRP
  If rise in inflammatory markers treat with 1st line antibiotics
  Consider steroids – PICU Consultant only approval
  Consider IVIG – PICU Consultant only approval
- Consider:
  Tension pneumothorax
  Pericardial effusion

Failing right ventricle

In LVAD supported patient it is essential to be aware of a failing right ventricle. Signs would include:

- Fall in flow because of reduction of preload to left ventricle.
- Increasing CVP, enlarged liver

Actions should include:

- Paired blood gases
- Echo re RV function and discussion with Cardiology & Cardiac Surgical teams

Pulmonary Hypertension

- If pulmonary pressures are greater than 50% systolic pressures then LVAD is **not** the support method of choice, VA-ECMO should be used.
- Inhaled nitric oxide, starting at a maximum of 20ppm, should be considered if PAP becomes >50% systolic pressures whilst on LVAD. Fentanyl 1-2mcg/kg should be given pre-endotracheal suctioning to mediate acute rises in PA pressures.

Respiratory management of VAD patients

- It is imperative to ventilate these patients normally, VAD does **not** have an Oxygenator.
- Set ventilator as routine, aiming for expiratory tidal volume of 6-10ml/kg with optimised PEEP and aim for an FiO2 <60% if possible to avoid "biotrauma" to lungs. It is essential for a successful VAD run to minimise any potential ventilator induced lung injury. Remember if the chest is open then tidal volumes of 4-6ml/kg may be adequate.
- Low threshold for investigating potential of ventilator associated pneumonia (VAP) with blind broncho-alveolar lavage (Blind BAL) or bronchoscopic BAL.
- Routine suctioning should undertaken. Fentanyl should be used (1-2mcg/kg) if there is evidence of pulmonary hypertension.
- Patient should be positioned with a head-up position and ventilator tubing dependant to avoid "rain-out" of ETT humidity. This should not overly the chest wound. When suctioning place red cap over end of ventilator tubing to avoid dispersal of secretions from ventilator tubing.
- Daily CXR should be undertaken
- The VAD specialist nurse and assistant nurse will perform regular respiratory care, to avoid respiratory compromise. As with all nursing interventions the VAD specialist should visualise the circuit and cannulae at all times. Ensure safety equipment available at all times. Medical staff should be available to provide support.
- The requirement for physiotherapy intervention will be discussed with the lead consultant and senior physiotherapist and risk factors discussed and assessed.
- The aim should be for extubation once enteral nutrition is tolerated if a VAD is expected to be required for more than 5 days. This can only be safely be undertaken once sedation and vasoactive medication is administered enterally.

Physiotherapy:

The VAD specialist nurse should perform regular respiratory care, to avoid respiratory compromise.

When Physiotherapy in attendance, as with all nursing interventions, the VAD specialist should visualise the circuit and cannulae at all times. Ensure safety equipment available at all times. Medical staff should be available to provide support if required.

The requirement for physiotherapy intervention must be discussed with the duty Intensivist and senior physiotherapist and the risk factors discussed and assessed.

Sedation in VAD patients

The aim should be to minimise intravenous sedation once bleeding has stopped. VAD patients with closed chests should be able to be extubated once enteral nutrition and sedation is tolerated.
Regular Comfort scores will help ensure sedation is optimised.
Infusion of paralytic agents should only be used if there are severe bleeding concerns or in the process of a planned wean from VAD.

Renal management of VAD patients

Diuresis drive with furosemide is almost always essential. Furosemide infusions (in 20% albumin) and enteral spironolactone are the method of choice. Close monitoring of potential renal dysfunction is essential.

- Furosemide (in 20% Albumin) 0.1-1mg/kg/hr ivi
- Peritoneal dialysis: Catheter should be left on free drainage if not used
- Haemofiltration: Groin line can be wired up to allow CVVHF. This line will be sited in theatre. Haemofiltration will not be possible via VAD circuit to minimise risk of air embolus.

Anti-coagulation management in VAD patients

Coagulation management

- 50 iu/kg Heparin bolus to patient prior to cannulation if not initiated in theatre direct from bypass.
- Start Heparin ivi at 25iu/kg/hr once ACT falls to 250 **and** blood loss less than 5ml/kg/hr.
- 1/4hrly ACT until within parameters then 1hrly if stable.
- ACT should be used to guide heparin dosing. If out of keeping with clinical picture then review extended coagulation results with duty Intensivist and plan coagulation management.

Baseline targets

- ACT 200-220 if stable
- Platelets >100
- Fibrinogen >2.0

The standard extended ECLS anti-coagulation assays (aPTT, Anti-thrombin, D-dimers, anti-Xa, Fibrinogen) should be sent which are highlighted in the lab investigation section. These should not replace the ACT as the bedside test to manage the heparin dosing. They may help guide the duty Intensivist in planning or updating the daily VAD orders. If bleeding is a concern a ROTEM is advised to target specific blood product replacement or to highlight normal coagulation and therefore surgical aetiology of bleeding.

Procedures such as nasal re-intubation, chest drain placement, arterial or central line placement should only be undertaken under Consultant supervision and if coagulation is carefully controlled and stable.

Standard ACT ranges for centrifugal ECMO or VAD circuit:

- ***Low risk of bleeding***
  - ***ACT's 200-220 sec***
    - ***When Pediyas circuit & flow <300 ml/min: run ACT's 210-230 sec***

Bleeding patient

Some bleeding is to be expected, up to a maximum 5 ml/kg/hr. If there is a rapid increase or rapid decrease contact PICU Consultant and reduce ACT to 180 – 200

The incremental strategy for bleeding ("Bleeding Bundle"):

1. Aggressive correction of coagulopathy
- Discuss urgently with ECLS physician/surgeon
- Fibrinogen >2 using cryoprecipitate
- Platelets > 120,000 up to 150,000
- Correct PT using FFP +/- Vitamin K
- Reduce ACT parameters to allow lower heparin dosing
- Check APTT, TEG
- Maintain high ionised calcium levels

2. Consider aprotinin (1ml/kg/hr)

3. Call ECLS physician & surgeon if blood loss is excessive
- >10ml/kg/hr despite optimising above
- Discuss with ECLS physician and surgeon regarding re-exploration of chest

4. If bleeding remains excessive after re-exploration or 2nd exploration within 12-24 hours
- Novo7 (recombinant Factor VIIa) can also be used at a dose of 95mcg/kg (see Novo7 guideline in appendix or medicine guideline on CIS) at the discretion of the ECLS physician. A clear primed circuit should always be available and surgical and perfusion staff should be aware of the first administration because of the risk of clotting within the circuit, especially if there is evidence of existing significant thrombus.
- Consider conversion to neck cannulation

5. Ongoing correction of clotting screen

Extreme circumstances (bleeding >30ml/kg/hr) despite all of the above.
- Run ECLS circuit without heparin
- ? decannulation

Clots in circuit

This is an emergency as this can lead to systemic embolisation.
- Contact PICU consultant, Perfusion & Cardiac surgeon
- Increase ACT parameters by 20
- Consider checking Heparin level, aPTT and anti-thrombin
- Consider increasing flows to safeguard circuit integrity

Emergency procedures

See also laminated "emergency ready reckoner" on pump trolley

- **Clamping circuit**
  - Always clamp red lumen (return limb)
  - Only clamp red (return) then blue (outward limb) if changing pump head
- **CPR**
  - This has inherent dangers in a fully heparinised patient with large cannulae in the heart which may become dislodged or entrain air.
  - 1 Nurse watches circuit for possibility of air entrainment and to look after pump.
  - If LVAD support then during CPR wean flows to 75% set levels (ensure above minimum flows).
  - If RVAD support then during CPR wean flows to 30% set levels (ensure above minimum flows).
  - Do not clamp off VAD unless air entrained during CPR.
  - Switch on back-up pump in case of unanticipated pump failure.
  - Consider pacing.
  - Inform cardiac surgical consultant if CPR on-going or resolved +/- perfusion.
  - Be aware that significant increase in afterload secondary to high dose adrenaline will increase the resistance that the pump has to work against therefore rpm may need to be increased significantly until this has worn off.
- **Defibrillation**
  - This can be safely done on VAD
  - Wean flows to 50-75% baseline during defibrillation (ensure above minimum flows)
  - The patient's haemodynamic stability should be carefully monitored during this time.
  - Ensure rpm and flow of VAD returns to baseline
  - Switch on back-up pump in case of unanticipated pump failure
- **Pump failure**
  - Clamp red lumen (return limb).
  - Turn on power back-up console (switch at R side)
  - Turn off main console (press stop button for 2 secs or switch off at R of console
  - Once rotor stationary unlock retaining screw & rotate head clockwise, lift out from motor.
  - Transfer head to back up motor. Insert and twist anticlockwise then tighten retaining screw (ensure groove matches screw position).
  - If alarms "pump not inserted" then acknowledge alarm as cannot increase revs till alarm acknowledged.
  - Increase revs to 1500-1700rpm.
  - Unclamp red lumen
  - Attach flow meter of back up console and slowly increase revs to previous level
  - Call perfusion for replacement console

Trouble shooting Levitronix VAD centrifugal pump

Never clamp venous (blue) lumen without clamping arterial (red) lumen first as this will cause cavitiation of gas in blood within device.

- **Retrograde flow**

  Retrograde flow can occur at low RPM or during significant increases in BP and will cause acute clinical deterioration. Retrograde flow will be displayed on Levitronix console as - - - . If this is displayed immediately clamp tubing post pump head.

  Action: Immediately clamp tubing post pump head
  Observe that flow probe is correctly placed on tubing
  Increase RPM by approximately 10%.
  Slowly release clamp observing for signs of forward flow.
  Once support has been achieved wean RPM to give desired flow rate.

- **Pump failure**

  See "emergency procedures"
  If pump has been stopped for more than 5 mins there is a significant risk of thromboembolism.

- **Clots in circuit**

  See "Anti-coagulation management in VAD" section

- **Reduction in flow**

  Can be caused by fall in patient's intravascular volume or by obstruction to flow into **venous (blue) cannula**.

  Action: Assess patients filling pressures (CVP)
  Volume 5ml/kg PPS
  Reduce rpm by 10% to allow filling of RA or LA depending on venous cannula position before slowly increasing rpm watching flows on pump console.

Laboratory investigations
(As per ECMO times)

- **Admission: ECLS1**

UE	FBC	CXR – order separately
LFT	Coagulation	Cr USS - order separately
Mg	ACT	Blood Culture
CRP	TEG	
	Anti-Xa	
	Anti-thrombin	
	D-Dimers	

- **0700Hrs: ECLS1**

UE	FBC	CXR - order separately
LFT	Coagulation	Cr USS - order separately
Mg	ACT	Blood Culture
CRP	TEG	
ABGas	Anti-Xa	
	D-Dimers	

Venous gas to calibrate mvSaO2 line

- **1300Hrs: ECLS2**

UE	FBC
AB Gas	Coag
ACT	

- **2100Hrs: ECLS3**

UE	FBC
LFT	Coagulation
Mg	ACT
CRP	TEG
AB Gas	Anti-Xa

- **Hourly:** ACT

VAD Specialist shift plan

Patient care

The care of the patient on VAD is very similar to that of any critically ill patient who requires intensive medical and nursing management. The added care involved is that of managing the VAD circuit and understanding the interaction between the VAD and the patient.

Whilst on VAD the patient will be cared for by two nurses. At the beginning of the shift care can be divided such that one cares for the child and the other the VAD circuit.

Every shift:

1. Complete VAD checklist.
2. Check available blood components with blood bank.
3. Confirm emergency drill
    - Who is duty Intensivist / Perfusionist / Surgeon
    - Spare circuit in VAD trolley
4. Verify and compare accuracy of the Heparin infusion and all other infusions
5. **Discuss emergency procedures with support staff and outline procedures if patient has to come off VAD SUPPORT.**

Hourly checks:

1. Vital signs recordings.
    - Record pump RPM and Flows in Litres per minute
    - Check upper and lower alarm limits.
    - ACT and maintain within set parameter
2. Ventilator recordings and check air entries.
3. Check arterial and venous cannula sites.
4. Check VAD cannulation site.
5. Measure and chart all drain losses, urine output and gastric losses.
6. Chart all input, infusions, feeds etc.
    - Replace blood products as required within parameters
    - Check fluid balance

4 hourly checks:

1. Patient blood gases arterial and venous, more frequently if weaning or unstable.
2. Endotracheal and oral suction.
3. Neurological checks, more frequently if concerns.
4. Pressure area care.
5. Eye and mouth care.
6. Passive limb exercises.

8 hourly checks:

Blood is sent for routine laboratory testing circa 8 hourly. This may be required more frequently e.g. during CVVH or excessive bleeding.
Bloods are taken 0700, 1300 and 2100hrs.
See investigation sheet next page

Microbiology Testing

Routine screening
Nil

Blood cultures
Blood cultures daily or as clinically indicated.

Appendix

VAD competencies

Name……

Date……

	Procedure	Performed
1	Discuss indications and contraindications for VAD support	
2	List Components of Levitronix System and functions	
3	Discuss rationale for right heart and left heart support on VAD	
4	Demonstrate set up of system, insert pump head and place flow probe correctly	
5	Demonstrate the selection of appropriate RPM/flows, setting of alarms and battery checks	
6	Discuss minimum recommended RPM for adult/child during initiation of VAD support	
7	Discuss maximum recommended flow rates for Centrimag /Pedivas pump	
8	Demonstrate sequence for clamping on and off VAD	
9	Discuss reasons for increased/reduced flows	
10	Discuss which patient parameters can assist in the assessment/troubleshooting of changes to flows	
11	Discuss the role of ECHO in management of VAD patients	
12	Discuss initial and ongoing management of anticoagulation on VAD	
13	Demonstrate management of Console/Motor failure	
14	Demonstrate management of retrograde flow	
15	Demonstrate management of air in system	
16	Discuss the management of CPR and Defibrillation during VAD support	
17	Discuss weaning guidelines and minimum flow rates	
18	Discuss specialist responsibilities at decannulation	
19	Discuss cleaning of equipment	
20	Discuss the correct storage of Levitronix Consoles	

Assessor Signature: …….

Specialist Signature: ……..

Appendix

ECMO Circuit sizing chart

Patient / Circuit specifications				
Patient weight	**< 4.5kgs**	**4.5-10kgs**	**10-18kgs**	**> 18kgs**
Arterial connector size	**1/4 “**	**1/4 “**	**1/4 “**	**3/8 “**
Venous connector size	**1/4 “**	**1/4 “**	**3/8 “**	**3/8 “**
Pedivas Pump Prime	14ml	14ml	-	-
Centrimag Pump Prime	-	-	31ml	31ml
Oxygenator	Medos 0800 LT	Medos 2400 LT	Medos 2400 LT	Medos 7000 LT
Oxygenator priming volume	55ml	95ml	95ml	275ml
Max Blood Flow through Oxygenator	800 ml/min	2400 ml/min	2400 ml/min	7000 ml/min
Min Pump Blood Flow	200ml/min	200ml/min	500ml/min	500ml/min
Max Gas Flow through Oxygenator	1.6 l/min	4.8 l/min	4.8 l/min	14 l/min
Min Gas Flow through Oxygenator	0.2 l/min	0.2 l/min	0.2 l/min	0.2 l/min
Flow Probe	1/4 “	1/4 “	1/4 “	3/8 “
Total Circuit prime	**155ml**	**195ml**	**270ml**	**490ml**

ECMO Cannula sizing chart

Cannulae Selection Guidelines							
VA			VV				
Biomedicus cannulae			Origen DL cannula		Avalon bi-caval cannula		
Weight	Arterial cannula size	Venous cannula size	Weight	Cannula size	Weight	Cannula size	Max flow
2 – 3 kg	8F	8F/10F	2.5 -3.5 kg	12F	2.5-5 kg	13F	0.5l/min
3 – 6 kg	10F	10F/12F/14 F	3.5 – 6 kg	15F	4 -9 kg	16F	0.9l/min
6 – 11 kg	12F	14F	5 – 11 kg	18F	<12 kg	19F	1.2l/min
11 – 14 kg	12F	14F/15F	>11 kg	N/A	<15 kg	20F*	1.5l/min
14 – 20 kg	14F	15F/17F	-	-	<20 kg	23F	2.0l/min
20 – 30 kg	15F	17F/19F	-	-	Size clinically **	27F	3.5l/min
30 – 40 kg	17F	19-21F	-	-	Size clinically **	31F	5.0l/min
> 40 kg	21F	21-23F	-	-	> 75 kg	31F PLUS additional drainage cannula	

ECMO Circuit specifications

Console / Circuit specifications		
Pump head selection	**Pedivas <10 kg**	**Centrimag > 10 kg**
Pump Speed Range (revolutions/min)	0 - 5500 RPM.	0 - 5500 RPM.
Pump Flow: Minimum (litres/min)	0.2 L/min	0.5 L/min
Pump Flow: Maximum (litres/min)	1.7 L/min	9.9 L/min
Recommended RPM at initiation of flow	1500	1500
Speed step resolutions	50ml	100ml

VAD cannula & circuit sizing chart

VAD Console / Circuit specifications		
	Centrimag	**Pedivas**
Tubing Size	3/8" inlet / outlet >10kgs	¼" inlet / outlet < 10kgs
Pump Speed Range	0 - 5500 revolutions per minute (RPM).	0 - 5500 revolutions per minute (RPM)
Pump Flow: Minimum	0.5 litres per minute (LPM)	0.2 litres per minute (LPM)
Pump Flow: Maximum	9.9 litres per minute (LPM)	1.7 litres per minute (LPM)
Priming Volume		

Patient specifications		
	Centrimag > 10kgs	**Pedivas <10kgs**
Recommended RPM at initiation of flow	1200	1500 – 1700
Speed step resolutions	100ml	50ml

VAD Cannulae Flow Specifications			
Venous side All Terumo Tenderflow wired angled		**Arterial side**	
ml/min flow	**Cannula**	**ml/min flow**	**Cannula**
0 -500	14Fr	0 -400	2.6 mm Stockert
500 – 1000	16Fr	400 – 750	3.0 mm Stockert
1000 – 1500	18Fr	750 – 1000	3.5 mm Stockert
1500 – 2000	20Fr	1000 – 1400	4.0 mm Stockert
2000 – 2500	22Fr	1400 – 1700	14Fr DLP
2500 - 3000	24Fr	1700 – 2000	16Fr DLP
		2000 - 2500	20Fr DLP
		2500 – 3000	21Fr DLP

Predicted cardiac output calculations

NEONATAL CARDIAC OUTPUT CALCULATIONS

Name: .....
Hospital number: .....
Date of birth: .....

Weight: ..... kg

Calculate Cardiac Output at 200 ml/kg/min = 100%

100%	..... ml/min
90%	..... ml/min
80%	..... ml/min
70%	..... ml/min
60%	..... ml/min
50%	..... ml/min
40%	..... ml/min
30%	..... ml/min
20%	..... ml/min
10%	..... ml/min

This is just an estimate as it is based on average neonatal cardiac output

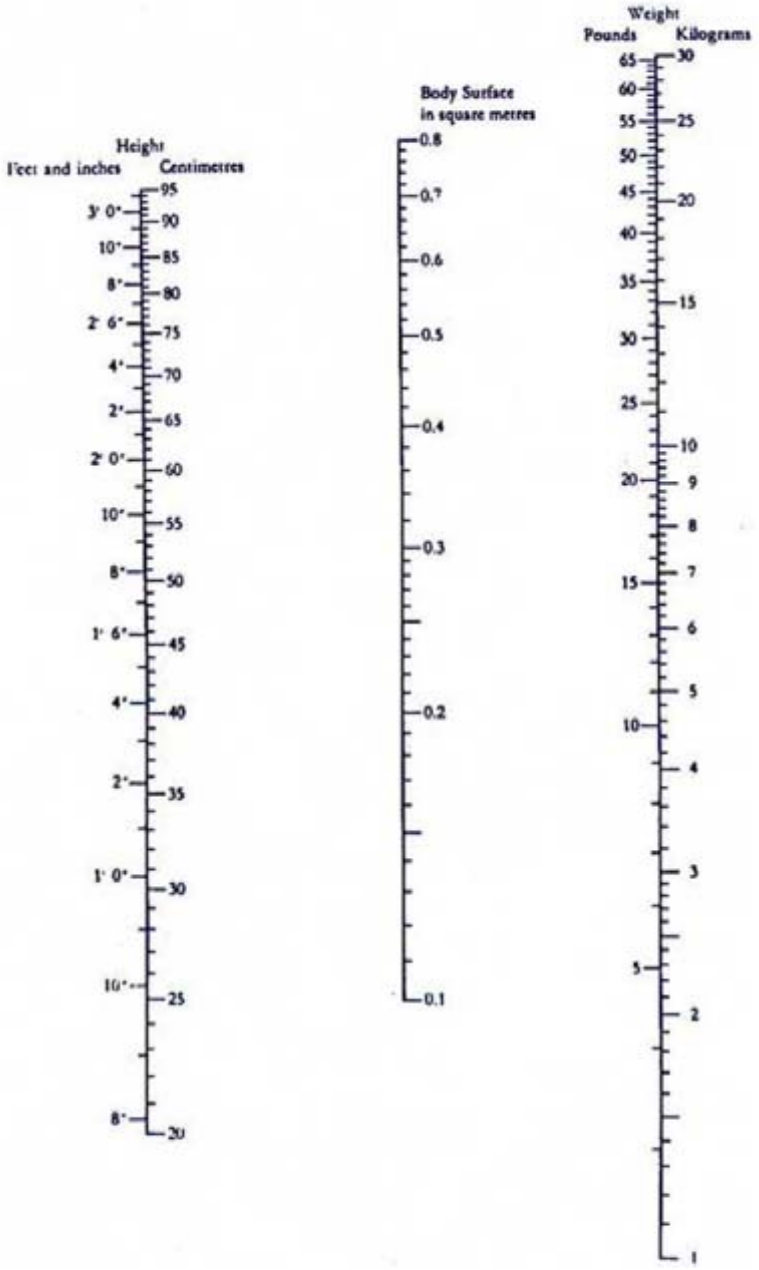
Round results to the nearest "10"

C Davis, December 2004

CARDIAC OUTPUT CALCULATIONS ≤30 kg

Find the weight in the right column and the height (length) in the left column. Place a straightedge on the nomogram so the weight and height are connected. The point where the straightedge crosses the centre column denotes the body's surface area in square meters.

Make note of the body surface area for calculations.



C Davis, December 2004

CARDIAC OUTPUT CALCULATIONS ≤30 kg

Name: .....
Hospital number: .....
Date of birth: .....

Weight: ..... kg

Calculate Cardiac Output:

Body surface area (m^2) = ... x 2.4 = ... ml/min = 100% CO

100%	..... ml/min
90%	..... ml/min
80%	..... ml/min
70%	..... ml/min
60%	..... ml/min
50%	..... ml/min
40%	..... ml/min
30%	..... ml/min
20%	..... ml/min
10%	..... ml/min

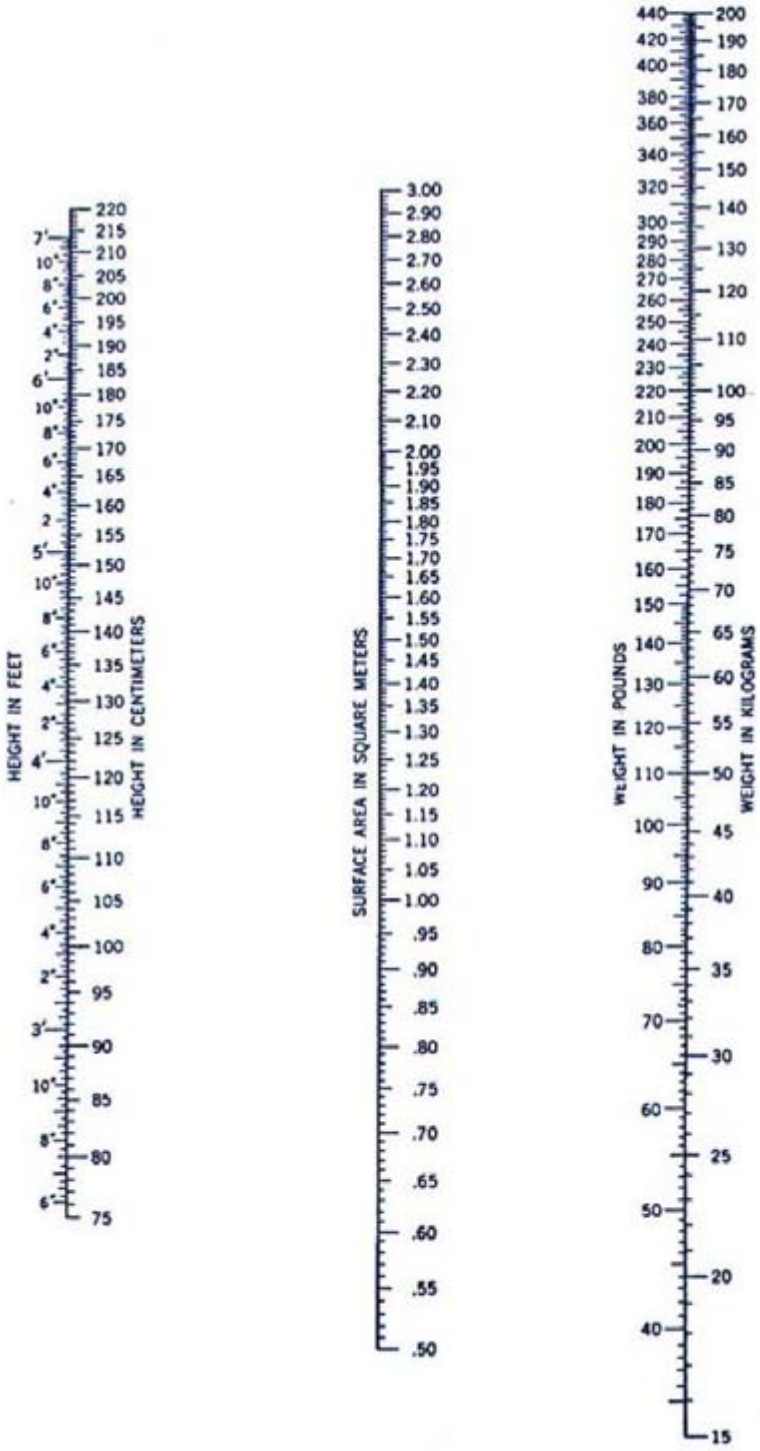
This is just an estimate as it is may be altered by the patient's clinical condition and medication.

Round results to the nearest "10"

CARDIAC OUTPUT CALCULATIONS >30 kg

Find the weight in the right column and the height (length) in the left column. Place a straightedge on the nomogram so the weight and height are connected. The point where the straightedge crosses the centre column denotes the body's surface area in square meters.

Make note of the body surface area for calculations.



C Davis, December 2004

CARDIAC OUTPUT CALCULATIONS >30 kg

Name: .....
Hospital number: .....
Date of birth: .....

Weight: ..... kg

Calculate Cardiac Output:

Body surface area (m^2) = ... x 2.4 = ... ml/min = 100% *CO*

100%	..... ml/min
90%	..... ml/min
80%	..... ml/min
70%	..... ml/min
60%	..... ml/min
50%	..... ml/min
40%	..... ml/min
30%	..... ml/min
20%	..... ml/min
10%	..... ml/min

This is just an estimate as it is may be altered by the patient's clinical condition and medication.

Round results to the nearest "10"

ECLS CIRCUIT BLOOD PRIME

Addressograph	Date: ..... Time: .....

Batch/Laboratory Number: ..... &
Expiry date of Albumin 20%: .....

To each Unit of PRBC in Prime:

1. Add 20 ml 20% Albumin
2. Add 25ml THAM
3. Add 10 mmol $NaHCO_3$
4. Add 150 units Heparin
5. Agitate well (by rocking)
6. **Then**, add 3 ml 10% Calcium gluconate and agitate again

UNIT	UNIT NUMBER:	ADDED BY:	CHECKED BY:
1.	.....	.....	.....
2.	.....	.....	.....
3.	.....	.....	.....
4.	.....	.....	.....

Prime volume in the circuit:

Add volume to the primed circuit until the post-oxygenator pressure measures 100 mmHg at a pump rate of 200 ml/min

Doctor's signature: .....

Heparin calculations

HEPARIN CALCULATIONS

(If <40kg in weight)

Name: .....
Hospital number: .....
Date of birth: .....

Weight: ..... kg
Heparin batch number: .....

A. LOADING DOSE OF HEPARIN

Loading dose: ..... *Units Heparin/kg weight*

Calculation:

..... Units Heparin/kg = ... Units Heparin

..... Units Heparin = ... ml Heparin (1,000 Units/ml)

B. HEPARIN INFUSION

Calculation:

..... (kg) x 25 Units/ml = ... Units Heparin/ml

..... Units/ml x 50 = ... Units/50 ml 5% dextrose

$$\frac{\text{.....}}{\text{1000 (Units/ml)}} = \text{..... ml Heparin}$$

50 ml 5% dextrose - ..... ml Heparin = ... ml 5% dextrose

Date: .....

Calculated by: .....
Checked by: .....

Doctor's signature: .....

Heparin infusion must be prescribed on Drug Kardex and Fluid Prescription Chart

C Davis, April 2008

HEPARIN CALCULATIONS
(*If over 40kg in weight)*

Name:	.....
Hospital number:	.....
Date of birth:	.....
Weight:	..... kg
Heparin batch number:	.....

A. LOADING DOSE OF HEPARIN

Loading dose: ..... *Units Heparin/kg weight*

Calculation:

..... Units Heparin/kg = ... Units Heparin

..... Units Heparin = ... ml Heparin (1,000 Units/ml)

B. HEPARIN INFUSION

Calculation:

..... (kg) x 12.5 Units/ml = ... Units Heparin/ml

..... Units/ml x 50 = ... Units/50 ml 5% dextrose

$$\frac{\text{.....}}{\text{1000 (Units/ml)}} = \text{..... ml Heparin}$$

50 ml 5% dextrose - ..... ml Heparin = ... ml 5% dextrose

Date:	.....
Calculated by:	.....
Checked by:	.....
Doctor's signature:	.....

Heparin infusion must be prescribed on Drug Kardex and Fluid Prescription Chart

C Davis,May 2009

ECMO Perfusion checklist

DATE: _____ HOSP.NO:_____

PATIENT: _____ WEIGHT _____ kg

PERFUSIONIST 1. _____ PERFUSIONIST 2. _____

PACK: _____ ser.no. _____

OXYGENATOR: _____ ser.no. _____

PUMP HEAD: _____ ser.no. _____

BASE NO: _____ WATER HEATER NO: _____

PUMP NO: _____ GAS BLENDER NO: _____

K^+: _____ mmol/l

1.	Check both consoles are fully charged	
2.	Correct circuitry selected based on weight	
3.	Circuit primed and bubble free	
4.	Flow probe connected	
5.	Bionectors on all pigtails	
6.	Pre and Post pressure lines zeroed and taped upright	
7.	Venous pressure cable from patient monitor connected and zeroed	
8.	Potassium level measured (only if blood primed)	
9.	Gas blender FiO_2 set at 100%	
10.	Water heater connected and temperature set	
11.	Check O_2 cylinder is over ½ full	
12.	Spare console/motor/flow probe available and plugged in	

CANNULAE: _____ ser.no. _____

_____ ser.no. _____

ECMO HANDOVER

ECMO SPECIALIST 1._____ ECMO SPECIALIST 2._____

1.	Heparin infusion connected & tap open	
2.	Heparin Infusion running	
3.	Venous pressure displayed on patient monitor and 5 to -50mmg	
4.	Pre/Post pressure displayed on console	
4.	Upper and lower pressure alarm limits set	

COMMENTS:

VAD Perfusion checklist

DATE: _____ HOSP.NO:_____

PATIENT: _____ WEIGHT _____ kg

PERFUSIONIST 1. _____ PERFUSIONIST 2. _____

PACK: _____ ser.no. _____

PUMP HEAD: _____ ser.no. _____

BASE NO: _____ WATER HEATER NO: _____

PUMP NO: _____

K^+: _____ mmol/l

1.	Check both consoles are fully charged	
2.	Correct circuitry selected based on weight	
3.	Circuit primed and bubble free	
4.	Flow probe connected	
5.	Potassium level measured (only if blood primed)	
6.	Spare console/circuit/motor/flow probe available and plugged in	

CANNULAE: _____ ser.no. _____

_____ ser.no. _____

VAD HANDOVER

ECLS SPECIALIST 1._____ ECLS SPECIALIST 2._____

1.	Heparin infusion connected to patient & tap open	
2.	Heparin Infusion running	

COMMENTS:

ECMO VA Trial Off Checklist

PATIENT NAME _____ **DATE** _____

1.	Wean and bridge off discussed/documented with ECLS physician & team • Ventilation optimised (? Bronchoscopy / NO) • Cardiac support optimised (inodilators/pacing etc) • Adequate sedation & paralysis	
2.	Recent ECHO performed to assess cardiac function	
3.	ECMO flows weaned as per plan *Minimum blood flows for the circuits are as follows:* • *Pt wt <10Kg minimum blood flow is 0.2l/min* • *Pt wt >10kg minimum blood flow is 0.5l/min*	
4.	Surgeon aware of planned trial off and potential for decannulation	
5.	Patient's ABG/VBG and vital signs stable on reduced ECMO flows	
6.	Recent CxR performed & reviewed	
7.	Inotrope / Vasodilator infusing to the patient	
8.	Circuit ACT 210-230 (**any deviation** from this must be clearly documented)	
9.	Prepare a new Heparin infusion (at the same concentration as the circuit Heparin) and connect to the patient's IV line (ensure minimal dead space in line)	
10.	**Immediately prior** to bridging off: • Reduce circuit heparin infusion to half previous rate • Commence patient Heparin infusion at full rate (*Target ACT 210-230*)	
11.	Clamp patient off the ECMO circuit **A – B – V** *Ensure blood flow when clamped off (ie through bridge) is a minimum of half the rated flow for the oxygenator in use.* • *0800 Medos = 0.4L/min* • *2400 Medos = 1.2L/min* • *7000 Medos = 3.5L/min*	
12.	Remove green gas line from oxygenator during trial off (to prevent supersaturation)	
13.	Check immediate pump and patient ACT and repeat both every 15mins (Prior to flashing cannula) during trial off. Patient ACT parameter should be 210-230 *You must add volume (2ml) to circuit prior to each circuit ACT to prevent cavitation*	
14.	Flash cannulae to prevent clots by releasing (V-B-A) and clamping (A-B-V) every 15mins, observe pre-membrane pressures post-flash. *NB. You may need to increase rpm to allow forward flow of blood when "flashing".*	
15.	Check ABG/VBG ~20mins post-clamping off & • optimise ventilation / inotropes support as required & plan decannulation or re-institute ECMO support	
16.	If ECMO re-instituted **ensure green gas line is reattached**, circuit Heparin rate increased and patient Heparin infusion discontinued. Review flows and ACT parameters. May need to sigh membrane.	

ECMO VV Trial Off Checklist

PATIENT NAME _____ **DATE** _____

1.	Gas flow wean discussed/documented with ECLS physician & team • Ventilation optimised (? Bronchoscopy / NO / HFOV) • Cardiac support optimised (inodilators / Hb etc) • Adequate sedation & paralysis *NB. VV wean involves gas flow and blender wean not blood flow wean. No need for bridge off or heparin to patient*	
2.	Recent CxR performed & reviewed	
3.	Surgeon aware of planned cap off and potential for decannulation	
4.	Are present ECMO settings suitable for capping off • Blender FiO2 <30% • Sweep gas at low flow for oxygenator size (see charts) • Satisfactory ABG/VBG • Maintain previous ACT parameter and check ACT 1hrly as per normal protocol	
5.	To cap off remove green gas line from in line filter and loop round onto bottom of oxygenator	
6.	Check ABG/VBG ~20mins post-capping off then every 1-2hrs • optimise ventilation / inotrope support as required & plan decannulation or re-institute ECMO support	
7.	If ECMO re-instituted **ensure green gas line is reattached**. May need to sigh membrane.	

ECLS briefing support tool

The aim of this tool is to encourage a joint team approach to surgical procedures on patients supported with ECLS. It can be used for any procedure on ECLS.

Short patient summary & plan

Confirmation of roles

- NICU/PICU staff
  - If cannulating who will hold ECMO circuit to surgeon
- Theatre staff
- Perfusion
- Echo technician

Theatre staff

- Patient position/height
- Suction
- Diathermy pad
- Cannulae:
  - Correct sizes
  - 2 of each (If VVDL is intention, have VA cannulae available)
  - LA vent: yes/no
  - Tubing size & Cannula size – need connectors?
- Hep Saline for flushing/connection – who will perform this?

PICU/NICU

- Drugs
  - Sedation/Paralysis
  - Timing of Heparin (2nd heparin if weaning off)
  - Antibiotics
  - Resus drugs
- Blood products
  - Red cells (Cool box)
  - Platelets?
- Pacemaker
- ACT machine
- Ventilation settings

Perfusion

- Target flows
- Pressures zeroed (inc. inlet pressure)
- Initial
  - FiO2 (if E-CPR situation – 40% FiO2)
  - Sweep gas (low sweep gas if high arterial pCO2 value)
  - Heater temperature

ECLS de-briefing support tool

The aims of these points are to encourage a joint team review following surgical procedures on patients supported with ECLS. It can be used following any procedure.

Post-cannulation confirmation of:

- Target flows
- Inotrope strategy
- Cannula position
- Ventilator settings
- ECMO “orders”

Post-decannulation confirmation of:

- Inotrope strategy
- Ventilator settings
- Fluid targets

Brief summary & learning points

- Theatre staff
- NICU/PICU nursing
- Perfusion
- Cardiology
- Surgeon
- Intensivist

Title: **Obtaining an ACT from the circuit**

Rationale: To monitor the level of anticoagulation by measuring activated clotting time (ACT)

Personnel: ECMO Specialist

Equipment:
1 x 1ml leur syringe
1 x empty 2ml leur lock syringe
1 x 2ml leur lock syringe containing 0.9% or 0.45% sodium chloride
1 x bionector
1 x ACT-LR cuvette
- check expiry date (3 months if kept out of fridge)
1 x Haemochron Elite analyser, transformer and mains cable
1 x chloraprep wipes
1 x pair of gloves
1 x apron
1 x sharps box

ECMO Specialist Action:

Action:	**Rationale:**
Gather supplies, put on apron and wash hands	Infection control precautions
Ensure Cuvette is at room temperature this may take upto 60mins if previously in fridge. If taken from fridge ensure box is dated with new 3 month expiratory date	This can effect results Cuvettes have a 12 month expiratory date if kept in fridge this falls to 3 months at room temperature. Ensure 1 box at a time is used to prevent wastage
Scan cuvette then insert Cuvette into right side of analyser, once cuvette is at 37oc 'Add Sample' and 'Press Start' will be displayed on screen	Unit will remain in this ready mode for five minutes; if test is not started within this period cuvette should be discarded.
Scan / Enter patient ID Scan / Enter operator ID This should take approx 30 secs Use alcohol gel and put sterile gloves on	With each test to comply with near patient testing regulations (This can be done during test if required) More in depth instructions are available in ECLS manual

Connect bionector or clean existing connector at the post pump #3 pigtail for 30 seconds with a Chloraprep wipe, allowing to dry for 30 seconds.	To ensure sterility and minimise infection risk
Attach the empty 2 ml syringe, draw back and discard 0.9ml blood into sharps box	To account for the 'dead space' in the pigtail
Using a half twist, attach the 1ml syringe to the bionector and draw back 0.1ml blood	Only 150l (a drop) is required
Dispense one drop of fresh whole blood into the sample well of the test cuvette, filling from the bottom of the well up. A sufficient quantity of blood must be added directly to the center of the sample well to fill it flush to the top and press start immediately.	Any surplus will be taken by analyser into discard well To start the timing immediately as the blood starts to clot
Attach the 2 ml leur lock syringe containing sodium chloride, debubble and flush the pigtail	To ensure the pigtail remains patent To ensure no air enters the circuit
Test is complete when the Elite analyser 'beeps'. The result is displayed in seconds and should be recorded on the ECMO chart/CIS. The result remains on the display until the cuvette is removed or the unit goes into standby mode If ACT's are very erratic consider: - a different batch of cuvettes - QC ACT Elite machine - use a different ACT machine - changing Heparin infusion - Check coagulation screen	Allows regular monitoring and initial treatment of the patient's coagulation status (see heparin management protocol) ACT and heparin management results can be affected by: - urine output - platelets count - Fibrinogen levels - metabolic rate - blood product infusion If the ACT is >30 seconds (higher/lower) than the previous the specialist should check: - when platelets were last given - when the heparin infusion was changed - the volume of the patient's last urine output - Repeat test if required

If ACT result is not in keeping with clinical condition or circuit condition check: - APTT level - Platelet level - Heparin assay level - ATIII level - Rotem ACT parameter may need to be adjusted based on other anticoagulation markers. Common faults: • Sample not seen – Repeat test. • Sample too large – Repeat test. • Sample too small – Repeat test. • Bubbles in sample – Repeat test. • Pump fault – Repeat test.	Test results should correlate with patient and circuit condition, and/or treatment, abnormal results must be repeated. Recommended ACT Parameters are a starting point only and may need increased. If repeats contact Gillian Wylie / Bioengineering at ext. 89329 or 80132.
Safely dispose of all equipment in the sharps box	All used cuvettes should be considered as potentially infectious, handled with care and disposed of by following standard waste disposal policy. To ensure health and safety
The bionectors should be changed every Wed and Sunday	As per guidelines

Title: **Heparin Management Centrifugal Pump**

Rationale: To ensure safe management of initial heparin bolus and a continuous heparin infusion into the Centrifugal ECMO circuit

Personnel: ECMO Specialist x 2
ECMO consultant

Equipment: Heparin calculation form
- completed and checked by PICU/NICU consultant and ECMO specialists x 2
Required dose of heparin sodium solution for infusion (1,000 U/ml)
- ensure the same batch number is used
1 x dressing pack/drape
2 x syringes for heparin bolus
1 x syringe for infusion dose
1 x 500ml bag of 5% or 10% glucose
1 x 50ml leur lock syringe
1 x alaris syringe pump extension set
2 x filter needles
1 x green needle
1 x 2 ml leur lock syringe containing 0.9% or 0.45% sodium chloride
Drug labels
Alaris syringe pump and power cable
1 x chloraprep wipe
1 x bionector
1 x sterile gloves
1 x apron
1 x calculator
LR-ACT Cuvette
Haemochron Elite and charger

ECMO Specialist Action:

Action:	**Rationale:**
Confirm heparin calculation form completed, checked and signed by ECMO specialists X 2 and ECLS consultant	To ensure correct dose of heparin is prepared
Prepare and clearly label, boluses x 2 for cannulation and heparin infusion as per calculation form:	Bolus - **50units/kg** x 2 Infusion - **kg x 25 Units/ml** (under 40kgs) **kg x 12.5 Units/ml** (over 40kgs) **Patients of ≥80kgs (Neat Heparin)**

Initial heparin bolus **50 Units/kg** given to the **patient** by ECLS consultant after initial incision and blood vessels isolated by surgical team. Use central line access if available. Document time given	To rapidly achieve systemically anticoagulated stated (ACT's 350 seconds) To ensure iv line is patent.
If not on ECMO 20 minutes after the initial bolus check ACT. If ACT is <300 seconds a further **25-50 Units/kg** bolus can be given (dose at ECLS consultants discretion)	To prevent clots forming in the blood vessels during cannulation
Identify pigtail designated for heparin infusion	To prevent contamination with ACT result
Attach 2ml syringe containing sodium chloride and a bionector to pre membrane pigtail, draw back, de-bubble and flush the pigtail	To ensure patency and no air bubbles prior to connecting the heparin infusion
Simultaneously the 2nd ECMO specialist can put the heparin infusion in the alaris syringe driver and programme the pump, ensuring the syringe and extension set is clearly labelled. This can be completed prior to cannulation Purge the infusion for 2 ml ensuring no air is evident in the line and, using a bubbleless connection attach to the bionector. Otherwise see speak with ECLS consultant	To prevent clot formation in the ECMO circuit. Ensure programme checked by both specialists To prevent any air bubbles entering the circuit and in turn the patient
If the patient is not post op and drain losses < **5ml/kg/hr** commence at **50Units/kg/hr** = **2ml/hr**	This may seem a high rate for such a high ACT but the blood in the circuit has very little heparin will fall rapidly when the heparinised patient and non heparinised circuit blood mix
Open the tap and commence infusion	
Carry out an ACT immediately then every 15 minutes for at least the first hour, or until the ACT's stabilise within parameters, followed by at least hourly thereafter	To ensure adequate coagulation status and decrease clot formation
Be aware of written ACT parameters and ensure they are updated by ECLS consultant at least once per day The prescribed parameters are an initial guide,	Changes may be made depending on patient's status

if the clinical condition is out of keeping with ACT levels ie clots / bleeding the parameters will need to be reassessed more frequently.	
If ACT's fall out with parameters changes to heparin infusion may be required possibly along with a bolus of heparin **Low** ACT's should result in the infusion being increased by **2.5-5 Units/kg/hr**. If ACT is below 180, the minimum parameter or dropping rapidly a heparin bolus **10-20 Units/kg** given to the patient should be considered. **High** ACT's should result in the infusion being decreased **2.5-5 Units/kg/hr**. If the ACT's remain high despite decreasing the infusion over a period of time discuss with ECLS consultant. The infusion must not be reduced below 10u/kg without discussing with consultant and obtaining extended coagulation screen and TEG If for some reason, other than immediately post cannulation, the ACT is greater than 350 seconds the heparin infusion should be continued at 2.5 Units/kg/hr until a normal value achieved – **ECLS CONSULTANT SHOULD BE AWARE/PRESENT** **Regular ACT's at least every 30 minutes are required at this time until ACT's stabilised**	To ensure adequate coagulation status ACT and heparin management results can be affected by: - urine output - platelet count - metabolic rate - blood product infusion Individual judgment is necessary and care should be taken to avoid making abrupt changes in heparin producing a 'roller coaster' effect. It is rare that heparin requirement changes by more than 30 Units/kg in 1 hour Low levels of heparin will increase the risk of micro clot formation in the circuit and DIC If the heparin infusion is stopped altogether minor clotting may occur
Check ACT's more frequently during and immediately after blood product administration – **at least every 30 minutes** – including during Novo7 and Antithrombin administration and at the beginning of Aprotinin infusions.	Blood product administration can have a transient effect on ACT's
Continue to regularly monitor and treat coagulation screen	To ensure coagulation parameters are optimised

If ACT result is not in keeping with clinical condition or circuit condition check: - APTT level - Platelet level - Heparin assay level - ATIII level - Rotem ACT parameter may need to be adjusted based on other anticoagulation markers.	Fibrin degradation products can affect the ACT's and heparin management due to poorly solidified clots that the Elite machine has difficulty determining the end point of clotting
If ACT's become extremely erratic check: - Haemochron Elite machine - LR-ACT Cuvettes expiry date - urine output - recent platelet count - batch number and expiry on heparin vials - heparin infusion calculations - time of last blood product administration	To ensure adequate coagulation status
Change the infusion every 24 hours	

IF THE PATIENT IS BLEEDING SEE PROTOCOL FOR THE BLEEDING PATIENT (P.143)

STANDARD ACT RANGES FOR CENTRIFUGAL ECMO CIRCUIT

- **High** risk or **active** bleeding
  - ACT's 180-200 seconds
- **Low** risk of bleeding
  - ACT's 200-220 seconds
- When **pedivas** circuit and flows **<300 ml/min** or
- **centrimag** circuit and flows **< 750 ml/min**
  - ACT's 210-230 seconds
- **Prior** to trialling off
  - ACT's 220-240 seconds

Title: **Heparin Management Roller Pump**

Rationale: To ensure safe management of initial heparin bolus and a continuous heparin infusion into the ECMO circuit

Personnel: ECMO Specialist x 2
ECMO consultant

Equipment: Heparin calculation form
- completed and checked by NICU consultant and ECMO specialists x 2
Required dose of heparin sodium solution for infusion (1,000 U/ml)
- ensure the same batch number is used
1 x dressing pack/drape
2 x syringes for heparin bolus
1 x syringe for infusion dose
1 x 500ml bag of 5% glucose
1 x 50ml leur lock syringe
1 x alaris syringe pump extension set
2 x filter needles
1 x green needle
1 x 2 ml leur lock syringe containing 0.9% or 0.45% sodium chloride
Drug labels
Alaris syringe pump and power cable
1 x chloraprep wipe
1 x bionector
1 x sterile gloves
1 x apron
1 x calculator
LR-ACT Cuvette
Haemochron Elite and charger

ECMO Specialist Action:

Action:	**Rationale:**
Confirm heparin calculation form completed, checked and signed by ECMO specialists X 2 and ECLS consultant	To ensure correct dose of heparin is prepared
Prepare and clearly label, boluses x 2 for cannulation and heparin infusion as per calculation form:	Bolus - **50units/kg** x 2 Infusion - **kg x 25 Units/ml** (under 40kgs) **kg x 12.5 Units/ml** (over 40kgs) **Patients of ≥80kgs (Neat Heparin)**

Initial heparin bolus **50 Units/kg** given to the **patient** by ECLS consultant after initial incision and blood vessels isolated by surgical team. Use central line access if available. Document time given	To rapidly achieve systemically anticoagulated stated (ACT's >350 seconds) To ensure iv line is patent.
If not on ECMO 20 minutes after the initial bolus check ACT. If ACT is <350 seconds a further **25-50 Units/kg** bolus can be given (dose at ECLS consultants discretion)	To prevent clots forming in the blood vessels during cannulation
Identify pigtail designated for heparin infusion. The Pre Bladder / Bladder Pressure Monitoring pigtail	To prevent contamination with ACT result
Place the heparin infusion in the alaris syringe driver and programme the pump, ensuring the syringe and extension set is clearly labelled. This can be completed prior to cannulation Purge the infusion for 2 ml ensuring no air is evident in the line and, using a bubbleless connection attach to the bionector. Otherwise see speak with ECLS consultant	To prevent clot formation in the ECMO circuit. Ensure programme checked by both specialists To prevent any air bubbles entering the circuit and in turn the patient
If the patient is not post op and drain losses < **5ml/kg/hr** commence at **50Units/kg/hr** = **2ml/hr** The first couple of ACT's may be >400, reduce the infusion by 2.5 / 5u initially.	This may seem a high rate for such a high ACT but the blood in the circuit has very little heparin will fall rapidly when the heparinised patient and non heparinised circuit blood mix.
Open the tap and commence infusion	
Carry out an ACT immediately then every 15 minutes for at least the first hour, or until the ACT's stabilise within parameters, followed by at least hourly thereafter.	To ensure adequate coagulation status and decrease clot formation
Be aware of written ACT parameters and ensure they are updated by ECLS consultant at least once per day. The prescribed parameters are an initial guide, if the clinical condition is out of keeping with	Changes may be made depending on patient's status

ACT levels ie clots / bleeding the parameters will need to be reassessed more frequently.	
If ACT's fall out with parameters changes to heparin infusion may be required possibly along with a bolus of heparin **Low** ACT's should result in the infusion being increased by **2.5-5 Units/kg/hr**. If ACT is below 200, the minimum parameter or dropping rapidly a heparin bolus **10-20 Units/kg** given to the patient should be considered. **High** ACT's should result in the infusion being decreased **2.5-5 Units/kg/hr**. If the ACT's remain high despite decreasing the infusion over a period of time discuss with ECLS consultant. The infusion must not be reduced below 10u/kg without discussing with consultant and obtaining extended coagulation screen and TEG If for some reason, other than immediately post cannulation, the ACT is greater than 350 seconds the heparin infusion should be continued at 2.5 Units/kg/hr until a normal value achieved – **ECLS CONSULTANT SHOULD BE AWARE/PRESENT** **Regular ACT's at least every 30 minutes are required at this time until ACT's stabilised**	To ensure adequate coagulation status ACT and heparin management results can be affected by: - urine output - platelet count - metabolic rate - blood product infusion Individual judgment is necessary and care should be taken to avoid making abrupt changes in heparin producing a 'roller coaster' effect. It is rare that heparin requirement changes by more than 30 Units/kg in 1 hour Low levels of heparin will increase the risk of micro clot formation in the circuit and DIC If the heparin infusion is stopped altogether minor clotting may occur Other clotting tests Coag, Hep assay, TEG must be assessed
Check ACT's more frequently during and immediately after blood product administration – **at least every 30 minutes** – including during Novo7 and Antithrombin administration and at the beginning of Aprotinin infusions.	Blood product administration can have a transient effect on ACT's
Continue to regularly monitor and treat coagulation screen	To ensure coagulation parameters are optimised

If ACT result is not in keeping with clinical condition or circuit condition check: - APTT level - Platelet level - Heparin assay level - ATIII level - Rotem **ACT parameter may need to be adjusted based on other anticoagulation markers.**	Fibrin degradation products can affect the ACT's and heparin management due to poorly solidified clots that the Elite machine has difficulty determining the end point of clotting
If ACT's become extremely erratic check: - Haemochron Elite machine - Internal QC - LR-ACT Cuvettes expiry date - urine output - recent platelet count - batch number and expiry on heparin vials - heparin infusion calculations - time of last blood product administration	To ensure adequate coagulation status
Change the infusion every 24 hours	

IF THE PATIENT IS BLEEDING SEE PROTOCOL FOR THE BLEEDING PATIENT (P.143)

STANDARD ELITE ACT RANGES FOR ROLLER ECMO CIRCUIT

- **High** risk or **active** bleeding
  - ACT's 210-230 seconds
- **Low** risk of bleeding
  - ACT's 220-240 seconds
- When **weaning** flows **<300 ml/min** or if visible clot in circuit
  - ACT's 240-260 seconds
- **Prior** to trialling off in VA ECMO
  - ACT's 260-280 seconds

There is more variability in the new ACT's so prescribing 30 between the parameter rather than 20 maybe more appropriate

Title: **Aprotinin (Trasylol) Protocol**

Rationale: Safe administration of Aprotinin (Trasylol) in the bleeding ECMO patient

Personnel: ECMO Specialist x 2
PICU consultant
Pharmacist

Equipment: IVAC pump
Required dose of Aprotinin

ECMO Specialist Action:

Action:	**Rationale:**
Check ACT, FBC, coagulation screen (APTT, fibrinogen and D-dimer), TEG prior to commencing infusion	To ensure coagulation parameters have been optimised
Follow "Bleeding Bundle" (P.143)	Ensure all steps taken
Administer initial loading dose of 1 ml/kg over 20 minutes followed by continuous infusion of 1ml/kg/hr (10,000 iu/kg/hr) to the patient either via central or peripheral access	To decrease the risk of clot formation in the circuit Dose can be decreased if/when bleeding risk recedes
Check circuit regularly	To observe for clot formation
Monitor blood loss - drain losses - rate of decrease in Hb - rate of packed red cell requirement	To observe for a reduction in blood loss
Monitor renal function - urine output - urea and creatinine	To prevent drug accumulation Aprotinin is accumulated in the kidney for metabolism and excreted in urine
Recheck FBC and coagulation screen as clinically indicated • Check ACT every 30mins at start of Aprotinin infusion	To observe Hb and ensure coagulation parameters remain optimised

Title: **Novoseven (Novo7) administration**

Rationale: Safe administration of novoseven for control of excessive bleeding

Personnel: ECMO Specialist x 2
Consultant PICU
Senior medical support
Perfusionist aware/on site
On call consultant haematologist aware

Equipment: Emergency backup equipment (including spare circuit primed to saline if clots in circuit or perfusion/specialist/Intensivist concern over circuit viability)
Emergency drugs and volume (including priming blood available in blood bank)
Required dose of novoseven

ECMO Specialist Action:

Action:	**Rationale:**
Check ACT, FBC, coagulation screen (APTT, fibrinogen and D-dimer), RoTEM prior to administration	To ensure coagulation parameters have been optimised as much as possible Efficacy of Novoseven relies on the presence of at least moderate coagulation factor levels, especially fibrinogen.
Alert perfusionist	To allow time to prepare Plasmalyte-primed circuit **before** administration **if appropriate** Perfusionist **is required** to be on site if: - circuit running heparin free - being managed with ACTs consistently <200 - evidence of clots in circuit
Contact duty Consultant Haematologist	Order Novoseven from Blood Bank – Contact Duty Haematology technician Order 5ml/kg Cryoprecipitate from blood bank
Ensure blood products available - priming and top up units - platelets	In the event of full circuit change being required
Administer 5 ml/kg cryoprecipitate over 20 minutes prior to novoseven	To optimise fibrinogen level and optimise Novo7 effect
Prepare & dilute Novoseven aseptically – Prepare a 200microgram/ml solution as follows:	To prevent bacterial contamination To enable administration of correct dose

– Dilute powder using supplied diluent to give a 1mg/ml solution. – Take 0.5ml of this solution and further dilute to 2.5ml using **water for injection** to give a 200micrograms/ml solution	
Administer dose (100 mcg/kg) as a slow IV bolus minutes to the **patient** either via a central or peripheral line	To reduce the chance of the circuit clotting .
Clearly label vial and place in the fridge	Vial can be kept for up to 6 hours in the fridge and use for subsequent doses
Record administration in case notes and CIS – including dose and batch number	To allow an audit trail of unlicensed medication Audit questionnaire sent following its release – to be completed and returned to haematology
Check circuit every 10 minutes	To check for evidence of new clot formation
Monitor blood loss - drain losses - rate of drop in HB - rate of packed red cell requirement	To observe for bleeding tailing off Often seen within 20 minutes
Recheck ACT, FBC, coagulation screen (APTT, fibrinogen and D-dimer), TEG approximately 20 minutes after administration	To monitor effects on the coagulation screen
Recheck FBC and coagulation screen as clinically indicated	To continually monitor effect. They may not be long lasting due to Novoseven having a short half life and subsequent doses may be required

Title **Administration of blood products**

Rationale: The safe and appropriate administration of prescribed blood products and the adherence to YORKHILL blood transfusion policy

Personnel: ECMO Specialist
Nurse
ECMO physician

Equipment: Blood product
Administration set
Prescription
Sterile drapes
Gloves/Apron
Chloraprep wipes

ECMO Specialist Action:

Action:	**Rationale:**
Check prescription chart/ CIS documentation	For correct patient, HN/CHI number, D.O.B, correct date, time and dose, any allergies or special instructions, and if signed by medical staff
Check product correlates with patient documentation	Check: Surname, Forename, Gender, D.O.B, HN/CHI NO. Check ISBT 128 13 digit G101 unit number on the blood component matches the blood bank issue slip. Check that the blood group and the rhesus type on the blood component label matches, or is compatible with the patients group and type. Check expiry date.
Ensure correct signing and documentation On Pink slip, blue label and Prescription Chart/CIS	To ensure correct procedure for blood product traceability process
Ensure blood products utilised within correct time parameters	Concentrated Red Cells (CRC) transfused within 4 hours of arrival in department FFP and CRYO utilised within four

	hours of thawing, usually transfused over 1 hour Platelets transfused at once, never store in refrigerator. Return all unused units within time frame to blood bank
Prepare blood products for infusion	Filter all products utilising a micron pore 170-200 mesh filter. Utilise a blood giving set, or an appropriate platelet/cryoprecipitate giving set. To ensure removal of large macro organisms from products
Infuse all products directly into patient if possible	Avoid utilisation of circuit due to high negative pressures and minimal ports available. Only utilise circuit ports if absolutely necessary, and ensure bubble free connections If circuit used clotting products must be infused post Oxygenator
Dose required for adequate transfusion	Each case will be treated individually. Dose guidance: CRC 10-20mls/kg Platelets/FFP 10-20mls/kg Cryo 5-10mls/kg
CMV and irradiated products	As a guide patients < 1year all will receive CMV negative products. Immunocompromised patients, cardiac patients with Di-George (or Di-george status not confirmed) and post bone marrow transplant patients, will receive irradiated and CMV negative products. If in doubt discuss with Consultant/ and/ or haematology consultant
Observe patient for side effects and reactions and stop the infusion/ inform medical staff and blood bank, return products and documentation to blood bank	Undertake observations every 15 mins, for first hour, then hourly. Stop infusion if reaction occurs and manage symptoms. Document transfusion reaction form, available from blood bank

Title **Blood product requirements**

Rationale: To ensure availability of correct blood products during cannulation, and daily blood availability.

Personnel: ECMO Specialist / PICU / NICU support nurse.

Equipment: Blood request forms CIS/HISS

ECMO Specialist Action:

Action:	**Rationale:**
Order blood products for emergency cannulation of patient for ECMO. Check pre ECMO platelet count of >100,000 in all centrifugal pump patients. **Ensure Packed red cells available** < 5kgs: 1 priming unit with additions given to perfusion 1 large unit in checked cool bag 1 Paedipak drawn up at bedside 5 – 10 kgs 1 priming unit with additions given to perfusion 1 large unit in checked cool bag 1 Paedipak drawn up at bedside >10 kgs 1 large unit in checked cool bag **Platelets** < 5kgs 4 Paedipak platelets for top-up 5 – 10 kgs 3 units apheresed platelets >10 kgs 3 units apheresed platelets Other products FFP and Cryo may be required, this will be dependent on patient blood screen, FBC and Coagulation screen	The centrifugal ECMO circuit can be utilised with a clear prime in an emergency If platelet count less than this or roller pump patient will require platelets pre cannulation or at cannulation During cannulation if the patient is less than 10kg the circuit will be blood primed. 1 Paedipak of blood should be available at the bedside. The circuit will be clear primed if patient >10kg and no blood needs to be drawn up. 1 large unit of blood will be available in a "blood bank box" which can be checked and then returned to blood bank if unused within 3 hrs for re-issuing Other products will be ordered as required.

Daily product availability checks. The ECMO specialist will liaise with Blood Bank and haematology on a daily basis to ensure products available. (see next page for ready reckoner) All products available must be documented clearly on the daily checklist chart All blood products issued by BTS are Leukocyte depleted. CMV and irradiated products should be ordered as required.	It is the responsibility of the ECMO Specialist on shift, to ensure blood products are available at all times, in the event of emergencies. Communication should occur with blood bank during the shift to ensure availability and ordering if required. As a guide patients <1 year should have CMV neg products requested. All immuno-compromised patients, cardiac patients with Di-George or post bone marrow transplant, will receive irradiated and CMV negative products. If in doubt discuss with Consultant/ and/ or haematology consultant

CENTRIMAG SAFETY CHECKS

Primary Console

1. Ensure pump plugged into mains power.
2. On AC power indicator illuminated.
3. Battery level meter indicating battery fully charged.
4. Flow limit sensitivity set to normal.
5. Upper and lower flow limit alarms set: Pt <10kg 50ml/min below current flow. Pt> 10kg 100ml/min below current flow.
6. High and low alarm limits cannot be set within 0.3l/min of each other. High flow limits should therefore be set 0.3l/min above set low flow limits.

Backup Console

1. Backup console with motor present.
2. Console switched on and self test successfully completed at start of ECMO run and weekly thereafter and documented on ECMO flow chart.
3. Backup motor positioned so as to enable easy transfer of pump head in an emergency.
4. Backup console plugged into mains power at all times (Mains power indicator not illuminated when console switched off.)

Title: **CENTRIMAG SAFETY CHECKLIST**

Description: Checklist for Specialist

Personnel: ECMO Specialist

Equipment: Centrimag console charging.
Motor drive and flow probe
Pump of appropriate size and tubing for circuit with connectors.
Clamps x2
Saline and volume (PPS).
Heparin 1000u/ml
Emergency drugs
Perfusionist contact numbers.

ECMO Specialist Action

Action:	**Rationale:**
Ensure supplies/equipment are checked at the beginning of shift	To ensure equipment is ready in case of an emergency
Ensure above supplies/equipment are available and at hand at all times in case of circuit emergency	For immediate use in circuit emergency

Specialists Shift Plan Protocol

PATIENT CARE

The care of the patient on ECMO is much the same as the care of any critically ill patient who requires intensive medical and nursing management. The added care involved is the ECMO circuit.

Whilst on the ECMO the patient will be cared for by two nurses. At the beginning of the shift care can be divided such that one cares for the child and the other the ECMO circuit.

Care of the patient:

Hourly checks:

1. Vital signs recordings.
2. Ventilator recordings and check air entries.
3. Check ECMO circuit hrly.
4. Check ECMO cannulation site.
5. Measure and chart all drain losses, urine output and gastric losses.
6. Chart all input, infusions, feeds etc.
7. Check fluid balance.
8. Flash Bridge (more frequently if ACT's low)

4 hourly:

1. Patient blood gases arterial and venous, more frequently if weaning or unstable.
2. Endotracheal and oral suction.
3. Neurological checks, more frequently if concerns.
4. Pressure area care.
5. Eye and mouth care.
6. Passive limb exercises.

8 hourly:

Blood is sent for routine laboratory testing circa 8 hourly. This may be required more frequently e.g. during CVVH or excessive bleeding. Bloods are taken 0700, 1300 and 2100hrs.

PRN:
Assist Physiotherapist

Portable CXR and cranial ultrasound required as per protocol. These must be selected on Trakcare as not part of order sets.

Microbiology Testing.

Routine screening NICU

Twice weekly (Mon and Thur)
1. ET and OP secretions.
2. Urine and chest drain specimens.
3. Swabs from sites as required.

Blood cultures

Blood cultures post cannulation, circuit change and as clinically indicated.
BAL as indicated

CARE OF THE ECMO CIRCUIT;

Get a thorough report from the person working the shift before you:

Specialists Shift Plan Protocol

1. History of the patient.
2. Course on ECMO therapy.
3. Present condition
4. Discuss and observe any problems with ECMO circuit. Check for fibrin/clot formation and document size and location.

Check the ECMO circuit carefully at beginning of shift:

Assessment of each component should include the following:

VENOUS CANNULA

- What Cannula is been used
- Is the Cannula in a good position via x-ray
- Is the suture secure and dressing intact
- Is the wound bleeding
- Are there any clots in the tubing
- Is the Cannula secured to the bed in a proper holder
- Is the venous pressure satisfactory > -50
- Are the pre membrane pressures within normal limits

ARTERIAL CANNULA

- What Cannula is being used
- Is the Cannula in a good position via x-ray
- Is the suture secure and dressing intact
- Is the wound bleeding
- Are there any clots in the tubing
- Is the Cannula secured to the bed in a proper holder
- Are the post membrane pressures within normal limits

PUMP HEAD

- Is the pump head secure in the motor
- Is the motor function smooth
- Are there any clots in the pump head
- Are the tie straps secure
- Is the flow probe attached

CONNECTORS

- Are they secure
- Are the tie straps secure
- Are there any clots

OXYGENATOR

- Is the sweep gas rate within the range for the oxygenator
- Is the blood flow rate within the range for the oxygenator
- Is the exhaust port unobstructed
- Is the sweep gas connection secure
- Is the moisture draining from the exhaust port clear
- Is there air at the top of the oxygenator
- Are there clots at the top of the oxygenator
- Is it secure in it's holder

WATER HEATER

- Is the water bath level adequate
- Is the water circulating freely
- Is the water in the water bath clear

Specialists Shift Plan Protocol

- Are blood flow and water flow countercurrent
- Is the patients temperature stable
- What is the water heater temperature

ENVIRONMENT

- Are there enough tubing clamps
- Air box or spare raceway available
- Is there a sufficient supply of blood products in blood bank
- Is there a stocked supply trolley
- Is the back up console plugged in and fully charged

1. Verify checklist.
2. Acquaint yourself with IV fluids and medications.
3. Verify and compare accuracy of the Heparin infusion.
4. Check IV fluids against prescription chart.
5. Ensure emergency back-up equipment available.
6. Check battery supply.

Hourly:

1. Record pump RPM AND FLOWS/L, Inlet pressure and circuit pressures.
2. Check circuit for clot or air
3. Check Water heater temperature
4. Check upper and lower alarm limits.
5. Check ACT and maintain within set parameter by adjusting heparin
6. infusion rate and document on ECMO flow chart.
7. Trouble shoot for erratic or inconsistent ACT results.
8. Observe cardiovascular status in relation to bypass flows.
9. Check the cannulation site placement and stability.

PRN:

1. Make changes to the ECMO circuit RPM to maintain cardiovascular status in the set parameters and document on the ECMO flow chart.
2. Make changes to the membrane gases and blender to maintain respiratory status in the set parameters and document on the ECMO flow chart.
3. Trouble shoot the system at all times.
4. Replace blood products as required.
5. Assist with all nursing care.
6. Alert medical staff and perfusionist to problems with bypass.

Every shift:

1. Complete ECMO daily checklist.
2. Check pump ABG and Venous saturation and calibrate sat monitor
3. Aspirate and flush all pigtails
4. Re-zero Inlet pressure
5. Check all pigtails/3 way taps and Bionectors are secure
6. Check available blood components with blood bank.
7. Check emergency equipment.
8. **Discuss emergency procedures with support staff and outline procedures if patient had to come off ECMO SUPPORT.**

Title: CONSOLE AND/OR DRIVE MOTOR FAILURE

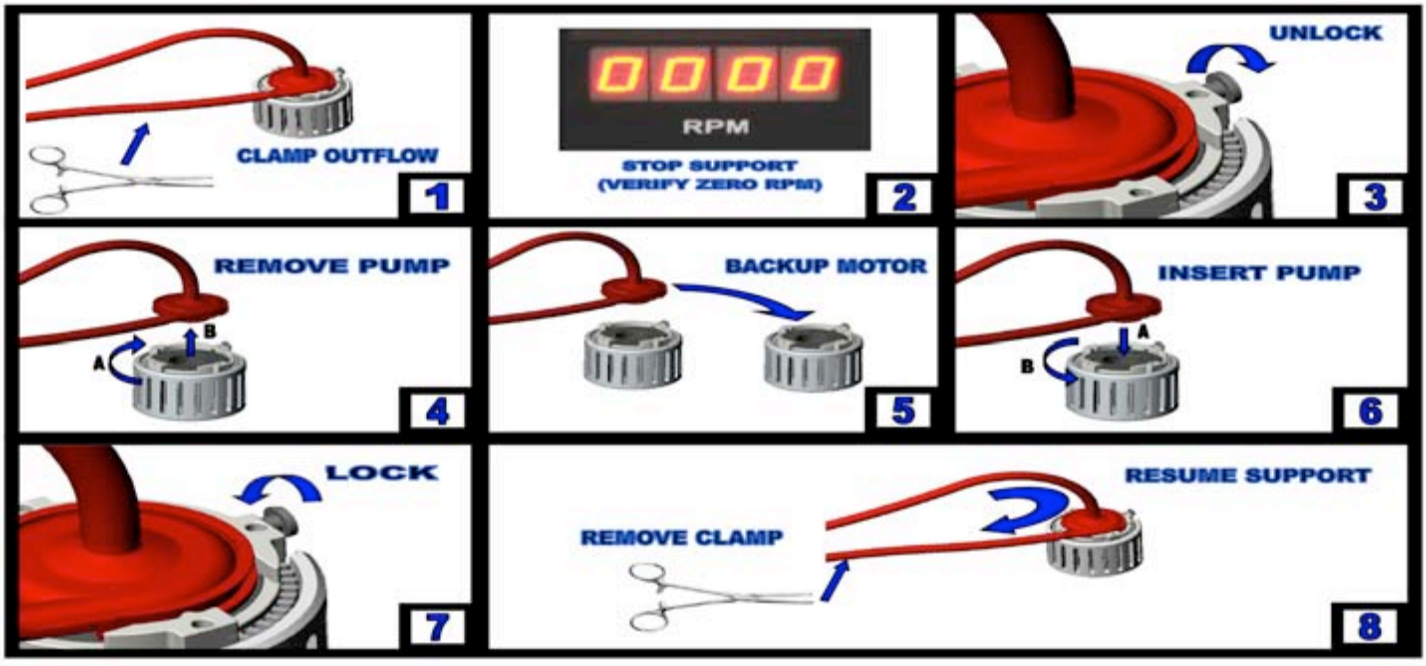
IF THE CONSOLE REPORTS A FAILURE, BOTH CONSOLE AND DRIVE MOTOR UNIT MUST BE CHANGED.

Description: Procedure to replace console and drive motor unit.

Personnel: ECMO Specialist x 2
Senior medical/Consultant PICU/NICU
Perfusionist on call

Equipment: Spare console and motor drive
Clamps x1
Emergency drugs

ECMO Specialist Action:

Action:	Rationale:
Clamp Arterial tubing post pump. Depress red key on Centrimag console and hold for 2 seconds to stop pump if still active	Rapid cessation of bypass in an emergency and avoid danger to patient.
Turn on the backup console.	Console takes 30 seconds to complete self check before becoming fully operational
	
Remove the pump head from the motor housing by loosening the locking screw. Rotate pump head clockwise and remove.	To change out motor.

Fit the pump head in to the backup drive motor unit, rotate counterclockwise and ensure the locking screw is tightened in the centre of the small semicircular cutout.	To secure pump in new head.
Set the RPM on the backup console to **1500** and remove the clamp then adjust RPM upwards to give flows at original pre emergency level	To re-establish ECLS support Flows will be initially displayed on original console.
Fit the flow probe to the back-up console and switch off nonfunctioning console.	To provide flow readings on new console
Remove pressure monitoring cables from the back of the malfunctioning console and click into ports labeled P1 and P2 on backup console	To allow pre and post membrane pressures to be monitored.
Access menu on backup console and ensure: 1. Flow limit sensitivity set to normal. 2. Pressure display - active 3. Upper and lower flow limit alarms set: Pt <10kg 50ml/min below current flow. Pt> 10kg 100ml/min below current flow. 4. High and low alarm limits cannot be set within 0.3l/min of each other. High flow limits should therefore be set 0.3l/min above set low flow limits 5. On AC power indicator illuminated. 6. Battery level meter indicating battery fully charged 7. Re-zero P1 and P2 pressures on console	Set up backup console to ensure appropriate alarm settings
Source replacement backup console and motor from perfusion.	
Have a cup of tea!	

March 2013

Title: **Management of Retrograde blood flow**

Rationale: To understand the risks, causes and management of retrograde blood flow.

Personnel: ECMO Specialist x 2.

Equipment: Circuit Clamp

ECMO Specialist Action:

Action:	**Rationale:**
Retrograde flow will be displayed on Levitronix **version one** console as - - - . If this is displayed, immediately clamp tubing post pump head.	Retrograde flow can occur at low RPM or during significant increases in MAP and will cause acute clinical deterioration. A disconnected or malfunctioning probe will display a single dot
On **version two** console Retrograde flow of up to 2.0 LPM is displayed as a negative number such as “-0.65 LPM”. Retrograde flow greater than 2.0 LPM, is displayed as downward arrows “vvvv LPM”	A disconnected or malfunctioning probe will display dashes “--.--“..
Increase RPM by approximately 10%. i.e 2000 to 2200.	To overcome patients Systemic Vascular Resistance
Slowly release clamp observing for signs of forward flow. If more RPM are required increase in 10% increments.	Forward flow of blood Flows displayed on console Improving patient condition
Once adequate support has been achieved wean RPM to give desired flow rate.	

Title: Management of air in venous cannula, tubing or *centrifugal head*

Rationale: To understand the risks, causes and management of air in the venous tubing and Centrifugal pump head.

Personnel: ECMO Specialist x 2.
PICU / NICU Consultant
On call Perfusionist

Equipment: Circuit Clamps x 1, 20ml syringe

ECMO Specialist Action:

Action:	**Rationale:**
<u>If Air Is Observed In Venous cannula, Tubing or The Centrifugal Head **ONLY**:</u> • Place clamp on **arterial** tubing only, leaving bridge clamp in place • Turn the pump off, remove head from pump casing • Initiate emergency ventilation and inotrope settings. • If air is noted in venous cannula, support cannula site and cannula securely and elevate tubing, tap tubing gently to attempt to move air forward • Walk all circuit air to pigtail #7 at the top of the oxygenator and aspirate **gently** using patient as volume reservoir • Replace head in pump casing, select previous RPM and **restart pump flow** by **removing** arterial clamp. • Check top of membrane and remove any trapped air. • Observe circuit for source of air. • Document total length of clamp off period in specialist evaluation • If air reaccumulate in cannula, venous tubing or head on reinstituting ECMO attempt to remove it again but if this is	To prevent micro air embolism To allow rotation of head to facilitate de-airing To provide adequate support whilst off ECLS support To prevent accidental decannulation whilst removing air from cannula To provide volume reservoir To prevent pulling air across the membrane To ensure all air has been removed from circuit

not possible • Take patient off ECMO immediately, clamp on **arterial** tubing, remove **bridge** clamp and clamp **venous** tubing. **A-B-V** • Contact the on call surgeon, perfusionist and theatre team • Attempt to circulate through the bridge • Consider a Heparin bolus to patient to prevent the cannulae clotting	To determine source of air problem To preserve ECMO circuit To increase patient ACT

Title: **Management of *Gross air* in circuit**

Rationale: To understand the risks, causes and management of gross air in the ECMO circuit.

Personnel: ECMO Specialist x 2.
PICU / NICU Consultant
On call Perfusion

Equipment: Circuit Clamps x 2, 1 litre 0.9% saline, iv giving set, 50ml syringe

ECMO Specialist Action:

Action:	**Rationale:**
If Gross Air Identified In The Circuit:	**Gross Air within the ECMO circuit is a clinical emergency.**
• Take patient off ECMO immediately, clamp on **arterial** tubing, remove **bridge** clamp and clamp **venous** tubing. **A-B-V**	To prevent an air embolism
• Depress red stop button to cease pump revolutions	
• Initiate emergency ventilation and inotrope settings.	To provide adequate support whilst off ECLS support
• Contact on call perfusionist	
• Prime giving set with saline and connect to pigtail #1 (pre-pump head) and open flow up fully.	To provide volume reservoir
• Place 50ml syringe on pigtail #7 at top of oxygenator	To allow air to rise towards membrane
• Remove head from pump casing and rotate round to allow air to rise toward pigtail #7	To allow rotation of head to facilitate deairing
• Aspirate air with 50ml syringe on pigtail #7 gently tap oxygenator ensuring head is kept below membrane. May need to remove oxygenator from pole to allow full air withdrawal.	To ensure all air has been removed from circuit Water heater may need to be disconnected – remember to switch it of prior to disconnecting
• Leaving saline open, replace head in pump casing and restart pump flow, circulating through bridge.	
• Check all pigtails, head and membrane. Close access to saline bag.	
• Place patient back on ECMO **V-B-A**	To assess these for air entrainment – if not seen here most likely to be coming from patient side of clamps ie cannulation site or connection with cannula to circuit
• Observe circuit for source of air.	
• Document total length of clamp off period in specialist evaluation	

Title: **Clamping on and off ECMO Centrifugal Pump**

Rationale: To safely place patients on and off ECMO

Personnel: ECMO Specialist x 2.

Equipment: Circuit Clamp x 2

ECMO Specialist Action:

Action:	**Rationale:**
Ensure both specialists are aware of procedure and someone is able to hand ventilate the patient throughout or mechanical ventilation is increased appropriately	To maintain patient oxygenation off ECMO
Ensure any emergency drugs (which may be required) are available and that IV lines are accessible	To maintain patient stability throughout the procedure
If a procedure is to be performed, gather all supplies in advance	To minimise time off ECMO
Clamping off VV/VA ECMO: Clamp arterial tubing **A** Remove bridge clamp **B** Clamp Venous tubing **V**	To prevent cavitations in centrifugal head When no pump flow is present the arterial tubing must be clamped immediately
Going on VV/VA ECMO: Set RPM to 1500 on pump Remove Venous Clamp **V** Clamp Bridge **B** Remove Arterial Clamp **A** Adjust RPM to give desired flow	To prevent cavitations in centrifugal head To over come patient afterload

Title **Changing an ECMO circuit Three-way Tap**

Rationale: To replace an ECMO circuit tap at prescribed intervals and in the event of cracking/clotting

Personnel: ECMO Specialist x 2

Equipment:

1x Sterile three-way tap	1x Clamp
Chloraprep wipes	5ml syringe flush
Gloves	Bionector x1

ECMO Specialist Action:

Action:	**Rationale:**
Gather supplies	To ensure all equipment at hand for procedure
Wash hands and put on gloves and apron	Observe universal precautions
Attach syringe to new Bionectors and 3 way tap and flush through all the ports and bionectors	To remove air from the tap
Clamp the appropriate pigtail for tap change	Ensure pigtail protected from damage.
Whilst holding the pigtail clean port with ChloraPrep wipe, allow to dry for 30 secs and remove the old tap	As per universal precautions
Attach new tap and bionector to the pigtail, making a bubble less connection	As per bionector protocol
Remove clamp from pigtail and ensure connection air free	Avoid air embolus to patient

Title **Changing a Pigtail**

Rationale: To replace an ECMO circuit pigtail

Personnel: ECMO Specialist x 2
PICU Consultant
ECMO Coordinator (if required)

Equipment: 3 x Clamps
1 x Pigtail
1 x Three- way Tap
Gloves
Bionector x1
5ml syringe flush
Chloraprep wipes

ECMO Specialist Action:

Action:	**Rationale:**
If emergency change, clamp post pump and press stop button on console for 2 secs	To ensure patient safely removed from bypass and supported
Inform nurse and Consultant.	Emergency care of patient provided as required by support
Gather supplies from emergency box	To have everything at hand for procedure.
Wash hands and put on gloves	To observe universal precautions
Attach three-way tap to the Bionecter and pigtail. Flush, leaving the syringe attached to the three-way tap	To prevent air embolus
Clamp tubing on either side of the circuit pigtail	To prevent blood loss on removal of old pigtail
Clean port with chloraprep wipe, leave for 30 secs, disconnect the old pigtail, connect the new pigtail with the three-way tap and syringe attached	To observe universal precautions To aspirate air during the change over of pigtails
Remove the clamp nearest to the patient's venous limb, draw back to de-bubble. Turn tap off to the circuit and remove syringe and second clamp. Set RPM to 1500, Check circuit for air, then remove post pump clamp and adjust RPM to give required flows	The patient is the reservoir for aspiration To ensure it is safe to return patient to ECMO

Title: **Management of external cardiac massage**

Rationale: To understand the risks and management of patients during external cardiac massage on ECMO.

Personnel: ECMO Specialist x 2.
PICU/NICU Consultant.
On Call perfusionist
On Call surgeon (cardiac if post-op cardiac patient)

ECMO Specialist Action:

Action:	**Rationale:**
Ensure appropriate help is sought ie On call perfusionist, consultant and surgeon	Cannulae may be dislodged during resuscitation. ECMO cannulae may clot during cardiac arrest
External cardiac massage carries a significant risk of dislodging the ECMO cannula. VA ECMO patients should have flows increased to provide cardiac support during arrhythmias (may require extra volume); however patients on VV ECMO are reliant on native cardiac function.	If open chest is cannulated Internal cardiac massage may be safer if appropriately trained member of staff is available. Remember VV ECMO provides only respiratory support
One specialist observes circuit for air entrainment.	To reduce increased risk of air entrainment caused by reduced filling.
If air is entrained clamp patient off circuit immediately on arterial tubing	To prevent air embolism.
Troubleshoot and treat cause of arrest	Abnormal Rhythm – defibrillation Electrolyte imbalance Tamponade
Observe circuit and cannulae for clots prior to resumption of full flows.	To prevent emboli

Title: **Defibrillation on ECMO**

Rationale: To understand the risks and management of a patient on ECMO during defibrillation.

Personnel: ECMO Specialist x 2
PICU Consultant.

Equipment: Defibrillator

ECMO Specialist Action:

Action:	**Rationale:**
Patients can be defibrillated with the device running, but it is mandatory to have a backup console available.	To ensure console can be changed if malfunction occurs
Prior to defibrillation, the tubing should be checked to ensure they are dry from blood and other fluids.	To prevent conduction of current along tubing
Ensure all staff stand clear of the patient, device and console during defibrillation. (European Resuscitation Guidelines)	To prevent injury
The console should be checked for continued normal operation immediately after each defibrillation attempt.	To detect any console malfunction
Note that an alteration in patients rhythm may require an alteration in the pump RPM and/or the patients filling pressures to maintain adequate pump flow.	Changes to rhythm can alter filling pressures, SVR, PVR and therefore affect pump flow rates.

Title: **Protective Hypothermia on ECMO**

Rationale: Managing patients who have been cooled on ECMO
Personnel: ECMO Specialist x 2.
PICU/NICU Consultant.
On Call perfusionist

ECMO Specialist Action:

Action:	**Rationale:**
Patients should be assessed on a case by case basis following multidisciplinary discussion	Cooling may be undertaken in those patients who have a clear history of profound hypoxia, cardiac arrest or who have inadequate oxygen delivery on available ECMO flows
Once a decision has been made to cool at cannulation please inform perfusion	To ensure that circuit is not pre-warmed and water heater is set to correct temperate at initiation of ECMO support
Patients should be managed at: NICU - 34oC for a period of 48hrs. PICU – 35/36oc for minimum of 48hrs Core temperature should be controlled by altering ECMO circuit water heater. Adjustments should be checked by both specialists When analysing blood gases the blood temp should be adjusted on the gas machine	To reduce basal metabolic demand and cerebral oxygen demand This gives excellent central control and response to temperature changes To ensure accuracy of result
A rectal or oesophageal temp probe must be used to monitor core temperature. Core and peripheral temp should be monitored continuously whilst cooled and entered on chart hourly.	To ensure accuracy of recording To detect any deviation from set levels.
Patients should not be enterally fed whilst cooling is underway or during the rewarming period.	Cooling and previous hypoxic hit may cause changes to the blood supply within the gastro-intestinal tract
At the end of the cooling period patients should be slowly rewarmed at a rate of no more than 0.5oc per hour.	To avoid sudden changes to SVR

Title: **Sighing a wet membrane**

Rationale: To safely determine and manage a wet membrane

Personnel: ECMO Specialist x 2.

Equipment: Sweep gases, ABG

ECMO Specialist Action:

Action:	**Rationale:**
Signs of wet membrane: • Deteriorating patient gases • Pump gas ↑ Co2 or ↓ Pao2 • Increased moisture at membrane exhaust	Membrane oxygenators, just like lungs can develop pulmonary oedema and carbon dioxide retention can occur when fluid (water or blood) accumulates in the gas phase of the oxygenator.
If wet membrane is suspected check: • Pt gas • Pump gas • Transmembrane pressure • Circuit and Oxygenator for clots	To access membrane function
Action: If ↑ Co2 or ↓ Pao2: • Check Gas line secure • Check gas flow meter • Increase blender to 100% • Sigh membrane for 2-3mins Medos 0800 – 2 litres, Medos 2400 – 5 litres, Medos 7000 – 10 litres • Recheck pump gas • Ensure pump gas is checked **prior** to returning blender to previous setting • Contact perfusion if problem not resolved • Observe membrane for blood in gas phase	To dry out membrane To ensure adequate incrementation of Pao2 is seen To discuss membrane or circuit change

Title: Pressure Area Management

Rationale: Pressure ulcers (also called pressure sores, bed sores and decubitus ulcers) are areas of tissue damage that occur in the acutely ill, in people who cannot reposition themselves, and the malnourished. Pressure ulcers negatively affect quality of life and impose a significant financial burden on healthcare systems

Personnel: ECMO Specialist.

Equipment: Pressure area assessment tool, Repose Mattress, Dermal Pads,

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ECMO Specialist Action:

Action:	**Rationale:**
Risk assessment must take place within 6 hours of admission, this can be an initial informal assessment (documented), followed by use of a formal assessment tool within 24hrs (PICU)	ECMO patients are at high risk of pressure ulcers due to pre cannulation history of immobility, inotrope use, oedema, poor nutrition, poor tissue perfusion and hypoxia
All ECMO patients should be nursed on a repose mattress of suitable size or specialist pressure bed. Assess skin within 6hrs of admission and during care. Document any pressure damage using the Scottish adapted EPUAP grading tool	There is clear evidence that individuals at risk benefit from products which are different from the standard NHS provision, eg pressure redistributing mattresses or fibre, foam, air, static or dynamic overlays
A dermal pad (Aderma) or Z flo positioner should be placed under the Occipital area, further pads can be used on heels, elbows and shoulders if required	Dermal pads or Z flo positioners are made from a unique polymer gel, similar to fatty tissue, which distributes pressure while protecting and padding bony prominences.
If the ECMO cannulae are marking the skin or a head bandana is to be applied to support the cannulae a piece of dermal pad should be placed between the cannulae and the skin. The Occipital dermal pad must be placed under the bandana and next to the skin	To minimise pressure under cannulae and prevent Ulceration of the occipital or cannula area. The bandana must be loosened daily to assess skin integrity and Micro movements of head should continue to relieve pressure
If superficial ulceration occurs hydrocolloid wound dressings should be applied, wound assessment tool used and referral to the tissue viability service considered	Evidence suggests hydrocolloid wound dressings are preferable to gauze dressings as they create a moist wound healing environment.

ECMO Patient Transfer Checklist

WEIGHT _____ kg PATIENT NAME _____

Personnel Required	ECMO Physician	☐
	ECMO Specialists x 2	☐
	Perfusionist	☐
	Cardiac Anaesthetist (for cath)	☐
	Nurse Runner	☐
	Senior Transport Fellow	☐
Equipment Required	ECMO trolley (with spare console)	☐
	ECMO Emergency Trolley	☐
	Portable Monitor	☐
	2 x Oxygen Cylinder > 1/2 Full for bagging / circuit sweep gas flow	☐
	Suitable Bed	☐
	Transfer bags / Resus drugs	☐
	Volume (50mls/kg drawn up)	☐
	Blood Products	☐
Prior to Move	ACT LR Cuvettes / Elite analyser	☐

1.	Remove all unnecessary equipment from patient ie.Haemofilter, Pumps.	
2.	Secure cannulae and arrange tubing to avoid kinks.	
3.	Change out CDI 101 to CDI 500 to access battery power	
4.	Switch on O2 cylinder, check level >1/2 full, hand ventilate.	
5.	Check CT/CATH lab ready for patient	
6.	**Immediately prior to move** turn cylinder O2 to same rate as sweep and connect cylinder to membrane instead of blender	
7.	Set up inlet pressure monitoring	
8.	Switch off water heater and unplug mains cable and gas blender	
9.	The machine will alarm as on battery power, press 'alarm acknowledge' button. This will reset the alarm	

On arrival in Cath/CT and on Return to Unit

1.	Plug in Mains cable.	
2.	Turn on water heater and set temperature	
3.	Plug gas lines into wall, set sweep gas rate and blender Fio2 then connect gas line from blender to membrane	
4.	Turn off portableO2 cylinder	
5.	Reconnect inlet monitoring to patient monitor	
6.	Un-tape and arrange tubing, secure cannulae	
8.	Conduct full patient and Circuit check after each move	

SCUF Priming and Connection Protocol

Goal

To safely prime a filter for Slow Continuous Ultrafiltration (SCUF), which is used for fluid overload with no or mild renal failure and minimal or no response to diuretics

Equipment

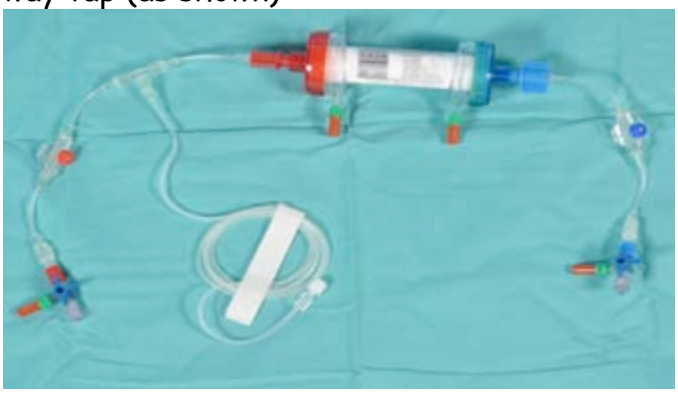
1 x sterile disposable drape
1x sterile green towel
1 x sterile gloves
Edwards Aquamax HF03 Filter
Gambro lines (AV-899-U)
Hospal re-infusion lines (1 SP-401)
2 x HOSPAL drainage bags (SP-C36)
2 x Braun Discofix (3SC) 3-way taps
6 x Vygon Bionecteur connectors Arterial (896.11)
1 x UHS Bile bag (UN400RK)
1 x Vygon green connector (800.01)
1 x Vygon 892.00 female-female connector
1 x sterile Scissors
2 x blue clamps
1 x 10ml syringes
2 x Litres of 0.9% NaCl
1000 IU Heparin (Pump Heparin)
2 x IVAC giving sets
1 x IVAC volumetric pump

Procedure

1. Wash hands and put on plastic gown

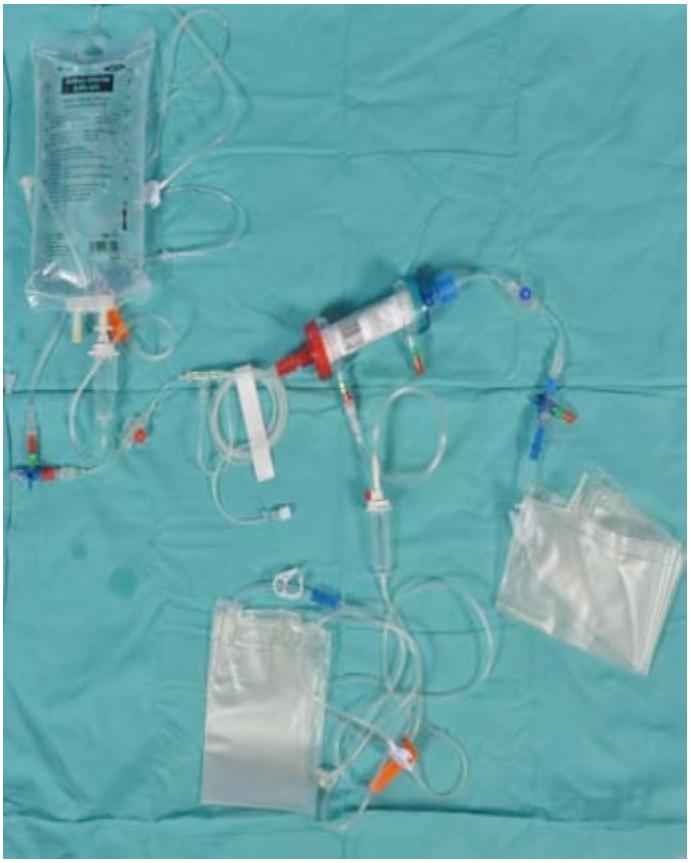
2. Open sterile drape and place green towel on top. Open all sterile items onto clean surface.

3. Attach AV-899 lines to filter. The red and blue ends attach to the same colour on the filter. Then attach a 3-way tap onto both ends. Place 1 Arterial Bionecteur onto each side port of the Edwards filter and each 3-way tap (as shown)



5. Take one of the 1 SP-401 lines and cut it around 12" from the luer lock connector. Connect the luer lock to the side port nearest the red end of the filter. Insert an IVAC giving set into the cut end. (Tubing may need stretched with end of scissors)
Attach the C-764 bags to the 3-way tap on the blue line and to the IVAC giving set. These will act as collecting bags during the priming process.

SCUF Priming and Connection Protocol



6. Inject 1000 IU of heparin into a litre bag of 0.9% NaCl and run this through the other IVAC giving set. Attach this onto the 3-way tap on the red line.

7. Place small clamps on the thin heparin line of the red line and the C-203 line. Begin to run through the heparinised saline ensuring the filter is held upright. Agitate the filter by knocking to ensure there are no air bubbles. The infused fluid will fill the C-764 bag attached to the blue line. This primes the inside of the filter so you will not see any fluid in the filter at this stage. Keep fluid high and the filter low to speed up the process. DO NOT RUN IT THROUGH AN IVAC PUMP.

SCUF Priming and Connection Protocol

8. Unclamp the thin heparin line and clamp distally to get rid of air.

10. With a blue clamp, clamp of the blue line and open up the 1 SP-401 line. Keep the hepsal flowing, and the filter upright and the outside portion of the filter will begin to fill up.

11. As the fluid accumulates there will always be small pockets of air. Get rid of these by clamping off the 1 SP-401 line and placing a 10ml syringe onto the Bionecteur on the spare side port. Aspirate and if the pocket of air is uppermost it will flush out.

12. Run the hepsal as in step 7 and 10 alternately until the litre is finished. Then attach a litre of unheparinised saline and repeat the process. The complete prime should take between 30 and 45 minutes.

Connecting the SCUF Filter

Equipment
Primed Filter
2 x 10ml syringes filled with saline

SCUF Priming and Connection Protocol

Haemofilter clamp
IVAC giving set
3 x blue clamps

1. Fit the blue haemofilter clamp onto the ECMO trolley upright and attach the primed haemofilter. Insert with the red side uppermost. Ensure it is secure. Ensure there are blue clamps on the red and blue lines and on the 1 SP-401 filtration line.

2. Attach a 10ml saline flush to the Bionecteur on the 3-way tap attached to the red line and ensure it is primed free of air.

3. Attach the three-way tap onto the Bionnector on the bottom post pump pigtail. Ensure there are no bubbles by aspirating and then flushing the pigtail via the three-way tap. Do not open the pigtail yet but take the clamp off the red line

4. Attach a 10ml saline flush to the Bionecteur on the 3-way tap attached to the blue line and ensure it is primed free of air.

SCUF Priming and Connection Protocol

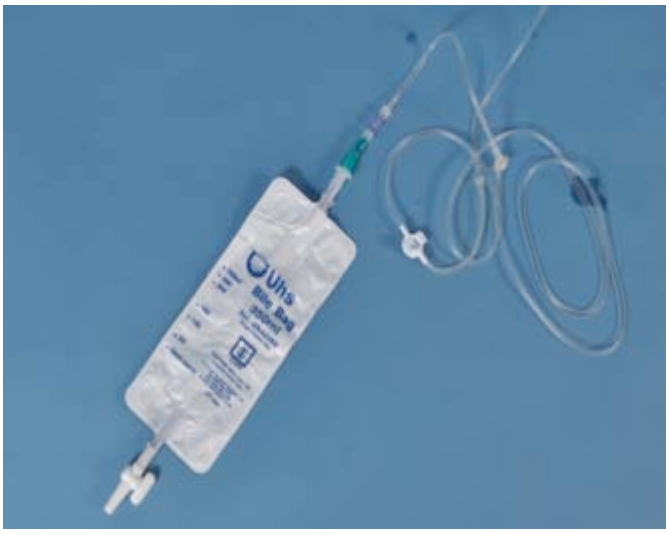
5. Attach the 3-way tap on the blue line to the Bionecteur on the venous pre-pump head pigtail (Proceed cautiously as this is potentially the negative, sucking side, of the circuit). Ensure there are no bubbles by aspirating and then flushing the pigtail via the three-way tap. Again, do not open the pigtail but take the blue clamp off the line.

6. Insert the IVAC giving set into the IVAC pump as normal. Attach the flow sensor to the chamber. Remove the blue clamp from the 1 SP-401 filtration line. Place one of the blue clamps on the small heparin line, which comes off the red line.

7. You are now ready to open up the filter. It is important to open the venous (low pressure) end immediately before the arterial end. The filter will now fill with blood from the top down. Ensure that you observe for air in the head and instigate the emergency procedure for removal if present.

8. The filter will shunt a portion of blood away from the pump so the venous saturations will drop. It is important to increase the RPM on the console to increase the flows and achieve the same venous saturations as before (~ 40-60 ml/min)

9. Set the IVAC to remove the desired volume of fluid hourly. It is essential to measure the amount of fluid filtered on an hourly basis, as the pump seems to be rather inaccurate. Attach the end of the Ivac giving set to a Vygon 892.00 female-female connector and then connect to a green connector. This can be attached to a bile bag to create a closed system that can be drained and measured hourly.



10. Place the green towel on the floor under the Ivac and giving set to ensure that any fluid loss is easily seen. Ensure red cap on Ivac burette is closed at all times.

14. If level of ultrafiltrate diminishes and the "air in line" detector on the volumetric pump begins to alarm this usually means the filter is clotting off. As it takes an hour to prime another filter circuit this should be done as soon as problems begin to arise to limit the time the patient is off SCUF. Otherwise filters should be electively changed at 72hours or with circuit changes.

Neck Cannulae Care Protocol

Goal

To clean ECMO cannulae insertion site and cannulae, to check security of sutures and to note position of cannulae markings at skin site on a PRN basis or weekly if dressing is intact.

If there has been bleeding previously from the cannula site, check with medical staff prior to proceeding.

Equipment

Dressing Pack
Non sterile plastic apron
Sterile Gloves
1 sachet normasol
2 sachets Apeel
Sterile scissors
Kaltostat Dressing
IV 3000 Dressing of suitable size

Pt < 10kgs
2 ChloraPrep Sepp 0.67ml Applicators

If Pt > 10kgs
1 ChloraPrep Sepp 0.67ml Applicator
1 ChloraPrep Frepp 1.5ml Applicator

Procedure

1. Wash hands and put on plastic gown

2. Open dressing pack and sterile items, including normasol, onto clean dressing trolley.

3. Place sterile dressing drape under patient's cannula site.

4. Remove old dressing **using Apeel** to lift dressing, take care not to pull or twist cannulae. Once dressing is removed ensure cannula is supported and secure (This may require second specialist to assist).

5. Clean hands with alcohol rub and then put on sterile gloves.

6. Using normasol, clean off any areas of dried blood. Proceed cautiously at insertion site. Work from insertion site outwards to avoid contamination of site.

7. If area around insertion site appears inflamed take a bacterial swab (prior to using ChloraPrep applicator).

8. Check patency of sutures securing cannula and note where the cannulae markings are in relation to the skin level at insertion site.

9. Activate ChloraPrep applicator by squeezing wings or tube and use to clean around insertion site and along first 10cms of cannulae. Rub skin and cannulae gently up and down and back and forth for 30 secs.

10. Allow to dry for 30 secs.

11. If site is dry place a suitably sized IV 3000 dressing over insertion site

12. If cannula site is oozing apply firm pressure with swab for 5 mins, remove swab and then place small piece of Kaltostat over insertion site. If still oozing apply small piece of surgicel around insertion site and apply pressure for a further 5 mins then apply Kaltostat and IV 3000 dressing.

13. Record condition of cannulae site, sutures and position of cannulae in nursing Kardex or on CIS. Report any concerns to medical staff.

Title **X-ray imaging on ECMO patients**

Rationale: To ensure the appropriate and safe requesting of portable chest x-rays both routine and emergent

Personnel: ECMO Specialist
Radiographer

ECMO Specialist Action:

Action:	**Rationale:**
All ECMO patients should have a chest X-ray (CxR) post cannulation.	To check cannula position
The ECMO specialist must ensure that the baby, visitors and all colleagues are adequately prepared and protected during the x-ray	There is a risk from exposure to radiation during x-ray. Screens, lead pads and aprons should be used where appropriate
Routine CxR should be ordered as part of the "/ECLS1" order set but may need to be manually selected in Trakcare. CxR should be performed daily to exclude ET tube misplacement, pneumo / haemothorax, collapse / consolidation, deteriorating or improving lung fields and check cannula placement. If patient requires a ET Tube, circuit change, cannula reposition or bronchoscopy CxR may be performed after the procedure rather than early in the morning During a prolonged ECMO run the ECMO consultant may omit an x-ray and this should be documented	To observe for clinical changes and aid management / weaning decisions To assess success of scheduled procedure To reduce x-ray exposure
The ECMO specialist must ensure that the CxR is examined immediately once on the PACS system to exclude acute changes. Any concerns must be checked by medical staff	To ensure that problems are identified immediately. The previous x-ray can be used for comparison.

If there are clinical concerns which are suggestive of acute clinical changes eg: • Changes to arterial or venous saturations, ABG, air entries, chest movement • Suspicion of tamponade due to pneumothorax • Pulmonary Haemorrhage • Signs of haemorrhage, causing increased neg inlet pressure or acute problems with ECMO flows An emergency CxR should be requested on Trakcare with a follow up call to X-ray. This should be discussed with the ECMO physician	Changes to clinical status should be investigated to exclude life threatening complications
ECMO specialists are required to be familiar with the IMER guidelines and a 2 yrly update is required. This can be given during an update day. Once patients are decannulated any x-ray requests should be completed by the medical staff	To ensure compliance with the regulations. Specialists are only authorized to order x-rays for patients on ECLS support

Title **ECMO Ultrasound Imaging**

Rationale: To ensure the appropriate requesting of skull ultrasound both routine and emergent

Personnel: ECMO Specialist
Radiographer

ECMO Specialist Action:

Action:	**Rationale:**
All Neonatal ECMO referrals and those Paediatric patients with a patent anterior fontanelle < 6 months should have a pre ECMO skull ultrasound prior to cannulation	To check for Intraventricular haemorrhage (IVH) or gross abnormalities
Routine ultrasound is ordered as part of the "/ECLS1" order set but may need to be manually selected in Trakcare. Skull ultrasounds should be performed on Day 1, Day 2, Day 3 of ECMO run and Mon, Wed, Fri thereafter. If Day 3 falls on a Sun, Tue or Thur the Ultrasound should be performed the following day and not omitted.	Infants on ECMO are systemically heparinised and at increased risk of intracranial haemorrhage. Any abnormality of the u/s must be documented in the patients case notes and the consultant informed
If there are clinical concerns which are suggestive of IVH eg: • Bulging Fontanelle • New Clinical or CFAM seizures • Pupil changes • Signs of haemorrhage, significant unexplained drops in Haemoglobin in conjunction with any of the above An emergency skull ultrasound should be requested on Trakcare with a follow up call to Ultrasound (ext 80095). This should be discussed with the ECMO physician.	Changes to neurological status should be investigated to exclude IVH

If a small IVH is confirmed ACT parameters should be tightened, Platelet parameters increased to 120,000 - 150,000, tight management of pCO_2 and Aprotinin considered. Serial cranial USS should be performed and early consideration of decannulation.	A large bleed associated with profound clinical deterioration including flaccidity and fixed pupils will require an urgent neurological review +/- CT brain with contrast and potential cessation of ECMO support on the grounds of futility.

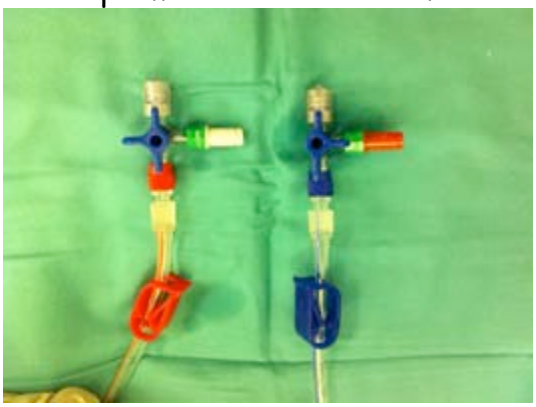
Title: **Attaching and running the Aquarius for CVVH**

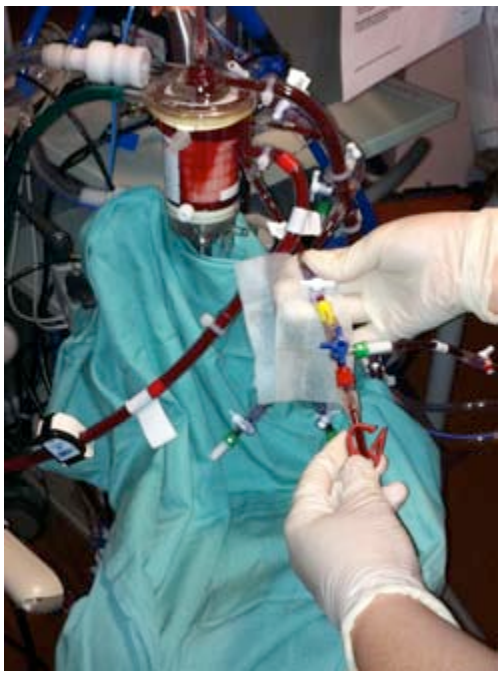
Rationale: To understand the procedure and risks for attaching and running the circuit

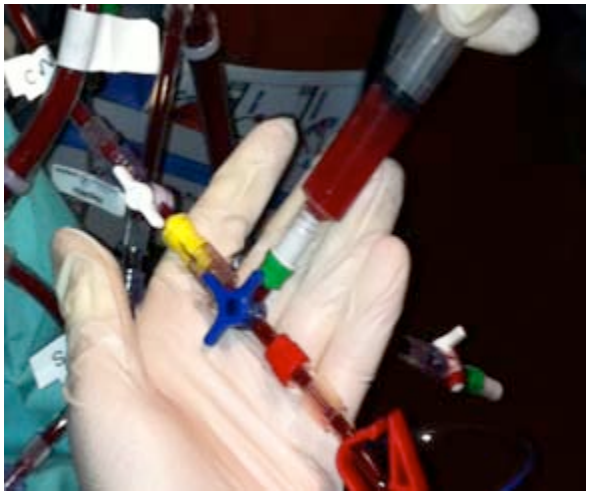
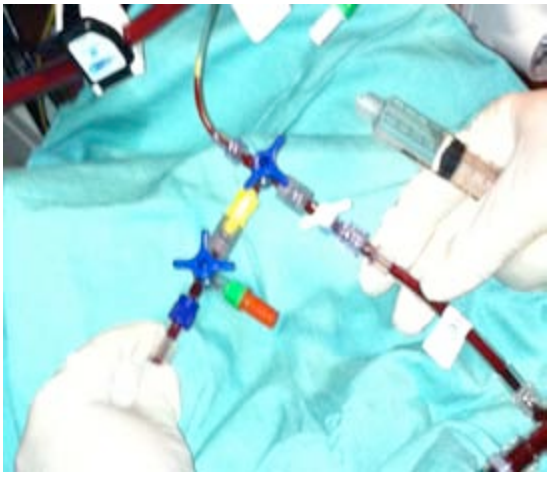
Personnel: ECLS Specialist & Haemofiltration specialist.

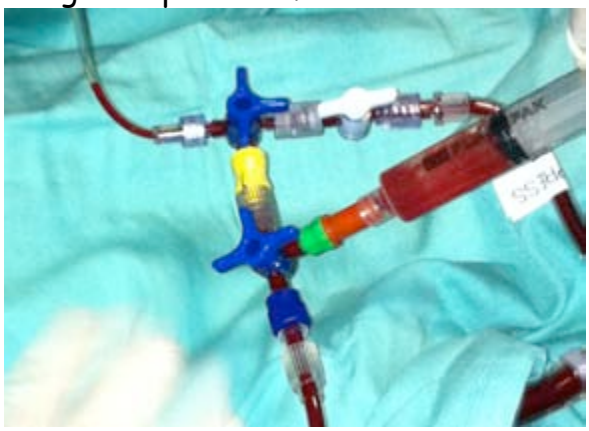
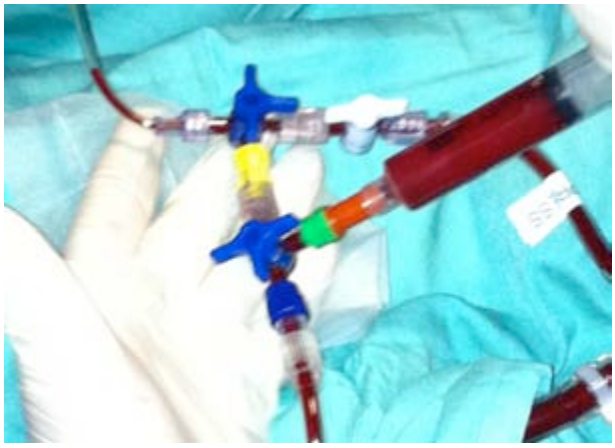
Equipment: Aquarius machine
Equipment as per Haemofiltration manual.

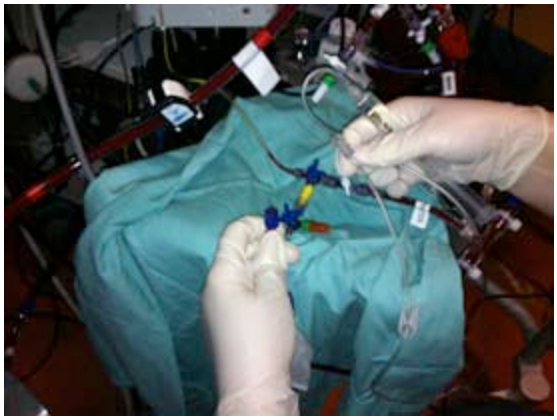
Specialist Action:

Action:	**Rationale:**
• Haemofiltration specialist to line & prime circuit as per Aquarius manual, however one (blue) Braun Disco flex 3-way tap to attach to each line. Attach and prime a green and white Bionector© on the side port of access line and a red one on the return line to be primed and connected. 	This is the key person experienced in using this equipment. To minimise risk of line contamination.
• Place a Tego® connector at the end of each line unless there are some already attached to the ECLS circuit. (These should be changed weekly). 	Needleless port for dialysis lines which will minimise risk of line infection and will allow for flow of 600ml / min.

• Circuit to be primed with 1 litre of 0.9% saline with 2000 units of heparin. • When complete the blood pump should be reprimed with 1 litre of heparin free 0.9% saline. • Proceed to recirculation for minimum 15 minutes. • Fill circuit with final prime fluid 50% mix of blood and 5% albumin or Attach using circuit to circuit prime. • The ECLS specialist will attach the (red) access line onto the pigtail#10 at the side of the oxygenator as shown below. There should be no bionector between the HF and ECLS circuit. A Tego® connector should be used. 	To cause minimum disruption to ECLS ACT's when circuit attached. To saturate membrane and prolong filter life. The mixture prevents blood with high haematocrit passing through the filter preventing clotting while not reducing the Hb significantly. Key personnel experienced in using this equipment. This will reduce the exposure to blood products.

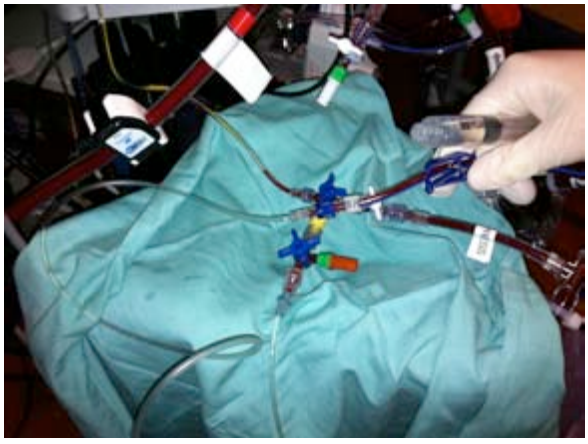
• Ensure there are no bubbles by aspirating from the ECLS circuit and then flushing the pigtail via the 3-way tap with a 10ml luer lock syringe containing 5mls 0.9% saline as shown below.  • They will then attach the blue (return) line to pigtail # (2) with the inlet pressure on the circuit. Proceed cautiously as this is potentially the negative, sucking side, of the circuit.  • When attaching the line turn the 3 way tap off to the ECLS circuit, the inlet pressure monitoring line and the HF circuit.	To prevent air entering the ECLS circuit.

• Ensure there are no bubbles by aspirating but NOT flushing the pigtail via the three-way with a 10ml luer lock syringe containing 5mls 0.9% saline. Take care to hold the plunger firmly so it is not sucked in by the negative pressure.  • The haemofiltration nurse will start the blood pump with the 3 way tap on the return line turned off to the ECLS circuit. The ECLS specialist gently aspirates any air from the 3 way tap.  • When satisfied that no air is present in the blue return line the 3 way tap should be opened to the ECLS circuit.	To prevent air entering the ECLS circuit.

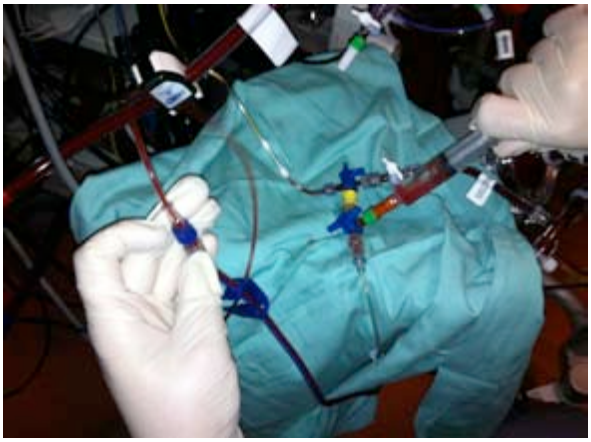
• The Emergency box should be available at the bedside in case of introduction of air into the circuit. • It is possible that the HF pump won't run due to the return pressures being too low. To troubleshoot try: 1. Increase the blood pump speed on the Aquarius. 2. Turn the 3 way tap on the return line slightly. or 3. If these manoeuvres don't work insert a narrow bore extension set. (UNANB800)  • Prime a narrow bore extension set with 0.9% sodium chloride.  • Stop the HF. Turn the 3 way tap	The protocol for evacuation of air from circuit should be used if air is present. This is due to the negative pressure in the ECMO circuit. To generate a positive pressure.

off to the ECMO circuit.

- Remove the return line from the 3 way tap.
- Attach the narrow bore extension set between the 3 way tap and the return line.



- Attach a 10ml leur lock syringe with 5mls 0.9% Sodium chloride to the bionector©. With the 3 way tap still off to the ECLS circuit the HF nurse will start the blood pump.
- The ECLS specialist will gently aspirate any air from the 3 way tap ensuring that enough discard has been removed to ensure that BOTH the connection at the return line and the 3 way tap have been de-bubbled.



- The 3 way tap should then be opened

to the ECLS circuit and the inlet pressure monitoring line.	
• It is possible that the HF pump will not run due to the access pressure being too positive. To troubleshoot this try: o Increase the HF blood pump speed. o Turn the 3 way tap slightly. o If these manoeuvres don't work insert a narrow bore extension set. Prime and attach the line as described above. When de-bubbling not that the blood is being pulled *from* the ECLS circuit therefore the 3 way tap should be closed to the HF line instead of the ECLS circuit as described above.	To generate a more negative pressure.
• If the filter is running with a narrow bore extension inline the maximum blood speed will be capped at 150ml/min.	Blood pump speeds greater than this will generate pressures out with the alarm limits.
• Blood pump speeds and pre-dilution rates on the Aquarius may need to be increased beyond normal HF parameters as a degree of recirculation will be present.	Due to the way the HF circuit is attached there will be a degree of recirculation. Monitor electrolytes closely.
• When starting the Aquarius blood pump, the ECLS flow and venous saturations should be noted. The RPM on the console may need to be adjusted to increase the flows and achieve the same venous saturations as before.	The filter may shunt a portion of blood away from the ECLS pump and the venous saturations may drop. (This is unlikely).

• Drugs should not be given into the ECLS circuit. Volume may be given.	Due to the way the HF circuit is attached any drugs given into the ECLS circuit will be in part filtered before they reach the patient.
• Inform HF nurse of any changes being made to ECLS flows.	Changing the ECLS flow will increase the pressures on the access and return limbs of the filter, potentially impairing its ability to run.
• The bags of Accusol must not be used after 24 hours. (see HF manual for reconstitution)	The bags lose stability after 24hours.
• The Aquarius should be set and adjusted (as per HF manual) only by the HF practitioner throughout the treatment.	This is the skilled practitioner to operate this equipment.
• Clinical assessment of fluid balance essential during the therapy.	Due to the high pressure / interaction between the two circuits especially on VA ECLS there is a risk of excess ultrafiltration.
• Circuits should be changed every 72 hours.	Manufacturer's guidelines.

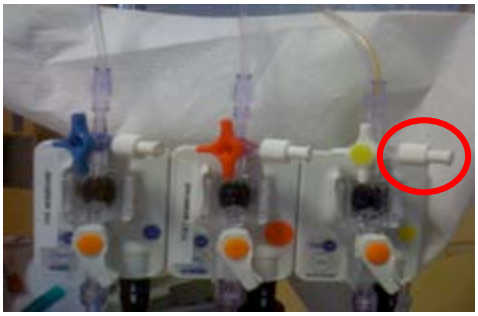
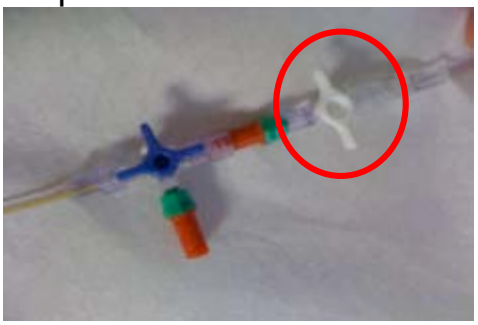
Title **To re-zero the inlet pressure**

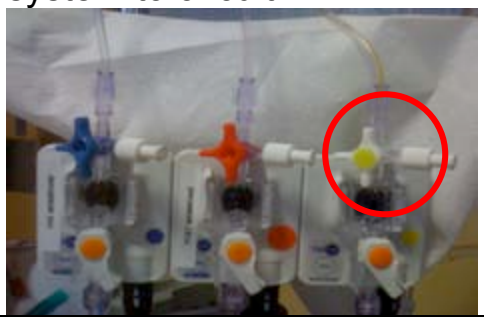
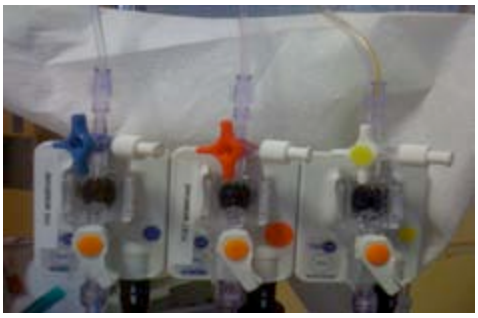
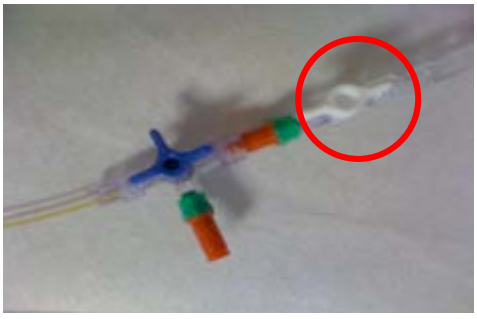
Rationale: To ensure circuit inlet pressure is accurate this should be carried out daily

Personnel: ECMO Specialist

Equipment: Chloraprep wipe
Sterile gloves
White Bung

ECMO Specialist Action:

Action:	**Rationale:**
Open supplies onto trolley and wash hands Put on sterile gloves Wipe white bung on 3-way tap (red circle in diagram) at transducer with Chloroprep wipe for 30 secs and then wait 30 secs to allow alcohol to dry.	To ensure sterility and minimise infection risk  To ensure decontamination of bung
Turn white 3-way tap at pigtail #2 off to the ECMO circuit.	To prevent accidental air entrainment 
Turn 3-way tap above pressure transducer off to the ECMO circuit and remove white bung.	To allow zeroing 

Hit pressure display on monitor Philips: • Scroll down to Zero pressure • Hit Zero P3 • Monitor will display zero done • Replace white bung and open 3-way tap at transducer to the circuit • Hit main screen	To zero pressure To reopen pressure monitoring system to circuit 
Once successful calibration is performed replace white bungs on transducer 3-way taps and turn taps to their original position	
Turn white 3-way tap at pigtail #2 back on to the circuit.	To display pressure on monitor 

NB if the inlet pressure is being measured on P1 on the console then if it becomes less than -99mmHg it will display as "---". Treat therefore as negative inlet pressure and assess cannula position, give volume, drop flows by 10% etc.

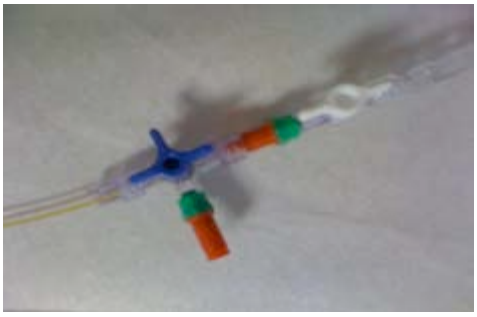
Title **Flushing of pressure monitoring lines**

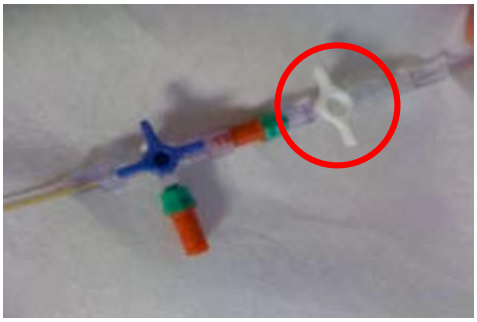
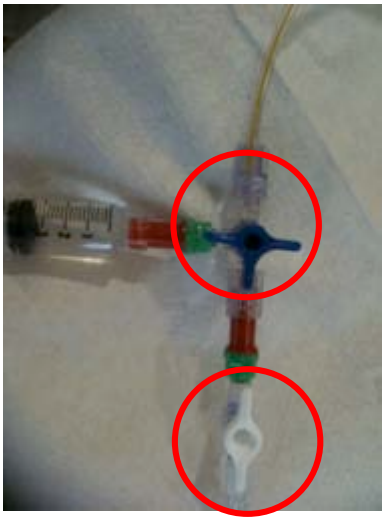
Rationale: To maintain patency of pressure monitoring lines by flushing each shift

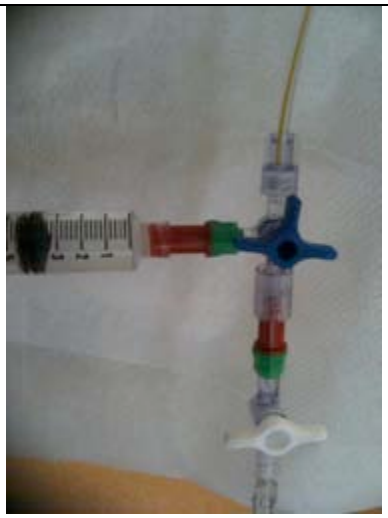
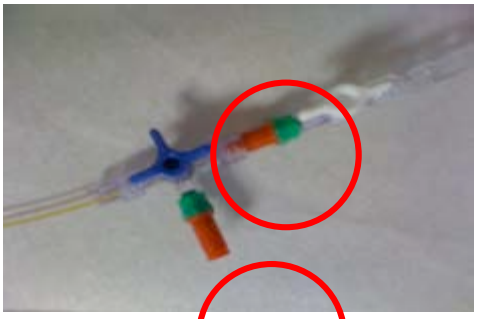
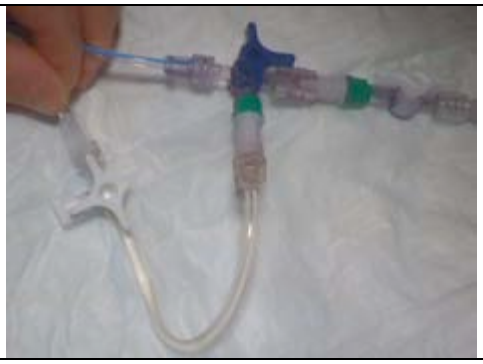
Personnel: ECMO Specialist

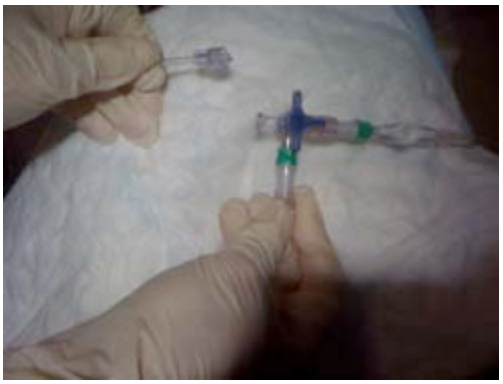
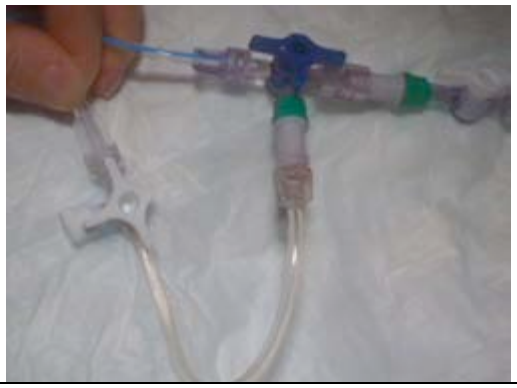
Equipment: Chloraprep wipe
Sterile gloves
2 x 3ml Leur lock syringes

ECMO Specialist Action:

Action:	**Rationale:**
The pressure monitoring lines should be taped up at all times.	To prevent blood tracking back up the line.
The Pre-oxygenator pigtail #5 line should not require flushing unless blood has tracked back past the 3-way tap.	Heparin infusion is infusing continuously through this pigtail.
To flush inlet #2 or post oxygenator # 7 pressure lines: • Pressurise Heparin/sodium flush line to 300mmHg. • Open pressure bag line clamp • Open white roller clamp on selected pressure line.	To allow flushing of pressure line through the pressure system
Open supplies onto trolley and wash hands Put on sterile gloves Wipe Bionecteur on 3-way tap at pigtail to be flushed with Chloroprep wipe for 30 secs and then wait 30 secs to allow alcohol to dry.	To ensure sterility and minimise infection risk  To ensure decontamination of bung

Turn white 2-way tap off to the ECMO circuit.	To prevent accidental air entrainment 
Draw up 0.5mls of 0.9% sodium chloride into 3ml leur lock syringe and attach to Bionecteur on 3-way tap Turn 3-way tap open to syringe and off to the pressure monitoring line and open white 2 way tap to allow aspiration of blood. Aspirate to ensure connection is bubble free and any clot is removed. Turn 3-way tap off to syringe. Draw up 2.0mls of 0.9% sodium chloride into 3ml leur lock syringe and attach to Bionecteur on 3-way tap. Turn 3-way tap open to syringe and off to the pressure monitoring line and open white 2 way tap debubble connection and flush pigtail.	

Close 3-way tap at pigtail off to circuit, open to monitoring line and close 2-way white tap. Press transducer flush lever and aspirate pressure monitoring line of any blood.	
Close 3-way tap to syringe and remove it, open taps to circuit	
• Close White roller clamp on line • Re-clamp pressure bag infusion line. • Depressurise Heparin/sodium flush line	To prevent accidental infusion of pressure line.
To flush Pre-Oxygenator pressure line turn 3-way tap off to pressure monitoring line at blue (pigtail #5) pre-oxy monitoring line therefore allowing continuation of heparin infusion.	
• Pressurise Heparin/sodium flush line to 300mmHg. • Open pressure bag line clamp • Open white roller clamp on selected pressure line.	

Clean pressure monitoring line tap connection for 30 secs with Chloroprep wipe and wait 30 secs then disconnect from heparin saline flush monitoring line. Press transducer flush lever on transducer until monitoring line is cleared	
Reconnect line to 3-way stopcock on pigtail #5 in a bubble free fashion and turn 3-way stopcock to its original position, open to heparin infusion and pressure monitoring line.	
• Close White roller clamp on line • Re-clamp pressure bag infusion line. • Depressurise Heparin/sodium flush line	To prevent accidental infusion of pressure line.

Title: **Antithrombin administration**

Rationale: Safe administration of antithrombin to enable effective Heparinisation

Personnel: ECMO Specialist x 2

Equipment: Required dose of antithrombin (see coagulation chapter)

ECMO Specialist Action:

Action:	**Rationale:**
Check ACT, FBC, coagulation screen (APTT, fibrinogen and D-dimer), Anti Xa (Heparin assay on HISS) antithrombin & ROTEM prior to administration	To ensure coagulation parameters have been optimised as much as possible
Discuss with consultant haematologist to release antithrombin from blood bank	To order antithrombin for named patient Order through Blood Bank
Prepare antithrombin aseptically	To prevent bacterial contamination
Administer dose ([100-Actual patient AT level] x body weight in kg) as a slow IV bolus over 3-5 minutes to the patient either via a central or peripheral line	
Clearly label vial and place in the fridge	Vial can be kept for up to 24 hours in the fridge and use for subsequent doses
Record administration in case notes and CIS – including dose and batch number	To allow an audit trail of unlicensed medication
Check ACT every 30 minutes till stable	Heparin requirement may fall as antithrombin will accentuate heparin effect
Recheck ACT, FBC, coagulation screen (APTT, fibrinogen and D-dimer), Antithrombin level, Anti-Xa & ROTEM approximately 60 minutes after administration	To monitor effects on the coagulation screen

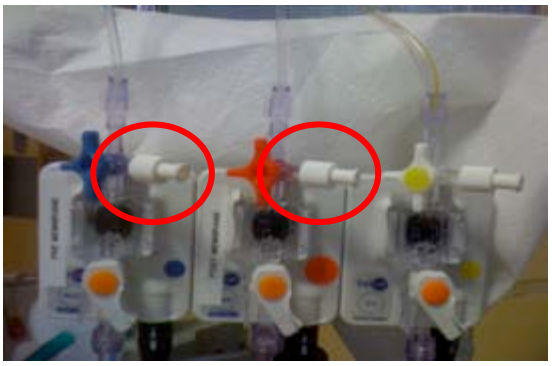
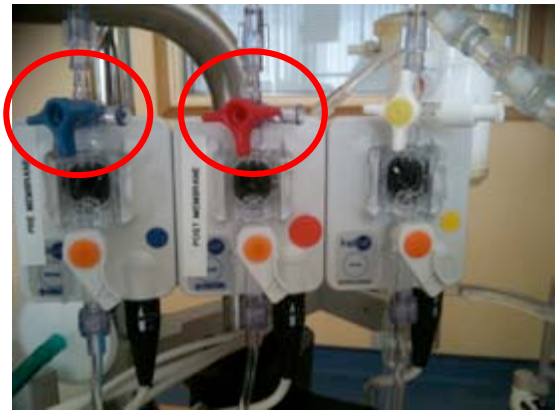
Title **To re-zero the pre and post Oxygenator pressures**

Rationale: To ensure circuit pressures are accurate

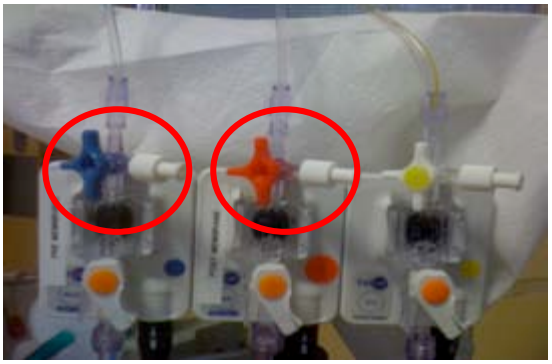
Personnel: ECMO Specialist

Equipment: Chloraprep wipes
Sterile gloves
2 x White Bung

ECMO Specialist Action:

Action:	**Rationale:**
Open supplies onto trolley and wash hands Put on sterile gloves Wipe white bungs on red and blue 3-way taps at transducers with Chloroprep wipe for 30 secs and then wait 30 secs to allow alcohol to dry.	To ensure sterility and minimise infection risk  To ensure decontamination of bung
Turn 3-way taps (Check these are connected to the pre/post Oxygenator pigtails) above red and blue pressure transducers off to the ECMO circuit and remove white bungs.	To allow zeroing of pressure lines and check correct pressure is re-zeroed 

Press Menu on console	
Press repeatedly until select pressure cal page displayed	
Press New Cal key	
Press Cal 1 and Cal 2 key	

After a few seconds zero should be displayed next to each pressure, if this is unsuccessful repeat above step.	
Once successful calibration is performed replace white bungs on transducer 3-way taps and turn taps to their original position	

Title: **Setting up inlet pressure monitoring**

Rationale: To ensure circuit inlet pressure is accurately setup and displayed

Personnel: Perfusionist

Equipment: Single pressure Phillips cable

ECMO Specialist Action:

Action:	**Rationale:**
PHILIPS MONITOR	
Attach single view link pressure cable from circuit transducer to pressure module. Press measurement selection key on screen	
Select relevant pressure label and activate	
Press set up measurement key scroll down if P3 not displayed press label and scroll down to P3 and select	To display correct pressure on monitor
Check default setting are: Alarm limits +20 to -40 (these may need to be adjusted for individual patient parameters) Alarm set to on Mean reading only Optimise scale To zero inlet pressure see protocol 27 “Zeroing of Inlet Pressure”	To ensure safe appropriate alarms are set

Title **ECMO Cannula care patency protocol**

Rationale: To maintain patency of cannulae during prolonged loss of ECMO support by flushing with heparinised saline.

Personnel: ECMO Specialist & PICU support nurse.
Senior doctor PICU / Cardiac

Equipment: Emergency cannulae patency pack
Clamps x2
2 Infusion pumps

Action: **Senior Specialist**:
See overleaf for action plan

Action:	Rationale:
<u>If ECMO cannulae flow is interrupted for more than 15mins and are unable to be flashed then cannulae must be flushed to maintain patency.</u> • Open sterile contents of patency pack onto dressing trolley. • Wash hands and put on gloves and gown. • Drape patient • Place infusion spike into heparinised saline bag and fill 2 x 50mls syringe. • Clean tubing at and 10cm below cannulae with Chloroprep then place sterile clamps above none sterile clamps. • Cut tubing, but <u>not</u> cannulae, in-between double clamps with sterile scissors. • Place white leur connectors into both ends of tubing at cannulae. <u>One cannulae at a time</u> • Place blunt ended needle onto filled 50ml syringe and fill tubing till meniscus is seen and all air is expelled from cannulae. Remove needle and place filled 50ml syringe onto connector using bubbleless connection. Release clamp and draw back to ensure connection is de-aired and any clot is aspirated then flush cannulae with 10mls of hep/saline. Re-clamp tubing at cannulae following flush leaving saline filled syringe connected. • **Repeat with 2nd cannula.** • **Unclamp and aspirate and flush both cannulae with 5mls hep/saline every 15mins until surgeon and perfusion arrive.**	**PUMP HEAD AND CIRCUIT WILL NEED CHANGED IF FLOW IS NOT ESTABLISHED WITHIN 5 MINUTES AND CIRCULATED THROUGH THE BRIDGE** To ensure sterility of procedure To ensure fluid for flushing cannulae is accessed safely. To remove bacteria. To prevent damage to the cannulae. This ensures that there is no leakage of blood from open ends of tubing. Leur lock end of connectors allows syringes to be attached to tubing. Blunt ended needle allows all air to be expelled from cannulae and replaced with fluid without risk of damage to tubing or operator. To prevent air embolism. To prevent stagnated blood clotting in cannulae. To overcome SVR and prevent backflow of blood into cannulae.

Title **Chest exploration on ECMO**

Rationale: To understand the risks of air emboli during re-exploration of the chest whilst patient is on ECMO support

Personnel: ECMO Specialist x2
PICU Consultant
Cardiac surgical consultant
Theatre team

Equipment: Spare Circuit & priming equipment at bedside
Clamps x2
1L de-airing bag 0.9% saline 50ml syringe

ECMO Specialist Action:

Action:	**Rationale:**
• Full team must be at bedside prior to routine re-exploration of chest. • During emergency tamponade senior doctor will decide if dressing needs to be key-holed.	To manage any air within the ECMO circuit which can occur during procedure. A balance of the potential risk vs benefits will be undertaken.
• Prime giving set with saline and connect to pigtail #1 (pre-pumphead) keeping bag 3 way tap off. • Place 50ml syringe on pigtail #7 at top of oxygenator keeping 3 way tap off to syringe	To provide volume reservoir if required to de-air To have syringe ready to de-air if required
• Team brief	To ensure all team aware of plans, and specific roles and responsibilities.
• One ECMO Specialist watches patient • One ECMO Specialist watches circuit for air entrainment with clamp for arterial (red) lumen in hand	To ensure no air entrainment and quickly deal with it if there is air entrained following appropriate protocol.

	,

Title **Cannula repositioning on ECMO**

Rationale: To understand the risks of air emboli during cannula repositioning on ECMO

Personnel: ECMO Specialist x2
PICU/NICU Consultant
Surgical consultant
Theatre team
ECHO Technician

Equipment: Spare Circuit & priming equipment at bedside
Clamps x2
1L de-airing bag 0.9% saline 50ml syringe
Spare cannulae and guidewires if required

ECMO Specialist Action:

Action:	**Rationale:**
Full team must be present prior to procedure Spare cannulae and guidewires should be available	To maintain patient oxygenation off ECMO If cannula requires to be replaced
Ensure any emergency drugs / volume (which may be required) are available and that IV lines are accessible	To maintain patient stability throughout the procedure
Team Brief	To ensure whole team aware of planned procedure and their role
Prime giving set with saline and connect to pigtail #1 (pre-pump head) and leave clamped off Place 50ml syringe on pigtail #7 at top of oxygenator	To provide volume reservoir if required in the event of an air emergency To easily and quickly remove air from the circuit

One ECMO Specialist watches patient vital signs One ECMO specialist observes ECMO circuit for air entrainment	To observe for instability To promptly react to any air entrainment
If RPM / flows are high or inlet pressure if very –ve then flows may need to be reduced during actual cannula manipulation to reduce risk of air entrainment	Patient may need increased conventional support during procedure
If converting from chest to neck cannulation, flow may need to be interrupted for several seconds by clamping arterial limb of circuit during insertion of new venous cannula	To prevent air being sucked through new cannula as it is advanced into the R Atrium This should be discussed prior to procedure to ensure perfusion and the ECLS consultant are aware

ECMO Perfusion checklist

DATE: _____ HOSP.NO:_____

PATIENT: _____ WEIGHT _____ kg

PERFUSIONIST 1. _____ PERFUSIONIST 2. _____

PACK: _____ ser.no. _____

OXYGENATOR: _____ ser.no. _____

PUMP HEAD: _____ ser.no. _____

BASE NO: _____ WATER HEATER NO: _____

PUMP NO: _____ GAS BLENDER NO: _____

K^+: _____ mmol/l

1.	Check both consoles are fully charged	
2.	Correct circuitry selected based on weight	
3.	Circuit primed and bubble free	
4.	Flow probe connected	
5.	Bionectors on all pigtails	
6.	Pre and Post pressure lines zeroed and taped upright	
7.	Venous pressure cable from patient monitor connected and zeroed	
8.	Potassium level measured (only if blood primed) 5mls of saline must be added to circuit as sampling to prevent cavitation	
9.	Gas blender FiO_2 set at 100%	
10.	Water heater connected and temperature set	
11.	Check O_2 cylinder is over ½ full	
12.	Spare console/motor available and plugged in	

CANNULAE: _____ ser.no. _____

_____ ser.no. _____

ECMO HANDOVER

ECMO SPECIALIST 1._____ ECMO SPECIALIST 2._____

1.	Heparin infusion connected & tap open	
2.	Heparin Infusion running	
3.	Venous pressure displayed on patient monitor and 5 to -50mmg	
4.	Pre/Post pressure displayed on console	
4.	Upper and lower pressure alarm limits set	

COMMENTS:

ECMO VA Routine Trial Off Checklist

PATIENT NAME _____ **DATE** _____

1.	Wean and bridge off discussed/documented with ECLS physician & team • Ventilation optimised (? Bronchoscopy / NO) • Cardiac support optimised (inodilators/pacing etc) • Adequate sedation & paralysis	
2.	Recent ECHO performed to assess cardiac function	
3.	ECMO flows weaned as per documented plan	
4.	Surgeon aware of planned trial off and potential for decannulation	
5.	Patient's ABG/VBG and vital signs stable on reduced ECMO flows	
6.	Recent CxR performed & reviewed	
7.	Inotrope / Vasodilator infusing to the patient	
8.	Centrifugal Circuit ACT 220-240, a bolus may be required to achieve this prior to clamping of (**any deviation** from this must be clearly documented)	
9.	Prepare a new Heparin infusion (at the same concentration as the circuit Heparin) and connect to the patient's IV line (ensure minimal dead space in line)	
10.	**Immediately prior** to bridging off: • Reduce circuit heparin infusion to half previous rate • Commence patient Heparin infusion at full rate *(target ACT 220-240)*	
11.	Clamp patient off the ECMO circuit **A – B - V**	
12.	Ensure blood flows are at least half the maximum rated flow of the oxygenator whilst on bridge to minimise risk of clotting oxygenator • Medos 0800Lt = 0.4 l/min blood flow on bridge • Medos 2400Lt = 1.2 l/min blood flow on bridge • Medos 7000Lt = 3.5 l/min blood flow on bridge	
13.	Remove green gas line from oxygenator during trial off (to prevent supersaturation)	
14.	Check immediate pump and patient ACT and repeat both every 15mins (after flashing cannula) during trial off. *2ml of saline must be injected into circuit prior to each episode of sampling to prevent cavitation*	
15.	Flash cannulae to prevent clots by releasing (V-B-A) and clamping (A-B-V) every 15mins, observe pre-membrane pressures post-flash. *RPM will need increased prior to flashing to move flow forwards*	
16.	Check ABG/VBG ~20mins post-clamping off & • optimise ventilation / inotropes support as required & plan decannulation or re-institute ECMO support	
17.	If ECMO re-instituted **ensure green gas line is reattached**, circuit Heparin rate increased and patient Heparin infusion discontinued. Review flows and ACT parameters. May need to sigh membrane.	

Title: **Low flow VA wean centrifugal ECMO set-up**

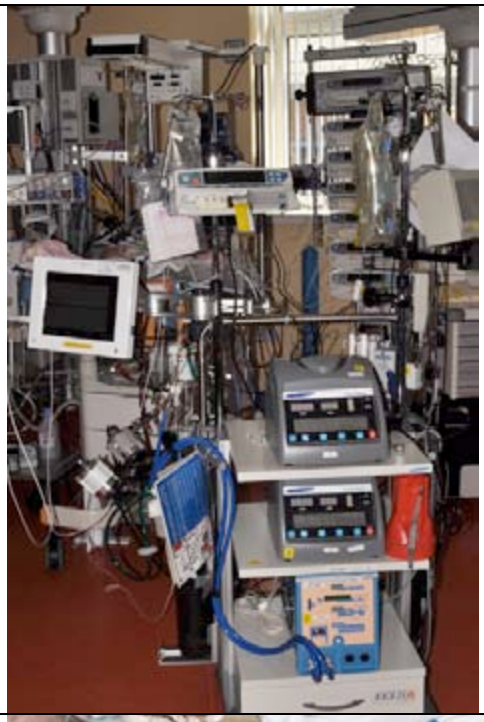
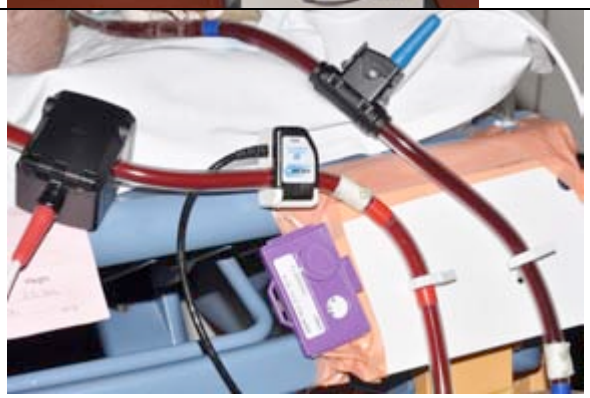
Rationale: To allow patient flows of less than 200ml/min, which is the minimum rated flow of the membranes, on VA ECMO

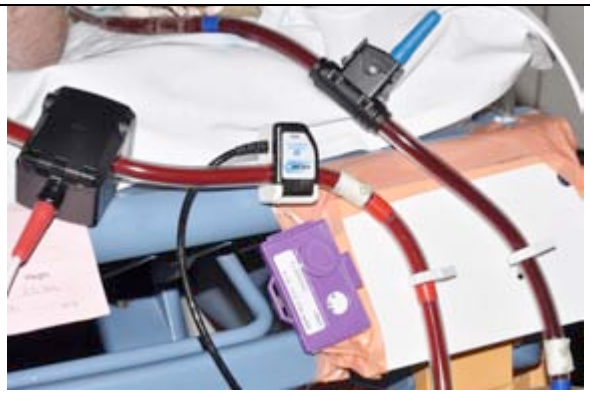
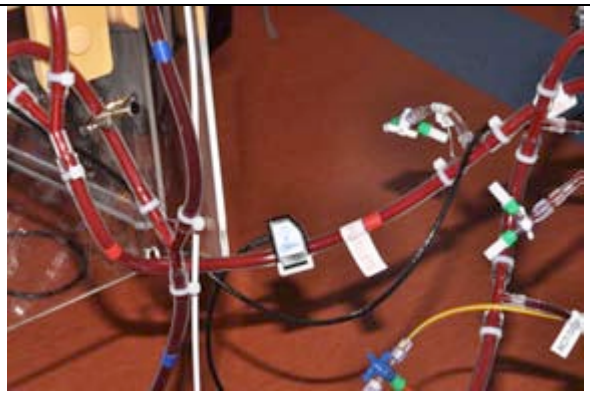
Personnel: ECMO Specialist, Perfusionist & Consultant

Equipment: Spectrum Medical SvO2 module (on mobile ECLS trolley) associated saturation probes and power cable
2nd flow probe for Levitronix
Adjustable gate clamp
Labels

ECMO Specialist Action:

Action:	**Rationale:**
Joint plan agreed for wean of patient on VA ECMO requiring trial of flows <200ml/min. ▪ This should be done during the day with all staff available for decannulation if successful. ▪ Do not undertake this overnight.	Flows <200ml/min are below the rated flow of the Medos membrane. Flows below this may result in the membrane clotting. By following this SOP a higher flow will be maintained within the circuit but low flow exiting to patient
Ventilation should be optimised and chest X-ray checked prior to wean.	▪ Ensure no non-cardiac reason for wean failure
Patient should be paralysed and sedated appropriately	▪ To minimise oxygen consumption
Liaise with Duty Perfusionist and access the following: ▪ Spectrum Medical SvO2 module (on mobile ECLS trolley) associated saturation probes and power cable ▪ 2nd flow probe for Levitronix in ECMO set-up room ▪ Low flow wean bag stored in ECMO set-up room. Contains: o Adjustable gate clamp o Coloured spare head for back-up console ▪ Labels	

	
Brief staff on duty including nurse in charge re plans for low flow wean	▪ Ensure no communication breakdown.
Gather supplies, wash hands, put on gloves and apron.	
Attach Spectrum medical SvO2 module to ECLS trolley as per photo. ▪ Spectrum Medical saturation monitor allows monitoring of arterial and venous limb saturations. ▪ It does not need calibrated. ▪ Do **not** press the data reset button. ▪ The Spectrum medical has an internal battery life of approx 30mins. ▪ Perfusion will set this up.	
Place venous and arterial Spectrum Medical saturation connectors onto tubing at patient side of bridge. ▪ These saturation connectors do not need to be moved.	

Place flow probe from top console on patient side of arterial return and label top console “patient” on front of console. This will allow the flow to the patient to be monitored.	
Place flow probe from back-up or lower console onto arterial limb on non-patient side of the bridge this enables the flow within the circuit to be monitored. Label bottom console “circuit” on front of console	
Increase ACT parameters by 20 to allow for lower flow through the cannulae	▪ To ensure no clotting of cannulae
Place adjustable gate clamp over bridge tightening to occlude bridge completely then release bridge clamp. ▪ Slowly open gate clamp in ¼ turns till patient flow at target for start of wean.	
Document venous SaO2 from Spectrum medical and not CDi101 (normal venous saturation monitor) Switch off CDi101 to ensure no confusion	▪ Venous SaO2 will now come from Spectrum Medical probe on venous drainage on the patient’s side of the bridge as this is true SvO2.

Document wean process and targets in CIS or notes. ▪ Ensure Blender O2 is weaned to at least 50% during wean ▪ Wean gas flow as required on circuit gas	▪ To ensure oxygenation adequate on ventilator ▪ Less CO2 removal will be needed as less blood passing through circuit from patient
Success of wean to be decided clinically and on echo and biochemical markers of cardiac function and oxygen delivery. ▪ In cardiac patients expect a rise in lactate as there is obstruction to aortic flow from aortic cannula. ▪ In patients with balanced physiology, eg HLHS then patients will be desaturated and NIRS will fall in line with this. The difference between arterial and venous SaO2 is the important thing that will guide a wean being successful or not.	
Decannulation should take place early pm at the latest. Ideally wean should happen during the day then fully support over night with a view to a short wean and decannulation the next morning.	
On completion of wean put all kit from "Low flow wean" bag back in ECMO set-up room. **Do not throw out spare head as these are expensive.** Return Spectrum Medical SvO2 module to mobile ECLS trolley	

ECMO VV Trial Off Checklist

PATIENT NAME _____ **DATE** _____

1.	Gas flow wean discussed/documented with ECLS physician & team • Ventilation optimised (? Bronchoscopy / NO / HFOV) • Cardiac support optimised (inodilators / Hb etc) • Adequate sedation & paralysis *NB. VV wean involves gas flow and blender wean not blood flow wean. No need for bridge off or heparin to patient*	
2.	Recent CxR performed & reviewed	
3.	Surgeon aware of planned cap off and potential for decannulation	
4.	Are present ECMO settings suitable for capping off • Blender FiO2 <30% • Sweep gas at low flow for oxygenator size (see charts) • Satisfactory ABG/VBG • Maintain previous ACT parameter and check ACT 1hrly as per normal protocol	
5.	To cap off remove green gas line from in line filter and loop round onto bottom of oxygenator	
6.	Check ABG/VBG ~20mins post-capping off then every 1-2hrs • optimise ventilation / inotrope support as required & plan decannulation or re-institute ECMO support	
7.	If ECMO re-instituted **ensure green gas line is reattached**. May need to sigh membrane.	

Fibrinogen Concentrate (Riastap)

Fibrinogen (factor I) is a soluble plasma glycoprotein which circulates in plasma as a precursor of fibrin. Fibrinogen is an important element in the clotting cascade as it impacts on the structure and stability of the clot formation. Normalization of plasma fibrinogen levels may be associated with satisfactory haemostasis and reduced bleeding. ECMO patients are at an increased risk of bleeding due to the effects of Heparin and the bonding of plasma proteins onto non-endothelial cell surfaces. Fibrinogen is one of the main proteins absorbed by the artificial surfaces of the ECMO circuit. Patients managed on ECMO will have a prescribed Fibrinogen parameter of > 200mg/dl. To attain and maintain this level soon after cannulation most patients will require 1 or 2 units of Cryoprecipitate, for a Neonate, and up to ~ 6 units for an adult. Levels are then usually well maintained during the run, except in certain patient groups.

ECMO patients that may be particularly at risk of increased consumption of Fibrinogen are those with active bleeding, sepsis, DIC and clots either within the ECMO circuit or body. If administration of Cryoprecipitate is not giving an adequate increment of Fibrinogen levels, or excessive volume load or accessibility of cryoprecipitate supplies becomes an issue, then Human Fibrinogen Concentrate may be given.

Human Fibrinogen Concentrate (RiaSTAP) has proven efficacy and is licensed for use in the control or prevention of bleeding in congenital fibrinogen deficiency. Increasing evidence from case reports and case series also indicate its potential usefulness in non-congenital fibrinogen deficiency patients with intractable bleeding, or even as prophylaxis prior to high blood-loss surgery. Human Fibrinogen Concentrate is at present unlicensed for such indications and is expensive (~£400). Nevertheless there may be a place for considering its use to optimise Fibrinogen levels in ECMO patients especially for the indications above.

The mechanism of action of RiaSTAP® serves as a physiological substrate of thrombin (factor IIa), which converts soluble fibrinogen to insoluble fibrin. Under the influence of factor XIIIa, fibrin strands are cross-linked to provide strength and stability to the blood clot—fulfilling an essential need for clot formation in patients with Fibrinogen deficiency.

How to obtain Human Fibrinogen Concentrate

The use of Human Fibrinogen Concentrate treatment must be approved by both the on-call Consultant Paediatric Intensivist (if PICU patient) or Neonatologist (if NICU patient) and the on-call Paediatric Haematologist. Vials can be obtained from blood bank on a named patient basis. Two doses should be ordered initially.

Pre- Fibrinogen Concentrate management

There is very limited experience of the use of Human Fibrinogen Concentrate in infants and children. The most serious adverse reactions that have been reported in adult subjects who received RiaSTAP® are thromboembolic episodes, including myocardial infarction and pulmonary embolism, and allergic-anaphylactic reactions. The most common adverse reactions observed are allergic reactions, including chills, fever, nausea, and vomiting. Patients should be monitored for early signs of allergic or hypersensitivity reactions, and if necessary, discontinue administration. The risk that Fibrinogen Concentrate may clot the Oxygenator of the ECLS circuit or cause thrombotic incidents appears to be reasonably low in our patient group but it is important that an accurate level of Fibrinogen and coagulation status is determined prior to administration of Human Fibrinogen Concentrate , therefore:

- Check ACT
- Full Coagulation screen "/ECLS1" order set on HISS
- ROTEM (inc FibTEM assay).

Thromboelastography/ thromboelastometry looks at the clotting process holistically, in real time, from formation of the initial fibrin strands through to clot lysis. The derived parameters are the clotting time (CT), clot formation time (CFT), maximum clot firmness (MCF) and maximum lysis (ML). In **FIBTEM**; platelets are blocked, so clot formation is dependant on fibrin formation and polymeristaion. Discrepancies will often be seen between the FIBTEM and the laboratory level of Fibrinogen as FIBTEM is more sensitive to clot polymerisation disorders. Normal parameters for children have not yet been determined but the adult parameters are a MCF of 9-25mm. Levels of < 9mm are associated with an increased risk of bleeding.

Human Fibrinogen Concentrate, Pasteurized (Riastap) Preparation

Riastap is a sterile, preservative free, lyophilized fibrinogen concentrate in a single-use vial. The labelled amount of Riastap is 1 g of fibrinogen with the actual potency for each lot indicated on the vial label and carton. Riastap is reconstituted with 50ml Sterile Water for Injection (20 mg/mL) and is administered intravenously. Each vial contains between 900 to 1300 mg fibrinogen and 400 to 700 mg human albumin. Riastap is stable for 24 hours after reconstitution when stored at 20-25°C and should be administered within this time period. Partially used vials should be discarded after 4 hours.

Human Fibrinogen Concentrate, Pasteurized (Riastap) dosing and administration

There are very few case studies which determine the recommended dosage of Riastap in children, although a shorter half life and faster clearance than in adults has been observed. There is some evidence from our limited clinical experience that the dosage recommendation for congenital fibrinogen deficiency is inadequate for our patient group. Therefore an initial dose of 80 mg/kg Fibrinogen Concentrate should be given by slow IV bolus (3-5 mins) to the patient and **not** pre-oxygenator, the injection rate should not exceed 5 mL per minute. A repeat Coagulation screen and ROTEM should then be obtained within 2 hours to recheck levels of Fibrinogen and access need for further doses. Further doses may be repeated if there is still evidence of hypofibrinogenemia as determined by Clauss Fibrinogen and ROTEM assay. Further doses of Riastap can be incremented by 20% if an insufficient response is obtained from the initial dose.

Post Human Fibrinogen Concentrate Monitoring

The following clinical variables should be monitored in any patient receiving Human Fibrinogen Concentrate:

- Monitor for signs of allergic reactions
- Check ECLS circuit after 30mins for evidence of new clot formation
- Monitor inlet, Pre/Post Membrane pressures
- Repeat Coagulation and ROTEM within 2hrs of dose

Record of Human Fibrinogen Concentrate

As with all blood/coagulation products, details of dose and batch number should be recorded in the case notes.

Patient weight	Calculated dose per formula	Actual dose given	Time given	Pre Fib level	Post Fib level
Pt no 1 - 2.5kgs	100mgs 40mg/kg	10mls/200mgs 80mgs/kg	18:00	1.32	1.43
	84mgs 33.5mgs/kg	10mls/200mgs 80mgs/kg	23:00	1.43	1.5
	107mgs 42.9mg/kg	15mls/300mgs 120mgs/kg	18:10	1.27	1.79
	138 55.3mgs/kg	20mls/400mgs 160mgs/kg	08:00	1.06	1.9
Pt no 2 - 3.5kgs	33mgs 9.4mgs/kg	12.5mls/250mgs 71.5mgs/kg	17:00	1.84	2.63
Pt no 3 - 3.73kgs	31mgs 8.4mgs/kg	14.8mls/296 80mgs/kg	11:30	1.80	2.61
		14.8mls/296 80mgs/kg	22:15	1.86	2.96

Review of RioSTAP dosing and increment of Fibrinogen in three ECMO patients in RHSC

References

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Heindl B, Delorenzo C, Spannagl M High dose fibrinogen administration for acute therapy of coagulopathy during massive perioperative transfusion. Anaesthesist 2005;54:787-90. doi:10.1007/s00101-005-0865-7. CrossRefMedlineWeb of Science

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Ucar HI, Oc M, Tok M, et al. Preoperative fibrinogen levels as a predictor of postoperative bleeding after open heart surgery. Heart Surg Forum 2007;10:E392-6. doi:10.1532/HSF98.20071065. CrossRefMedlineWeb of Science

Title: **Concentrated Fibrinogen administration**

Rationale: Safe administration of concentrated Fibrinogen to maintain parameter

Personnel: ECMO Specialist x 2

Equipment: Required dose of concentrated Fibrinogen (see coagulation chapter)

ECMO Specialist Action:

Action:	**Rationale:**
Check /ECLS1 on HISS (ACT, FBC, coagulation screen - APTT, fibrinogen and D-dimer, Anti Xa (Heparin assay) antithrombin & TEG prior to administration	To ensure coagulation parameters have been optimised as much as possible
Discuss with blood bank	To order concentrated Fibrinogen Order from HISS through BB – BPR –- Concentrated Fibrinogen
Prepare concentrated Fibrinogen aseptically. Riastap is reconstituted with 50 mL Sterile Water for Injection (20 mg/mL)	To prevent bacterial contamination
An initial dose of 80 mg/kg Fibrinogen Concentrate should be given by slow IV bolus (3-5 mins) to the patient and **not** pre-oxygenator, the injection rate should not exceed 5 mL per minute.	There is some evidence from our limited clinical experience that this dosage is required for correction of congenital fibrinogen deficiency
Check circuit every 15 minutes for first hour post dose	To check for evidence of new clot formation
Check ACT every 30 minutes till stable	Heparin requirement may increase as Fibrinogen level increases
Clearly label vial and place in the fridge	Vial can be kept for up to 4 hours in the fridge and use for subsequent doses
Further doses of Riastap can be incremented by 20% if an insufficient response is obtained from the initial dose.	To ensure adequate response to product

Record administration in case notes and CIS – including dose and batch number	To allow an audit trail of unlicensed medication
Recheck ACT, FBC, coagulation screen (APTT, fibrinogen and D-dimer), Antithrombin III level, Anti-Xa &TEG approximately 60 minutes after administration	To monitor effects on the coagulation screen

Title: **Circuit change**

Rationale: Safe elective or emergent change of circuit

Personnel: ECMO Specialist x 2
ECLS Physician
Perfusionist
Surgeon and assistant
Theatre team only if cannula repositioning or exploration planned

Equipment: New Circuit, 2 circuit connectors, Priming Unit, Priming drugs, Heparin Infusion, Sterile blade, Big scissors, Sterile clamps, theatre hats/masks/gloves, 2 packs sterile green drapes, 2 sterile gowns, Dressing pack, Foil bowl, scissors, saline, 50ml bladder tip syringe, Betadine for cleaning, emergency drugs, volume

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ECMO Specialist Action:

Action:	**Rationale:**
Contact all required personnel, if elective liaise with perfusion and medical staff to arrange suitable time for change	To ensure the whole team is available and prevent delays.
If appropriate delay routine CxR until after circuit change	To prevent repeated exposure to radiation
Prepare Emergency Drugs and Volume o Adrenaline o Atropine o Calcium o Sodium Bicarbonate o Volume PPS/0.9% Saline o PRBC if HCT borderline o Plts if <100,000	To support patient if unstable during circuit change or problem arises
Prepare new Heparin Infusion	To connect to new circuit
Request Priming unit and add priming drugs as per Priming form o 25mls of Tham 4% o 20mls of Albumin 20% o 10mmols of Sodium Bicarbonate 8.4% o 150units of unfractionated Heparin	To Buffer PRBC for blood prime (use 1ml syringe)

○ 3mls of Calcium Gluconate 10% Sign Yellow iv additions label	Give Priming unit to perfusionist
Discuss ventilation strategy during circuit change (Consider previous history of airleak) Consider Nitric if patient has significant PPHN	Hand Ventilation or increased ventilatory settings Prepare Nitric and connect if required
Prepare sterile trolley and assist surgeon whilst scrubbing and gowning Open sterile packs	To maintain sterile field
Ensure pre procedure Brief is undertaken prior to circuit change Discuss – Who will perform clamping sequence / support new circuit tubing – Where old circuit is to be cut – Circuit connectors required? – Who will adjust RPM / flows – New sweep gas flow (may need to be reduced on new circuit) – Who will assist ECMO physician	To ensure team is fully aware of proposed plan and individual role during procedure To allocate roles appropriately and minimise time of ECMO during circuit change
Procedure – Clamp patient off ECMO – Switch of old pump – Surgeon clamps cannulae – Surgeon cuts old circuit off – Surgeon inserts new connectors to cannulae if required – New circuit with clamps insitu handed to assistant and new sterile circuit tubing cut to size – Perfusion hands up circuit, arterial limb first then venous – Assistant uses Saline and bladder tip to provide bubbleless connection – Check connection for air bubble – All clamps removed other than two clamps on new circuit – Clamps removed, RPM incremented to achieve flow, Gas tubing connected, Heparin infusion started	ABV if centrifugal Pump VBA if Roller Pump To remove old circuit To connect new circuit To prevent the accidental connection of the circuit the wrong way ie venous tubing to arterial cannula To prevent air embolism VBA if centrifugal Pump ABV if Roller Pump

Circuit handover checklist completed by Perfusionist and ECMO specialist Conduct full circuit check 1/2hrly ACT's until stable, may initially be higher Check full set of ECLS bloods CxR post circuit change Check circuit and patient blood cultures within 12hrs	To ensure safe handover to ECMO specialists To detect any circuit problems Due to heparin in priming unit To assess if Blood products or electrolyte replacement is required To check cannula position and check for pneumothorax To detect any infection
Dispose of discarded blood filled circuit in Yellow bin. Clean ECMO cart / clamps / cables with Tuftie wipes, pay particular attention to blades on motor drive Store pump in ECMO office and ensure plugged in	To ensure adequate response to product To remove any blood and decontaminate equipment To maintain battery capacity.

ECLS CIRCUIT BLOOD PRIME

Addressograph	Date: ..... Time: .....

Batch/Laboratory Number: ..... &
Expiry date of Albumin 20%: .....

To each Unit of PRBC in Prime:

1. Add 20 ml 20% Albumin
2. Add 25ml THAM
3. Add 10 mmol $NaHCO_3$
4. Add 150 units Heparin
5. Agitate well (by rocking)
6. **Then**, add 3 ml 10% Calcium gluconate and agitate again

UNIT	UNIT NUMBER:	ADDED BY:	CHECKED BY:
1.	.....	.....	.....
2.	.....	.....	.....
3.	.....	.....	.....
4.	.....	.....	.....

Prime volume in the circuit:

Add volume to the primed circuit until the post-oxygenator pressure measures 100 mmHg at a pump rate of 200 ml/min

Doctor's signature: .....

Title: **EMERGENCY "COMING OFF" VAD SUPPORT**

Description: Procedure to follow to remove from bypass

Personnel: VAD Specialist.
PICU support nurse.
Consultant PICU
Senior medical support
Perfusionist on site/contact.

Equipment: Clampsx2
Emergency backup equipment.
Emergency drugs.
Volume as required.

VAD Specialist Action:

Action:	**Rationale:**
Clamp outflow (post head) tubing. Depress red key on Centrimag console and hold for 2 seconds.	Rapid cessation of bypass in an emergency and avoid danger to patient.
Utilise medical and nursing support to provide emergency care to patient.	Avoid cardiovascular instability in patient and adverse sequelae.
Call perfusionist and undertake technical procedure as required.	Perfusionist will provide expert knowledge changing circuit. Initiate emergency procedures as qualified to provide.
PUMP HEAD AND CIRCUIT WILL NEED CHANGED IF BYPASS NOT ESTABLISHED WITHIN 5 MINUTES.	Clots will accumulate in pump and circuit if anticoagulation not adequate.

Title: **MANAGEMENT OF RETROGRADE FLOW**

Rationale: To understand the risks, causes and management of retrograde blood flow.

Personnel: VAD Specialist.
PICU support nurse.

Equipment: Circuit Clamp x2

VAD Specialist Action:

Action:	**Rationale:**
Retrograde flow will be displayed on Levitronix console as - - - . If this is displayed immediately clamp tubing post pump head.	Retrograde flow can occur at low RPM or during significant increases in MAP and will cause acute clinical deterioration.
Observe that flow probe is correctly placed on tubing	If flow probe is incorrectly placed or not secure - - - can be displayed on console.
Increase RPM by approximately 10%. i.e 2000 to 2200.	To overcome patients Systemic Vascular Resistance
Slowly release clamp observing for signs of forward flow. If more RPM are required increase in 10% increments.	Forward flow of blood Flows displayed on console Improving patient condition
Once adequate support has been achieved wean RPM to give desired flow rate.	

Title: **CHEST EXPLORATION ON VAD**

Rationale: To understand the risks of air emboli during re-exploration of the chest whilst patient is on VAD support

Personnel: VAD Specialist & PICU support nurse.
PICU Consultant
Cardiac consultant
Perfusionist
Theatre team

Equipment: Spare Circuit & priming equipment at bedside
Clamps x2

VAD Specialist Action:

Action:	**Rationale:**
Full team must be at bedside prior to routine re-exploration of chest. During emergency tamponade senior doctor will decide if dressing needs to be key-holed.	To manage any air within the VAD circuit which can occur during procedure. A balance of the potential risk vs benefits will be undertaken.
Prior to chest being accessed the flows should be reduced by 25 – 50%(ensure above minimum flows). The patient's haemodynamic stability should be carefully monitored during this time.	A reduction in RPM/Flows will reduce the negative pressure exerted on the cannulae and make air less likely to be accidentally sucked into the circuit during manipulation of the cannulae
Great care should be taken during exploration to minimise any manipulation of the cannulae especially the inlet side.	To reduce the risk of air in the circuit.
Once the procedure is finished RPM/Flows should be carefully returned to original values.	Slight changes to cannulae position during procedure can result in flow problems.

Title: **DEFIBRILLATION ON VAD**

Rationale: To understand the risks and management of a patient on VAD during defibrillation.

Personnel: VAD Specialist & PICU support nurse
PICU Consultant.
Cardiac surgeon and perfusion to be informed

Equipment: Defibrillator
Clamps x2

VAD Specialist Action:

Action:	Rationale:
Patients can be defibrillated with the device running, but it is mandatory to have a backup console available and switched on.	To ensure console can be changed if malfunction occurs
Prior to defibrillation, the tubing should be checked to ensure they are dry from blood and other fluids.	To prevent conduction of current along tubing
Ensure all staff stand clear of the patient, device and console during defibrillation. (European Resuscitation Guidelines)	To prevent injury
The console should be checked for continued normal operation immediately after each defibrillation attempt.	To detect any console malfunction
Note that an alteration in patients rhythm may require an alteration in the pump RPM and/or the patients filling pressures to maintain adequate pump flow.	Changes to rhythm can alter filling pressures, SVR, PVR and therefore affect pump flow rates.

Title: **RVAD - Management of external cardiac massage**

Rationale: To understand the risks and management of patients during external cardiac massage on RVAD.

Personnel: VAD Specialist & PICU support nurse.
PICU Consultant.
On Call perfusionist
On call cardiac surgeon

VAD Specialist Action:

Action:	**Rationale:**
Ensure appropriate help is sought ie On-call perfusionist and cardiac surgeon	Cannulae may be dislodged during resuscitation. VAD circuit may clot during cardiac arrest
External cardiac massage carries a high risk of dislodging the VAD cannulae, and is therefore highly risky and should be performed only on the instruction of a doctor.	Internal cardiac massage may be safer if appropriately trained member of staff is available.
Wean Flows to 30% of baseline(ensure above minimum flows). One specialist observes circuit for air entrainment.	To reduce increased risk flooding of pulmonary bed. Flows continued to reduce risk of circuit clotting
If air is entrained clamp patient of circuit immediately on outlet tubing (post head)	To prevent air embolism.
Troubleshoot and treat cause of arrest	Abnormal Rhythm – defibrillation Electrolyte imbalance Tamponade
Observe circuit and cannulae for clots prior to resumption of flows.	To prevent emboli

Title: **LVAD - Management of external cardiac massage**

Rationale: To understand the risks and management of patients during external cardiac massage on LVAD.

Personnel: VAD Specialist & PICU support nurse.
PICU Consultant.
On Call perfusionist
On call cardiac surgeon

VAD Specialist Action:

Action:	**Rationale:**
Ensure appropriate help is sought ie On call perfusionist and cardiac surgeon	Cannulae may be dislodged during resuscitation. VAD circuit may clot during cardiac arrest
External cardiac massage carries a high risk of dislodging the VAD cannula, and is therefore highly risky and should be performed only on the instruction of a doctor.	Internal cardiac massage may be safer if appropriately trained member of staff is available.
Wean Flows to 75% of baseline(ensure above minimum flows). One specialist observes circuit for air entrainment.	To reduce increased risk of air entrainment caused by reduced filling of L side. Flows maintained to reduce risk of clotting circuit
If air is entrained clamp patient of circuit immediately on outlet tubing (post head)	To prevent air embolism.
Troubleshoot and treat cause of arrest	Abnormal Rhythm – defibrillation Electrolyte imbalance Tamponade
Observe circuit and cannulae for clots prior to resumption of full flows.	To prevent emboli

Title: **MANAGEMENT OF AIR IN VAD CIRCUIT**

Rationale: To understand the risks, causes and management of air in the VAD circuit.

Personnel: VAD Specialist & PICU support nurse.
PICU / Cardiac Consultant
On call Perfusionist
Theatre Team

Equipment: Circuit Clamps x 2
Cannulae Patency Pack

VAD Specialist Action:

Action:	**Rationale:**
<u>If Air Identified In The Circuit</u>: • Clamp off patient immediately on tubing pre arterial and venous cannula • Turn the pump off • Initiate emergency ventilation and inotrope settings • Contact on call perfusionist / cardiac surgeon and theatre team • (Give 20u/kg Heparin bolus to patient) **Document total length of clamp off period in specialist evaluation**	Air within the VAD circuit is a clinical emergency. **To prevent an air embolism** **To provide adequate support whilst off ECLS support** **To prime and connect new circuit.** **PUMP HEAD AND CIRCUIT WILL NEED CHANGED IF BYPASS NOT ESTABLISHED WITHIN 5 MINUTES.**

VAD patient transfer checklist

WEIGHT _____ kg PATIENT NAME _____

Personnel Required	VAD Physician	☐
	VAD Specialists x 2	☐
	Perfusionist	☐
	Cardiac Anaesthetist (for cath)	☐
	Nurse Runner	☐
	Senior Transport Fellow	☐
Equipment Required	VAD trolley (with spare console)	☐
	VAD Emergency Trolley	☐
	Portable Monitor	☐
	Oxygen Cylinder > ¾ Full for bagging	☐
	Suitable Bed	☐
	Resus drugs	☐
	Volume (50mls/kg drawn up)	☐
	Blood Products	☐
	ACT Tubes/Actalyke machine	☐

Prior to Move:

1.	Check CT/CATH lab ready for patient	
2.	Check backup console is fully charged and operational (switch on, wait for self check, ensure all battery lights are lit and then switch off)	
3.	Ensure all required staff are present	
4.	Remove all unnecessary equipment from patient.	
5.	Ensure cannulae are securely fixed to skin, tape tube fixing plate to bed.	
6.	Unscrew VAD motor from clamp and place securely on bed.	
7.	Move patient onto portable monitor.	
8.	Switch on O2 cylinder, check level >3/4 full, hand ventilate.	
9.	**Immediately prior to move** unplug main and backup console from mains power. Ensure that all battery lights are lit ie battery is fully charged.	
10.	Battery will give minimum of 60mins at 55rpm / 3litre flows. Higher flows will reduce battery time available.	

On arrival in Cath/CT and on return to unit:

1.	Check CT/CATH lab ready for patient	
2.	Check backup console is fully charged and operational (switch on, wait for self check, ensure all battery lights are lit and then switch off)	
3.	Ensure all required staff are present	
4.	Remove all unnecessary equipment from patient.	
5.	Ensure cannulae are securely fixed to skin, tape tube fixing plate to bed.	
6.	Unscrew VAD motor from clamp and place securely on bed.	
7.	Move patient onto portable monitor.	

VAD wean Checklist

PATIENT NAME _____ **DATE** _____

1.	Wean discussed/documented with ECLS physician & cardiac team • Ventilation optimised (? Bronchoscopy / NO) • Cardiac support optimised (inodilators/pacing etc) • Adequate sedation & paralysis	
2.	Recent ECHO performed to assess cardiac function	
3.	CxR performed & reviewed	
4.	Inotrope / Vasodilator infusing to the patient	
5.	VAD flows weaned as per documented plan • Generally wean during day when all staff present to minimal flows • Aim to decannulate before 12pm • May need increase support (VAD flow) overnight then planned decannulation following day	
6.	Surgeon & theatre team aware of planned trial off and potential for decannulation	
7.	Patient's ABG/VBG/NIRS and vital signs stable on reduced VAD flows	
8.	Centrifugal Circuit ACT 220-240, a bolus may be required to achieve this once flows less than 50% cardiac output (**any deviation** from this must be clearly documented)	
9.	Clamp patient off the VAD circuit **A then V**	

Guideline Index