INTERNATIONAL EDITION 1.0

The PERFUSION CRISIS MANUAL

CPB Cardiopulmonary Bypass
ECMO Extracorporeal Membrane Oxygenation

Paul Sadleir
Steve Same
David Borshoff

CPB PROTOCOLS
SECTION **1** | SECTION **2**
EMERCENCY PROCEDURES
ECMO PROTOCOLS

Before using this manual please visit

leeuwinpress.com.au/pcm-how-to-use

The Perfusion Crisis Manual (The PCM) has been developed as a cognitive aid written in aviation checklist format to reduce cognitive load when managing time-critical events during cardiopulmonary bypass and ECMO. It is the third crisis manual in the Leeuwin Press series and is based on protocols developed at one of Australia's largest private adult cardiothoracic surgery centers. The individual topics have been authored by cardiac anesthesiologists and perfusionists with a collective experience of more than a century.

The protocols assume the availability of a second machine and were written with reference to specific tubing packs (manufactured by Livanova and Medtronic) and the Sorin S5 cardiopulmonary bypass machine. The ECMO protocols are based on the Maquet Cardiohelp system. Use of this crisis manual is specific to our institution and surgical practice so local idiosyncrasies in tubing pack configuration may require minor protocol modification but general principles remain valid.

All protocols have been simulation tested during the development process. As with The Anesthetic Crisis Manual (The ACM) and The Resuscitation Crisis Manual (The RCM), we emphasise it is not a substitute for experience, clinical acumen and regular simulation training, but hope The PCM proves to be a valuable support for those involved in perfusion.

Paul Sadleir
Steve Same
David Borshoff

Dedicated to Dr John Peacock

He was there at the beginning and educated all.

First published 2019

Library of Congress Cataloging-in-Publication Data

The Perfusion Crisis Manual / by Paul Sadleir, Steve Same and David Borshoff

Emergency Medicine--Handbooks, manuals, etc.
Surgery--Complications--Handbooks, manuals, etc.
Medical emergencies--Handbooks, manuals, etc.
615.781

ISBN 978-0-6482702-1-8 Paperback

Designed and printed for Leeuwin Press by Graphic Source, www.graphicsource.com.au

HEART & GREAT VESSELS

MONITORED PARAMETERS

AORTIC DISSECTION

Pragnesh Joshi | Paul Sadleir

1. If dissection occurs during onset of bypass, reduce flow and consider immediate separation if practical.
2. If heart is arrested, reduce flow and blood pressure.
3. Begin cooling in anticipation of circulatory arrest.
4. Confirm by surgical inspection, TOE or epiaortic scan.
5. Consider alternative cannulation sites and LV vent to prevent chamber distension.
6. Use TOE identification of guidewire placement in true lumen.
7. Recannulate, transfer arterial perfusion line and restart bypass.
8. Ensure adequate flow with appropriate line and arterial pressures.
9. Confirm arch vessel perfusion with TOE and cerebral oximetry.
10. Check for competence of the aortic valve, coronary ostia flow and new regional wall motion abnormalities.
11. Review ABGs and monitor lactate.
12. Plan perfusion strategy for surgical correction.

AORTIC DISSECTION

Pragnesh Joshi | Paul Sadleir

Approach early high aortic line pressure as dissection until proven otherwise.

The use of femoral access and retrograde aortic perfusion is associated with higher rates of aortic dissection than central cannulation, although overall rate is 1:1000 cases.

TOE plays a key role in diagnosis, cannula position and assessing both extent of dissection and vessels involved. The true lumen is often smaller and the dissection flap moves towards the false lumen during systole. Flow in the false lumen will usually be of lower velocity and have a delayed peak in comparison to the true lumen. Spontaneous echo contrast, thrombus or *cobwebs* in the false lumen can be helpful diagnostic signs.

Signs

- high line pressure
- systemic hypotension
- poor venous return
- bulging bluish discoloration of ascending aorta
- false lumen or thickening of the aortic wall > 15 mm
- dampened radial artery waveform
- bleeding from aortic needle holes
- facial perfusion asymmetry
- cerebral oximetry desaturation
- myocardial ischaemia (coronary occlusion)
- aortic valve incompetence
- hypoperfusion and acidosis

5 Recannulation sites include femoral artery, distal ascending aorta, axillary artery and ventricular apex.

Prevention

- epiaortic scanning for site selection
- induce hypotension prior to cannulation
- confirm aortic line pulsatility before bypass
- check line pressure with small infusion volume before bypass
- gentle X-clamp manipulation at low flow
- awareness of high risk patients

CHAMBER DISTENSION

Pragnesh Joshi | Paul Sadleir

1. Check vent tubing not inverted in roller pump 27.
2. Reduce flow but maintain SvO_2 > 70%.
3. Do not cross clamp until cause established.
4. Treat arrhythmias or other causes of ineffective ejection.
5. Check venous drainage 07.
6. Confirm complete occlusion of placed X-clamp.
7. Consider pathological patient shunts.
8. Check the venting system.
   - correct cannula position
   - tip patent
   - sucker line patent
   - roller pump not occluded
   - vacuum release valve functioning
9. Consider intermittent manual compression of the heart.

CHAMBER DISTENSION

Pragnesh Joshi | Paul Sadleir

Distension can be detected by inspection, TOE or raised pulmonary artery or central venous pressures.

Causes

- aortic incompetence
- maloccluded X-clamp
- undetected PDA
- anomalous venous return to left heart (including PLSVC to LA)
- poor venous drainage
- exaggerated left-sided bronchial venous return
- non-coronary collateral blood flow

Abnormal shunts from the right heart (ASD or VSD) may cause left ventricular distension if poor venous drainage.

7 Pathological patient shunts include PDA and PLSVC to LA.

Effects

- poor surgical exposure (loss of bloodless field)
- increased myocardial O_2 consumption
- reduced myocardial perfusion
- myocardial rewarming and cardioplegia washout
- stretch injury
- pulmonary oedema or haemorrhage
- steal (reduced effective pump flows)

Normal right-sided venous return
SVC, IVC, azygous, Thebesian and bronchial veins (via azygous), coronary sinus
Incomplete venous drainage can increase venous flow to left side via pulmonary circulation.

Normal left-sided venous return
bronchial veins, non coronary collaterals
Thebesian vein draingage should cease after X-clamp is placed.

Vent position for LV distension
aortic root – only if X-clamped
LV via pulmonary vein or LV apex – all causes
LA – not for aortic incompetence (AI)
LV apex – for aortic incompetence when trans-mitral LV vent not possible (mitral stenosis)

FAILED CARDIOPLEGIA ARRESTS

Ann Ngui | Paul Sadleir

1. Check cardioplegia (CPL) solution composition and expiry date.
2. Systematically complete CPL delivery system checklist.
3. Check for surgical causes of antegrade or retrograde failure.
4. Consider requirement for LV vent if LV distension.
5. Proceed to retrograde CPL if antegrade failure.
6. Confirm flow/pressure in venous coronary system.
7. Consider alternative strategies.
   - direct ostial cardioplegia
   - intermittent X-clamp with induced VF
   - off-pump surgery
8. Consider removing X-clamp to re-establish myocardial perfusion.

FAILED CARDIOPLEGIA ARRESTS

Ann Ngui | Paul Sadleir

2 Cardioplegia delivery system checklist

1. Tubing spike inserted fully into cardioplegia bag.
2. Cardioplegia bag emptying when cardioplegia delivered.
3. Tubing not kinked or clamped.
4. Roller pump occlusions correctly set.
5. Purge lines closed from cardioplegia circuit to reservoir.
6. Blood and cardioplegia mixing in tubing.
7. Cardioplegia line pressure adequate during delivery.
8. Check line configuration on table - 3 way taps set correctly (not vented to reservoir).
9. Cardioplegia delivery ratio correctly set.
10. Correct heat exchanger settings and delivered cardioplegia temperature.

In catastrophic failure of cardioplegia delivery system, consider giving single strength crystalloid cardioplegia via sterile, debubbled IV giving set with pressurised bag from head of table as an emergency measure.

Causes of antegrade failure	
Flacid root	aortic incompetence delivery system failure
Normally distended root	wrong solution, flow or temperature patent LIMA graft non-occlusive X-clamp cardioplegia washout due to poor venous drainage or non-coronary collaterals
Abnormally distended root	aortic dissection involving aortic root cannula, coronary ostia or aortic valve (incompetent)

Causes of retrograde failure	
Low coronary sinus pressure	delivery failure, incorrect 3-way tap setting, leakage around catheter balloon, sinus rupture, malpositioned catheter in right atrium, coronary sinus fistula (unroofed sinus, persistent left SVC)
High coronary sinus pressure:	overinflated catheter balloon, catheter migration into coronary sinus tributary, failure to vent aortic root

6 Surgeon may observe distended cardiac veins or retrograde flow from coronary ostia if aorta open. Flow and distension of the great cardiac vein can be detected with TOE. Migration of coronary sinus cannula into tributaries (great cardiac vein or middle cardiac vein) leads to incomplete protection. TOE can also detect aortic incompetence, coronary artery cardioplegia flow, coronary sinus catheter position, abnormal anatomy and maloccluded X-clamp.

Cardioplegia temperature

Failure of the cardioplegia heat exchanger with 1:1 cold St Thomas' will result in the delivery of cardioplegia at approximately 20°C. Intermittent tepid cardioplegia at this temperature has been used intentionally with no inferiority to cold cardioplegia.

MASSIVE HEMORRHAGE

re-do sternotomy

Mark Schneider | Steve Same

1. Give 100% O_2.
2. Transfuse fluid volume to maintain circulation.
3. Consider cardiogenic shock from lacerated coronary graft.
4. Contact transfusion services and activate MTP.
5. Use vasopressor only to maintain vital organ perfusion.
6. Use maximal anticoagulant flow for cell salvage.
7. In overwhelming blood loss give 400 IU/kg IV heparin and return blood to oxygenator reservoir.
8. Add 400 IU/kg to pump reservoir and start cardiotomy suction if insufficient circulation for heparin redistribution.
9. Recirculate using large-volume recirculation line.
10. Transfuse salvaged blood through peripheral or aortic line.
11. Consider repurposing roller pump vent as second cardiotomy suction.
12. Establish bypass as soon as conditions permit.

MASSIVE HEMORRHAGE

re-do sternotomy

Treatment for hypotension in major blood loss should prioritise volume replacement. Vasopressor should be used judiciously to preserve vital organ perfusion. Rapid establishment of cardiopulmonary bypass will reduce venous pressures and allow efficient return of salvaged blood.

3 The clinical picture may be complicated by inadvertent damage to a previous coronary graft. Failure to respond to fluid therapy or transfusion may be due to co-existing cardiogenic shock.

Anticoagulation

Waiting 300s or longer for adequate heparinisation in normothermic circulatory arrest may result in patient harm. Administer additional heparin directly to reservoir and return blood via central or peripheral lines. A second full-dose of heparin administered to the reservoir will also minimise the effects of blood shed prior to heparin administration and later aspirated to the reservoir, or blood with significant tissue-factor exposure. A higher ACT target is justified.

Recirculating with a large-volume recirculation line prevents blood stasis.

Risk factors for haemorrhage with redo-sternotomy

- adhesions between sternum and anterior cardiac surface
- ascending aorta or bypass grafts close to sternum on CT scan
- RA/RV dilatation (TR, cardiomyopathy)
- previous mediastinitis

In high risk patients lines should be circulated and divided, cell saver ready, and cross matched blood located and cross checked before sternotomy.

MASSIVE HEMORRHAGE

re-do sternotomy

Mark Schneider | Steve Same

1. Confirm diagnosis with TOE and surgical inspection.
2. Determine the anatomical variant.
3. Monitor cerebral venous drainage using cerebral oximetry.
4. Plan cardioplegia strategy to avoid retrograde route.
5. Perform a trial occlusion of PLSVC during bypass if patent left innominate vein present.
6. Cannulate coronary sinus and vent (or snare) PLSVC when innominate vein present.
7. Check for associated congenital heart abnormalities.
8. Beware atypical LSVC drainage into left atrium predisposing to paradoxical embolism, right to left shunt, and LV dilatation.
9. Increase vigilance for potential coronary sinus rupture.

PERSISTENT LEFT SUPERIOR VENA CAVA (PLSVC)

Steve Same | Paul Sadleir

Diagnostic signs

- large coronary sinus (>10mm)
- low retrograde cardioplegia pressure
- flooding of the right atrium despite bicaval cannulation
- visible PLSVC on TOE or surgical Inspection

TOE will confirm by demonstrating agitated saline in coronary sinus (or left atrium) *before* the right atrium after injecting into left brachial vein.

Differential diagnosis

- dilated coronary sinus from raised right atrial pressure (any cause)
- coronary sinus to coronary artery fistula
- partial anomalous pulmonary venous drainage of the left upper lobe (drains left upper lobe pulmonary veins to left innominate vein)
- unroofed coronary sinus
- partial anomalous hepatic venous drainage

2 Anatomical variants

Left subclavian and left internal jugular vein drain into the coronary sinus and RA (90%). Occurs in 0.1-0.3% of the population but is more common in people with other congenital anomalies. 40% have other cardiac defects. *50% have innominate vein draining to right SVC.*

Variant classification

40% - no innominate vein
10% - no right SVC
10% - persistent left SVC drains into left atrium with or without unroofed coronary sinus. If the left SVC drains into the left atrium, it enters the left atrium between the pulmonary veins and atrial appendage. Invariably associated with ASD.

5 Clamp the persistent LSVC during cardiopulmonary bypass only if right SVC is present and joined by an innominate vein to the PLSVC. Otherwise, there will be complete obstruction to venous drainage of the left side of head and arm. Do not clamp left SVC or cannulate coronary sinus if no right SVC present.

6 Venting or snaring the PLSVC prevents flooding from coronary sinus with open right atrium.

Do not insert CVC in *left* IJV.

PERSISTENT VF

post X-clamp removal

Ann Ngui | Steve Same

1. Target myocardial perfusion pressure > 60mmHg and allow adequate reperfusion post X-clamp removal.
2. Check energy, pad position and discharge signal for each DC shock.
3. Consider myocardial ischaemia and use TOE to assist diagnosis.
4. Normalise K^+ and pH.
5. Give magnesium for pre-existing or recent QT prolongation.
6. Confirm venous temperature ≥ 32°C.
7. Reduce flows or vent LV to correct any LV distension.
8. Review pacemaker settings to avoid R-on-T phenomena.
9. If no response to DC shocks commence adjunct therapy.
10. Re-apply X-clamp, induce diastolic arrest with cardioplegia, restart checklist and re-attempt reperfusion after 15min.

PERSISTENT VF

post X-clamp removal

Ann Ngui | Steve Same

Myocardial perfusion pressure = Mean Arterial Pressure – Central Venous Pressure.

DC cardioversion

Limit to a maximum of 6 shocks.
Multiple shocks lower VF threshold and can damage myocardium.
Use 5-10 joules asynchronous with internal paddles.
Use 200J with external pads and inflate lungs to reduce impedance.

3 Causes VF

- myocardial ischaemia
- aortic dissection
- insufficient reperfusion time
- poor protection/inadequate cardioplegia
- ventricular distension
- pacing R on T
- Malignant Hyperthermia (rare)

Myocardial Ischaemia

- intramyocardial or coronary gas
- graft: dissection, compression, kinking, spasm, embolus, clamped
- surgical: valve obstructing coronary ostia, circumflex in MV suture, clamp left on previous mammary graft during re-do

9 Adjunct therapy

IV lignocaine 1.3mg/kg - limited effectiveness
amiodarone 300mg or 5mg/kg slowly - prolongs QT interval
MgSO4 20mmol IV - for polymorphic VT
KCL 20mEq into aortic root directed to aortic valve
esmolol 1mg/kg IV bolus

POOR VENOUS RETURN

Anton Van Niekerk | Steve Same

1. Make sure venous clamp is open.
2. Exclude venous air lock.
3. Inspect venous line for kinks or obstruction.
4. Temporarily reduce flows and add volume to reservoir.
5. Check for low CVP and exclude critical event.
   - aortic dissection
   - anaphylaxis
   - occult blood loss
6. Check positive relief valve is unobstructed.
7. Elevate table and ensure reservoir at lowest safe level.
8. Use TOE or surgeon to check venous cannula position.
9. Consider a second venous cannula.
10. Add vacuum assisted venous drainage.

POOR VENOUS RETURN

Anton Van Niekerk | Steve Same

Signs
low reservoir level, elevated CVP, chamber distension

Effects
reduced flow, chamber distension, poor surgical conditions, cardioplegia washout, poor cerebral perfusion pressure

4 Temporarily reducing flow improves surgical conditions, allows venous drainage to exceed arterial flow, and ensures safe reservoir levels.

Causes based on mean systemic filling pressure (MSFP)	
Reduced MSFP	**Increased MSFP**
reduced blood volume in venous system • aortic dissection • suction to cell salvage system • unrecognised blood loss *left pleural cavity (LIMA), vein harvest site, groin, retroperitoneal space* reduced venous tone • anaphylaxis • vasoactive drugs	kink in venous line cardiac manipulation – lifting/retracting venous cannula malposition or obstruction venous air lock tumour / thrombus line obstruction reduced siphon effect obstructed reservoir pressure relief valve venous cannula size mismatch to patient

Venous air lock source

- entrainment of air around cannulation sites
- cannula migration exposing holes
- lacerated atrium or IVC
- open left heart with ASD or VSD
- loose IV line or CVC connections

Venous cannula malposition usually involves innominate or hepatic veins.

Venous air lock management
To remove venous air lock, reduce flow or suspend CPB while correcting the source of air. Sequentially elevate tubing allowing air to rise before lowering the tubing allowing the blood column to drive air distally to reservoir. Otherwise clamp, disconnect and back-fill from table.

Adequate siphon effect requires appropriate table and reservoir height differential.
27cm height = 20mmHg VAVD

PREMATURE ELECTRICAL ACTIVITY

Ann Ngui | Steve Same

1. Check cardioplegia (CPL) composition and calculated dose.
2. Check delivery ratio with blood CPL or if using Bretschneider ensure no blood given.
3. Check no shunts are open in cardioplegia circuit.
4. Check CPL not recirculated to reservoir via table-line 3-way tap.
5. Confirm CPL given at correct rate, temperature and line pressure.
6. Confirm surgeon detects pressurised aortic root.
7. Use TOE or inspect LV vent effluent to exclude significant aortic incompetence.
8. Confirm X-clamp is properly placed and any previously grafted LIMA clamped.
9. Re-dose CPL once cause identified and corrected.
10. If anterograde effectiveness uncertain, redose through coronary ostia or use retrograde.

PREMATURE ELECTRICAL ACTIVITY

Ann Ngui | Steve Same

2 Bretschneider crystalloid

Cold crystalloid cardioplegia which produces an ischaemic tolerance of 180 min without re-plegia. Also known as histidine-tryptophan-ketoglutarate solution (or Custodial) it produces a hyperpolarised arrest (low Na, low Ca) in *diastole*. Isoelectric arrest is delayed compared with St Thomas' but should occur during the first minute or 500mL of antegrade cardioplegia.

5 Dose

25mL/kg at 300mL/min and 4°C for 6-8 min.
Isoelectric arrest occurs after 500-1000mL.
Aortic administration pressure is at 150mmHg until arrest, then 100mmHg.

For causes of delayed arrest see 03.

Resumption of electrical activity or VF immediately after completion of the dose of cardioplegia is highly suspicious of a maloccluded X-clamp. Bretschneider cardioplegia is very sensitive to washout. Distension of the aortic root or other causes of flow in coronary vessels (surgeon flushing grafts, poor venous drainage) may lead to resumption of activity.

Re-dosing

Half dose at 3 hours. Full dose if mechanical activity present.

Zero-balance ultrafiltration on X-clamp application minimises haemodilution from CPL volume. Severe hyponatraemia (112-120mmol/L) is tolerated as osmolality is normal.

Benign electrical activity

If after an appropriate initial dose there is electrical activity without mechanical contraction, and no evidence of administration error, take no action but monitor closely.

Temporary non-sinister electrical activity may be observed 10-20 min after the induction dose of cardioplegia but generally does not warrant re-dosing unless inadequate delivery, > 5min in duration, or there is associated mechanical activity.

Atrial activity does not warrant re-dosing, but is associated with an increased incidence of supraventricular tachycardia postoperatively. Non-persistent ventricular ectopics during cardiac handling are not an indication for re-dosing.

7 Aortic incompetence predicting antegrade failure

- vena contracta > 3mm
- LV distension on TOE
- crystalloid effluent in LV vent

Aortic root distenstion, aortic root color flow on TOE or bloody effluent in the LV vent suggests maloccluded X-clamp.

FAILURE TO REWARM

Ken Williams | Paul Sadleir

1. Check reservoir inflow and oxygenator outflow temperatures

2. Palpate heater-cooler lines.

3. If temperatures are appropriate, check patient core temperature at an alternative site.

4. If oxygenator or heater-cooler line temperatures are low, systematically work through heater-cooler failure checklist.

5. Consider oxygenator heat exchanger fluid path blockage and clear with flow reversal by swapping inlet and outlet hoses.

6. Check for open circuit recirculation shunts.

7. Consider alternative rewarming strategies.

7. If faulty oxygenator, consider change-out depending on patient oxygenation and clinical context 32.

FAILURE TO REWARM

Ken Williams | Paul Sadleir

Rewarming guidelines

When arterial outlet temp below 30°C, gradient between arterial outlet and venous reservoir inflow should be ≤ 10°C. When above 30°C, gradient should be 4°C or less.

The rewarming rate should not exceed 0.5°C/min.

Rewarming should be limited to a maximum nasopharyngeal or pulmonary artery temp of 37°C to prevent cerebral hyperthermia.

Duration of perfusion at > 37°C is an independent predictor of acute kidney injury.

Rewarming from 20°C when done according to guidelines can take 5min/°C.

3 Heater-cooler failure checklist

- power on, solenoids and motor working
- taps turned correctly
- correct water bath selected
- sufficient fluid level in water bath
- hoses connected to correct outlet
- hoses connected to oxygenator
- kinks or obstructions removed, water flowing
- correct temperature setting
- oxygenator water-path patent

6 Alternative rewarming strategies

Functional heater-cooler unit

Cardioplegia circuit: create shunt through cardioplegia heat exchanger (CHE) by opening recirculation line beyond CHE to reservoir. Flow 500mL/min using only blood limb through CHE at 39°C creating a rewarming circuit from oxygenator to reservoir. Efficiency index of CHE is typically 0.9 (0.4 for oxygenator) so overall heat transfer may approach 50%.

Non-functional heater-cooler unit

Fluid warmer: add an appropriate fluid warming device to an in-circuit recirculation line or line to the patient. A coil of tubing in a water tub can be used to rewarm or cool blood.

Rewarming is required to a temperature of 32°C to allow a stable, paced cardiac rhythm without VF. Consider hypothermia effects on coagulation after separation.

GASEOUS MICROEMBOLI

Paul Sadleir | Steve Same

1. Allow the reservoir level to rise.
2. Turn gas sweep blend to 100% O_2.
3. Reduce pump flow to 2.2 L/m^2/min.
4. Inspect venous line for visible air and inform surgeon.
5. Reduce vacuum-assisted venous drainage.
6. Decrease cardiotomy and vent suction flow rates.
7. Temporarily isolate cardiotomy reservoir from main reservoir allowing aspirated blood to de-bubble.
8. Minimise temperature gradients.
9. Minimise perfusionist interventions and execute each with care.

GASEOUS MICROEMBOLI

Paul Sadleir | Steve Same

Causes	
Surgical	venous line air entrainment • improperly snugged venous or coronary sinus catheters • the presence of an ASD or VSD with an open left heart chamber • vena cava laceration • migration of the venous cannula to expose fenestrations initiation of cardiopulmonary bypass cardiac manipulation
Perfusion	low reservoir levels excess VAVD pressure addition of fluid to reservoir rough handling giving drugs via sampling manifold excess temp gradients obstruction with downstream negative pressure excess cardiotomy or vent suction

Higher reservoir levels reduce vortexing and improve bubble buoyancy. Low levels are associated with carotid microemboli. Higher levels are achieved by reducing flows or adding volume.

100% O_2 decreases cavitation, microemboli formation and aids rapid dissolution.

Rapid rewarming predisposes to endogenous gas formation while rapid cooling promotes outgassing in aortic perfusate.

Perfusionist interventions should be done with caution. De-air manifold syringes, add reservoir fluids slowly and consider patient IV line for larger fluid volumes.

Use the minimum effective vacuum for satisfactory drainage. Vacuum pressures of 5-15mmHg are considered conservative, 15-40 routine, > 40 excessive and > 60 are dangerous. Reduce VAVD when flow rate reduced (to prevent bubble transgression across oxygenator membrane).

Potential effects

- neurocognitive deficit
- blood brain barrier injury
- systemic inflammatory response syndrome (SIRS)
- blood interaction: complement activation, microthrombi production, endothelial cell damage

HIGH AORTIC LINE PRESSURE

Paul Rodoreda | Paul Sadleir

Inform team, reduce flow until cause is identified and respond according to patient systemic blood pressure.

Hypotensive patient

1. Exclude arterial line kink, inadvertent clamping or X-clamp across aortic cannula.
2. Exclude aortic dissection using TOE, epiaortic ultrasound and surgical inspection.
3. Do not proceed with X-clamp, cooling or arresting the heart until resolved.

Normotensive patient

1* Look for signs of malperfusion and check aortic cannula position to exclude great vessel cannulation.
2. Exclude cannula-patient mismatch.
3. Re-zero transducers.
4. Recalculate BSA and target flows.

Hypertensive patient

1. Check depth of anaesthesia.
2. Exclude inadvertent vasopressors.
3. Recalculate BSA and target flows.
4. Confirm appropriate rpm.

HIGH AORTIC LINE PRESSURE

Paul Rodoreda | Paul Sadleir

1* Aortic cannula malposition includes great vessel cannulation, cannula tip against vessel wall and retrograde placement (facing aortic valve). This can result in asymmetrical blood pressures and/or cerebral oximetry, target flow failure and ventricular distension. Selective perfusion of the pressure-monitored limb can cause a high reading.

Left common carotid and innominate arteries can be imaged by TOE using left and right lateral flexion from neutral position in upper oesophagus.

Innominate artery cannulation

- high pulsatile radial artery pressure on right
- lateralised facial blanching with bypass onset
- unilateral facial hyperaemia and petechie with established bypass
- conjunctival oedema
- asymmetrical cerebral oximetry changes

Cannula selection		
Cannula size	**Flow (lpm)**	**Appropriate BSA (m^2)**
15	3	1.4
18	4	1.7
20	5	2.0
24	6	2.4 - 2.7

Always consult manufacture information.
Flow highly dependent on cannula model.
minimum aortic cannula size = 10 x BSA
maximum accepted pressure drop = 100mmHg

Maximum line pressure

Gradient between aortic line pressure and arterial pressure is relevent to prevent turbulence and haemolysis. Above critical velocity of 120-200mL/s haemolysis increases, particularly with turbulent flow.

The LivaNova Inspire 6F oxygenator connections of the main blood line are tested to a pull force of 15N for 15s without separating. Therefore maximum tolerated blood path pressure *before failure* is 750mmHg.

HIGH PROCESSED EEG

Paul Rodoreda | Paul Sadleir

1. Check volatile or TIVA delivery systems.

volatile

- adequate gas sweep, gas tubing connected to oxygenator, oxygenator functioning
- correct vaporiser dose setting and agent levels
- rotameter responds to occlusion test
- adequate agent concentration scavenge gas

TIVA

- correct patient data, syringe drive rate
- infusion starteed, syringe volume decreasing
- syringe tubing visualised and connected to patient
- IV line flushed and patent

2. If delivery failure change the technique (volatile ⇌ TIVA).

3. Inspect EEG sensor for contact and position.

4. Consider falsely elevated readings.

5. Review raw EEG.

6. Give muscle relaxant if high EMG activity.

7. Consider deepening anaesthesia.

HIGH PROCESSED EEG

Paul Rodoreda | Paul Sadleir

1 Agent concentration can be measured by using the anaesthetic machine gas analyser in scavenge line with low suction rate.

Artifacts
High processed EEG reading has good sensitivity for awareness but poor specificity.

4 **False readings**

- skeletal muscle electromyographic activity - inadequate paralysis
- ketamine - paradoxical rise
- high-dose opiates - less suppressed for same anaesthesia depth
- moderate hypothermia - paradoxical rise
- artifacts from electrocautery, roller pumps, forced air warmers, atrial pacing
- seizure activity

5 Inspection of raw EEG may assist in excluding artifact. Presence of burst suppression (alternating bursts of high voltage activity and isoelectric periods) indicates deep anaesthesia and may cause spuriously high readings. Seizure activity (poly-spike and wave) may also result in an abnormal value. Gamma band oscillations (broadband high-frequency, low-amplitude) suggest possibility of consciousness.

Causes of inadequate anaesthesia

- agent delivery failure
- increased requirement (decreased age, amphetamines, rewarming)
- inappropriate dosing

Volatile anaesthetic dose requirement decreases with temperature.
%ET sevoflurane to achieve 0.8 MAC at lower temperatures:

1.6%	37°C
1.0%	28°C
0.5%	20°C

Processed EEG and clinical correlation

90-100	awake
70-90	light to moderate sedation
60-70	superficial anaesthesia
40-60	adequate anaesthesia
0-40	deep anaesthesia

Awareness unlikely with value less than 60.

Awareness is supported by movement and/or attempts to breathe while on bypass.
In suspected awareness follow up using the Brice structured interview:

1. What was the last thing you remembered before you went to sleep?
2. What is the first thing you remember on wakening?
3. Did you dream or have any other experiences whilst you were asleep?
4. What was the worst thing about your operation?

HYPERKALEMIA

Kristine Wardle | David Borshoff

1. Cease or minimise potassium administration.
2. Review differential diagnosis to avoid potential crisis.
3. Check sample for haemolysis and consider repeating levels before treating unless symptomatic ECG changes.
4. Check adequate pump flow.
5. Increase gas sweep flow to target normal $PaCO_2$.
6. Correct acidaemia with 50-100mL 8.4% $NaHCO_3$.
7. Give 6.8-13.6g IV calcium to stabilise cardiac membrane.
8. Redistribute K^+ with IV insulin + glucose therapy.
9. Increase urine flow with diuretics.
10. Consider a low-dose adrenaline infusion for beta effect.
11. Start zero-balance ultrafiltration with potassium-sparing fluid replacement.
12. Monitor treatment with frequent ABGs.

HYPERKALEMIA

Paul Sadleir | Steve Same

Definition	**mild** 5.5 - 6.0mmol/L	**moderate** 6.0 - 6.5mmol/L	**severe** > 6.5mmol/L

2 Differential Diagnosis

Most common causes	Symptomatic of potential crisis
renal insufficiency red blood cell transfusion excessive cardioplegia drugs (ACEI, K^+ sparing diuretics) transient increase post X-clamp removal	haemolysis [23] Malignant Hyperthermia [17] cold agglutinins [22] sickle cell crisis [29] adrenal insufficiency rhabdomyolysis inadequate pump flow, gas sweep flow or both

Elevated K^+ can be symptomatic of sinister underlying etiologies but may be significant when unsuccessful restoration of cardiac activity.

ECG changes
Tall peaked T waves, prolonged PR interval, small or absent P wave, widening QRS, sinusoidal QRS, ectopics/VF, asystole

Minimise potassium administration by avoiding K^+ containing fluids, using single strength CPL, minimising transfusions and avoiding old red blood cells.

7 Cardiac membrane stabilisation
Calcium is available as the chloride or gluconate. 10mL of calcium chloride gives 6.8g calcium. 30mL calcium gluconate gives 6.78g calcium.

8 Redistribution of K^+
10 units actrapid IV with 25-50g 50% glucose IV
50-100mL 8.4% $NaHCO_3$ for associated metabolic acidosis

9 K^+ elimination
10-20mg frusemide
100mL 20% mannitol (20g)
zero-balance haemofiltration

Zero-balance haemofiltration
Ultrafiltrate should be replaced with a potassium-free replacement fluid such as albumin. Isotonic bicarbonate (150mL of 8.4% $NaHCO_3$ in 850mL sterile water) has the advantage of a low chloride concentration which prevents hyperchloremic acidosis.

HYPERLACTATEMIA

Kristine Wardle | David Borshoff

1. Check pump DO_2 > 270mL/min/m^2 and SvO_2 > 70%.
   - adequate PaO_2
   - appropriate effective flow (pump output - shunt flow)
   - haematocrit > 24%

2. Check oxygen delivery to tissues.
   - perfusion pressure > 50mmHg
   - arterial and venous cannulas in correct position
   - $PaCO_2$ upper limit of normal

3. Reduce oxygen demand.
   - adequate anaesthetic depth
   - paralysis
   - hypothermia

4. Exclude regional ischaemia of limb, bowel or liver.

5. Ensure euglycaemia.

6. Minimise vasopressors and consider adding methylene blue to preserve splanchnic blood flow.

7. Expedite weaning from bypass.

HYPERLACTATEMIA

Paul Sadleir | Steve Same

Severe Hyperlactataemia > 4mmol/L
Severe hyperlactataemia during bypass is associated with increased morbidity and mortality.

Causes

During cardiopulmonary bypass, severe hyperlactataemia usually develops as a consequence of inadequate global or regional oxygen delivery (Type A). May persist despite adequate global DO_2 due to bypass induced SIRS response (microcirculatory dysfunction and accelerated metabolism), beta agonists (adrenaline), massive transfusion or regional ischaemia.

1 Oxygen delivery (DO_2)

DO_2 = pump flow x [(Hb g/L x 1.34 x SpO_2 x 0.01) + (0.003 x PaO_2 mmHg)]
= 2.4 L/min/m2 x [(80g/L x 1.34 x 100 x 0.01) + (0.003 x 760mmHg)]
= 263mL/min/m2

Inadequate if DO_2 < 250mL/min/m2 or SvO_2 < 70%

TYPE A – tissue hypoxia from inadequate oxygen delivery

- **Tissue hypoperfusion:** circulatory failure, shock, regional hypoperfusion
- **Reduced O_2 delivery/utilisation:** hypoxaemia, anaemia, metabolic stress, CO poisoning

TYPE B – absence of tissue hypoxia

- **B1:** diseases including hepatic and renal failure (decreased clearance)
- **B2:** drugs or toxins including catecholamines, metformin
- **B3:** inborn errors of metabolism

Cerebral oximetry can be used as a proxy monitor of tissue perfusion.
Regional malperfusion may be improved with high/normal $PaCO_2$.

Cannula position

Arterial cannula malposition (eg. innominate artery cannulation) usually presents with high line pressures, but should be considered and excluded by surgical inspection.

Venous cannula malposition usually presents with raised CVP and poor venous return, but CVP may be normal with poor IVC or hepatic vein drainage. Malposition should be excluded by TOE to exclude hepatic vein cannulation or venous cannula advancement.

Perfusion pressure = arterial blood pressure – central venous pressure.

HYPOTENSION

Anton Van Niekerk | Steve Same

1. Use **aortic line pressure** to assist diagnostic pathway.

high

- Reduce flows.
- Consider separating from bypass until problem resolved.
- Check arterial line not clamped or kinked.
- Use TOE or epiaortic ultrasound to inspect for dissection 01.
- Review aortic cannula position.

normal/low

- Check for pump malfunction or inadequate flows.
- Eliminate large-volume circuit shunts.
- Use TOE and inspect for patient shunts.
- Review causes of low SVR.

2. Check transducers are positioned correctly, calibrated and arterial line aspirates freely.

3. Exclude external compression of arterial line arm.

4. Consider momentary flow alteration to confirm appropriate BP changes.

5. Verify the low pressure with aortic palpation.

6. Check SvO_2 > 70% and treat anaemia to maintain adequate DO_2.

7. Titrate vasoconstrictor.

HYPOTENSION

Anton Van Niekerk | Steve Same

Definition
Unexpected low blood pressure for apparent pump flow (e.g. < 50mmHg with 2.4L/m^2)

When commencing cardiopulmonary bypass, ensure adequate aortic line flows can be achieved before allowing free drainage via the venous line. Maintaining the patient intravascular volume until adequate perfusion conditions can be confirmed increase the likelihood the patient can be rapidly weaned if high line pressure or unexpected hypotension is encountered.

Aortic cannula malposition
The cannula may be abutting the aortic wall or the tip may be in the innominate, left common carotid or left subclavian artery.

Signs confirming underperfusion include falls in cerebral oximetry, venous desaturation and abnormal blood gases (hypercarbia, acidosis). A flaccid aorta, cannula malposition on TOE and facial color/asymmetry also support diagnosis. Variations in pressure reading with altered flow helps exclude artefact.

1 Causes of low SVR
- deep anaesthesia
- vasodilators
- anaphylaxis 19
- adrenal insufficiency
- transfusion reaction 24
- sepsis
- MH 17

Pathological patient shunts
- patent ductus arteriosis (PA trunk dilatation, LA distension)
- aortopulmonary window, hemitruncus arteriosus
- aortic incompetence
- dialysis A-V fistulas

CPB circuit shunts may occur when high-volume recirculation lines are open.

LOW CEREBRAL OXIMETRY

Steve Same | Paul Sadleir

1. Check arterial line color and exclude failure to oxygenate [32].

2. Correct inadequate cerebral blood flow.
   - treat hypotension and elevated CVP
   - normalise pump flow
   - check for open high-volume circuit shunt
   - increase $PaCO_2$ (40-45mmHg)
   - check aortic cannula position
   - consider raised intracranial pressure

3. Check haematocrit.

4. Optimise cerebral venous drainage.
   - neutral head position
   - no external neck compression
   - correct venous cannula position
   - adequate circuit venous drainage [07]

5. Avoid increased oxygen utilisation.
   - adequate paralysis
   - adequate anaesthesia
   - appropriate rewarming rate (5min/°C)
   - treat seizures, MH, thyroid storm (rare)

6. Consider differential hypoxia.

LOW CEREBRAL OXIMETRY

Steve Same | Paul Sadleir

Causes	
unknown (most common)	aortic dissection
inadequate systemic flows	oxygenator failure
excess CO_2 removal	arterial embolism
cannula malposition	increased O_2 consumption (MH, seizures)
carotid artery stenosis	large frontal sinus (artifact)

Cerebral oximetry monitors tissue perfusion of the frontal cortex and scalp. Evidence of malperfusion is likely to reflect global malperfusion and supporting evidence should be sought (lactic acidosis, oliguria).

Cannula position can be assessed by surgeon or TOE . Inspect face for evidence of malperfusion.

Increasing anaesthetic depth, inducing hypothermia and rewarming less aggressively to a lower target temperature (35°C) can all reduce O_2 consumption.

If suspect raised ICP, give 100-200ml 20% mannitol, position patient in reverse Trendelenberg and optimise cerebral perfusion with CPP of 50-70mmHg and $PaCO_2$ 35-40mmHg.

Intervention threshold

Intervene if cerebral oxymetry saturation < 50%, > 20% decrease from baseline or asymmetrical change. Values of < 35 – 40% for greater than 10 min are associated with cognitive decline and renal impairment.

Treatment

If intervention threshold reached and does not correct with troubleshooting, particularly if SvO_2 low, consider supranormal targets:

- arterial pressure 60-80mmHg
- Hb > 70g/dL
- increase pump flow
- $PaCO_2$ 45-50mmHg

Expedite separation from cardiopulmonary bypass.

6 Differential hypoxia

If aortic cannulation is via femoral artery with retrograde perfusion and aorta is not X-clamped, cardiac activity without lung ventilation may result in ejection of deoxygenated blood into the proximal aorta, coronary arteries and supra-aortic vessels. The result may be upper body cyanosis. Manage by optimising venous drainage and increasing pump flow. Ventilate lungs with 100% O_2 if tolerated by surgeon.

MALIGNANT HYPERTHERMIA

Kristine Wardle | David Borshoff

1. Declare emergency and designate roles.
2. Stop rewarming, remove volatile, turn sweep gas to 100% O_2 and increase gas flow rate 2-3 x normal.
3. Maintain anaesthesia with TIVA.
4. Actively cool to < 38°C.
5. Start mixing dantrolene and keep giving until crisis is over.
6. Abandon or curtail surgery if practical.
7. Do not separate from bypass until responding to dantrolene.
8. Treat asscociated hyperkalaemia 13, acidosis and arrhythmias.
9. Maintain urine output > 2mL/kg/hr.
10. Monitor temp, blood chemistry, coagulation and ABGs.

MALIGNANT HYPERTHERMIA

Kristine Wardle | David Borshoff

MH during cardiac surgery typically presents while warming on bypass or after separation.

Attempts to decontaminate gas supply and oxygenator after volatile anaesthetic administration are unnecessary.

Signs on bypass

- hypermetabolism (↓ SvO_2 or ↑ $PaCO_2$ may be only indication)
- metabolic acidaemia with increased lactate
- haemodynamic instability
- hyperthermia
- elevated CK and Mb
- dark urine

5 Give 2.5mg/kg dantrolene IV and repeat every 10-15min until crisis is over.
Dantrolene requires dedicated staff to prepare. Mix each 20mg vial with 60mL sterile water. Mobilise additional dantrolene as each dose may require 8-10 vials. Alternatively use **Ryanodex** (lyophilised formulation of dantrolene) 250mg reconstituted with 5mL of water. Dose may need to be repeated (1mg/kg 6 hourly).

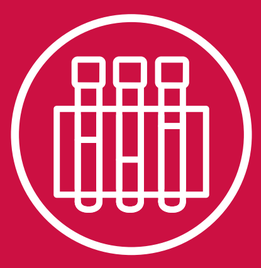
Dantrolene may interact with calcium channel blockers leading to hyperkalaemia associated cardiovascular collapse.

Cardiopulmonary bypass protects from arrhythmias, haemodynamic instability and low cardiac output. It also provides efficient temperature homeostasis and allows haemofiltration (effective for myoglobin clearance).

Patients often have periods of hypotension during and immediately after bypass which can delay diagnosis.

Diuresis
Maintain urine output at > 2mL/kg/hr (IV hydration, frusemide).
Each vile of dantrolene contains 3g of mannitol.
Ryanodex contains 125mg of mannitol.
Haemofiltration advised.

Arrhythmia treatment
amiodarone 3-4mg/kg over 30 min
lignocain 1-2mg/kg IV
metoprolol 1-2mg IV prn
esmolol infusion



BLOOD COMPONENTS

EQUIPMENT FAILURE

ANAPHYLAXIS

Paul Rodereda | Paul Sadleir

1. Notify surgical team and cease potential triggers.

2. Maintain reservoir volume by reducing pump flow and aggressive fluid resuscitation (typically 50ml/kg crystalloid).

3. Give adrenaline 100mcg boluses up to 2mg and start infusion.

4. If response inadequate start vasopressin and/or methylene blue.

5. Consider supranormal pump flows as a temporary measure to maintain arterial pressure.

6. Discuss with team aborting or limiting surgery.

7. If complicated by myocardial pump failure consider VA ECMO until resolved.

8. Request mast cell tryptase, monitor Hct, ABGs and coagulation.

ANAPHYLAXIS

Paul Rodereda | Paul Sadleir

Signs of anaphylaxis during bypass

Flow: reduced arterial blood pressure and perfusion line pressure
Venous return: significant reduction, falling reservoir level
Haematocrit: haemoconcentration may increase
ABG: increased lactate, hypokalaemia

Classification

Grade 1. skin signs and/or fever reaction
Grade 2. measurable non-life-threatening hypotension, respiratory signs
Grade 3. life-threatening shock and/or bronchospasm
Grade 4. cardiac and/or respiratory arrest

1 Common triggers

Timing can provide clues to the most likely trigger.
Post induction: muscle relaxant, antibiotics
Delayed post induction: antibiotics, chlorhexidine impregnated CVC, TXA, heparin
Bypass: gelofusine, fibrin glue
Post bypass: protamine

3 Adrenaline Infusion

3mg in 50ml saline started at 5ml/hr and titrated
Consider second line therapy if at 15ml/hr response not adequate.

Non-adrenergic agents (2nd line)

vasopressin	2U bolus: 0.03-0.06 IU/min infusion
methylene blue	2mg/kg bolus over 20 min: 1-2mg/kg/hr infusion

An elevated Hct may indicate inadequate resuscitation in the context of anaphylaxis.

6 Abandoning surgery

If anaphylaxis occurs prior to surgical procedure, a decision to abandon or proceed is made. Factors include the nature of the surgical pathology (physiological reserve may be improved by correction of critical left main stenosis or critical aortic stenosis), effect of anaphylaxis on ability to assess outcome (SAM after mitral valve repair is exacerbated by anaphylaxis) and patient comorbidities.

Most cases of anaphylaxis are brief in duration (20-60 min) and a period of support on cardiopulmonary bypass will be adequate prior to separation. VA ECMO may be considered as a supportive therapy in protracted anaphylaxis complicated by cardiac pump failure.

Avoid re-dosing potential triggers commonly used during CPB including muscle relaxants, gelofusine and chlorhexidine impregnated catheters.

BLEEDING POST BYPASS

Paul Sadlier | Steve Same

1. Check ACT and consider additional protamine dose.
2. Start cell salvage using high anticoagulant flow rate.
3. Send blood for INR, APTT, fibrinogen, Hb and ROTEM.
4. Notify blood bank to ensure availability of blood components.
5. Warm fluids, warm room and start surface warming devices.
6. Correct any acidaemia or hypocalcaemia.
7. Maintain SBP 80-100mmHg.
8. Guide therapy with test results but treat empirically if clinically suspect coagulopathy.
9. Aim for
   - Hb > 8g/dL
   - INR < 1.5
   - APTT < 40s
   - fibrinogen > 2mg/dL
   - platelet count > 100 x 10^9/L
10. Consider DIC if low fibrinogen and platelet count.
11. Reheparinise and suction to cardiotomy reservoir if loss becomes overwhelming.
12. Consider off-label use of recombinant Factor VIIa (50mcg/kg).

BLEEDING POST BYPASS

Paul Sadlier | Steve Same

Causes

- **Platelet dysfunction:** activation of platelets by contact with foreign surfaces, air/blood interface, mediastinum or pleura.
- **Haemodilution:** pump prime, cardioplegia, cell salvage, crystalloid/colloid volume expansion or fractionated products can dilute clotting factors or platelets.
- **Consumption:** activation of inflammatory and coagulation cascades during cardiopulmonary bypass, defibrination of mediastinal or pleural blood, DIC.
- **DOACs:** reverse dabigatran with idarucizumab: reverse rivaroxaban or apixaban with Andexanet Alfa, PCC and/or rFVIIa.

Acidosis and hypothermia < 34°C exacerbates coagulopathy.

When checking for heparin excess use ACT, heparinase controlled ACT and rotem analysis.

8 ROTEM Protocol

Abnormality	Treatment
FIBTEM A10 < 10mm	cryoprecipitate or fibconc
HEPTEM MCF < 50mm	platelets
EXTEM CT > 100s or HEPTEM CT > 240s	FFP or PCC
LYSIS A30 > 15%	tranexamic acid 1000mg

DIC syndrome is characterised by intravascular activation of coagulation without localisation. Consumption of platelets and clotting factors may result in bleeding or may present as catastrophic intravascular thrombosis.

DIC Diagnosis (supported by a score ≥ 5)	
associated underlying disorder	N=0, Y=2
platelet count	> 100 = 0, < 100 = 1, < 50 = 2
FDPs	none = 0, mod = 2, strong up = 3
PT	elevated by 3-6 sec = 1, > 6 sec = 2
fibrinogen	< 1g/L = 1

DIC Management

Correct underlying cause (sepsis, ischaemia, prolonged bleeding)

If primary feature is *bleeding*:

- confirm adequate protamine dose (rarely need > 0.6mg/mg total heparin)
- first line treatment is platelets and FFP (PCC does not contain PC, AT or TFPI)
- cryoprecipitate or purified fibrinogen concentrate if hypofibrinogenaemic
- tranexamic acid if evidence of hyperfibrinolysis

If primary feature is *thrombosis* (intravascular clot), consider heparin therapy.

Consider antithrombin III concentrate 3000 IU.

CLOTTING IN CIRCUIT

Warren Pavey | Paul Sadleir

1. Assess extent and significance of clot.

2. Wean from bypass if clinically appropriate and conditions allow.

3. If continuing bypass give additional 10,000 IU heparin, 500mL 4% albumin and check ACT.

4. Review all possible causes including cold agglutinins and HITTS.

5. Haemodilute in hypercoagulable states and use alternative anticoagulant if HITTS suspected.

6. Suction cardiotomy blood to cell salvage.

7. Start rapid cooling and 100% O_2 in preparation for oxygenator or full circuit change-out.

8. Change-out oxygenator [40] if primary problem or proceed to circuit change-out if clot more extensive.

10. Monitor and treat any associated haemolysis [23].

11. Plan post-op anticoagulation if embolism suspected.

CLOTTING IN CIRCUIT

Warren Pavey | Paul Sadleir

4 Causes

- failure to give or insufficient anticoagulant
- heparin resistance
- cardiotomy suctioning after protamine or rVIIa
- HITTS [26], cold agglutinins [22]
- echinocytosis
- procoagulant predisposition

Procoagulant conditions
thrombocytosis, polycythaemia, hyperfibrinogenaemia, deficiencies of protein C, protein S, factor V Leiden or antithrombin III, antiphospholipid antibody syndrome

Hypercoagulable states may require isovolemic haemodilution to a Hct < 42% and Plt < $400x10^9$/L.

Clues to cause

Inadequate anticoagulation: clots in low flow areas (right atrium, outside coronary sinus catheter) or surgical field
Cold agglutinins: clots in cold regions of circuit (oxygenator heat-exchange, CPL circuit)
HITTS: heparin given 5-14 days prior, associated thrombocytopenia

Indications for immediate oxygenator change-out

- oxygenator failure (PaO_2 < 100mmHg or SvO_2 < 50%)
- pre-membrane pressure > 500mmHg or transmembrane pressure > 300mmHg
- inability to maintain adequate pump flow
- systemic thrombus embolization risk

Albumin is useful as an agent to haemodilute and has also been used to coat bypass circuits to prevent adsorption of fibrinogen and platelet aggregation. It has been shown to reverse thrombosis secondary to echinocytes forming in alkalemic conditions.

High transmembrane pressure gradients may resolve with adding albumin and rewarming.

8 Full circuit change-out

If second machine not set up, delegate and follow emergency set up protocol [38] [39].

Move second machine with primed circuit near patient (both machines oblique), clamp lines and cut out old circuit at table, connect new circuit and resume cardiopulmonary bypass. Drain blood from clotted circuit, wash and return RBCs to patient.

COLD AGGLUTININS

Mark Schneider | Steve Same

1. Cease cooling and begin rewarming to 34-37°C.

2. Check for oxygenator failure [32].

3. Disconnect cardioplegia (CPL) line at table and flush with warm blood or tepid crystalloid CPL to clear.

4. If CPL line obstructed, change-out or abandon and switch to tepid crystalloid CPL given by pressurised, de-bubbled IV line.

5. Defer DHCA or consider higher arrest temperatures [45].

6. Use crystalloid-only or warm blood CPL, or avoid completely by using off-pump technique at 34-37°C.

7. Consider bicaval cannulation with snares to reduce warm blood and cold cardioplegia mixing, with suction to waste.

8. Send blood for thermal amplitude and titre.

9. Anticipate and treat haemolysis.

10. Use retrograde CPL prior to separation to wash out precipitates.

COLD AGGLUTININS

Mark Schneider | Steve Same

Cold agglutinins are autoantibodies (usually IgM) active at low temperature against red cell antigens. Red cell agglutination with complement attachment occurs with activation and haemolysis on rewarming. Agglutination relevant for perfusion is unlikely in absence of symptomatic disease with the exception of deep hypothermic cardiopulmonary bypass.

Causes

Primary disease
Secondary: infections (mycoplasma, IMN), lymphoproliferative disorders.

Signs

- haemolysis/anaemia
- cardioplegia obstruction
- intracoronary thrombosis
- vaso-occlusive phenomena
- oxygenator failure
- acrocyanosis
- red cell clumping on blood smears

Cold agglutinin titre is the highest dilution with agglutination seen at a specified temperature.
Thermal amplitude (TA) is the highest temperature at which agglutination is seen in vitro.
Symptomatic cold agglutinins usually have TA ≥ 30°C and a titre of 1:128 at 22°C.

Prevention when detected preoperatively

Consult haematology and discuss with team modification of hypothermic bypass to avoid temperatures lower than thermal amplitude.
Anti-B cell antibody (rituximab), IV immunoglobulin or plasmapheresis can all be used to reduce levels of IgM and IgG before proceeding.
Use crystalloid only or warm blood CPL continuously or at shorter intervals.

Cryoglobulinaemia

Different entity to cold agglutinins but results in temperature related precipitation of cryoglobulins. Both benign (infection related) and pathological (associated with specific diseases) variants. Precipitates activate complement and produces hyperviscosity syndrome leading to vasculitis, infarcts and SIRS-like syndrome.

HEMOLYSIS

Warren Pavey | Paul Sadleir

1. Repeat ACT, check transmembrane pressure and inspect circuit for abnormal noise, clot or fibrin strands.

2. Inspect cardioplegia circuit for evidence of cold agglutinins [22].

3. Check arterial line pressure and scan venous and arterial lines for kinks or flow restriction.

4. Assess oxygenator heat exchanger water path for leak [33].

5. Consider haemolytic transfusion reaction or red cell abnormalities [24] [29].

6. Minimise cardiotomy suction and vent output, and ensure roller pumps are not over-occluded.

7. Do not exceed ideal calculated pump flow rate.

8. Consider other measures to minimise gaseous microemboli.

9. Treat haemolysis effects and start renal protective strategies.

10. Expedite separation from bypass.

HEMOLYSIS

Warren Pavey | Paul Sadleir

Diagnosis
Tea-colored urine and plasma Hb >10mg/dL.

Causes

- prolonged bypass
- excess cardiotomy suction
- main line obstruction
- venous line air entrainment
- hereditary red cell abn [29]
- transfusion old RBCs
- transfusion reaction [24]
- cold agglutinins [22]
- heat exchanger leak [33]
- gaseous microemboli [10]
- excessive VAVD

High blood velocities (> 100cm/s) and air-blood interfacing (by cavitation, entrainment or suction) results in red cell damage. Cardiotomy suction is a major source and damage may be reduced by storing blood in the cardiotomy reservoir for as long as possible to reduce gaseous microemboli.

8 Minimising gaseous microemboli
Check no air entrainment to venous line.
Minimise vacuum-assisted venous drainage (40mmHg).
Allow blood to sit in cardiotomy reservoir before adding to main reservoir.
Consider autotransfusor for salvaged blood (removes 90% PFHb).

9 Effects

- anaemia, hyperkalaemia
- microvascular constriction
- platelet activation with thrombi formation
- increased blood viscosity (with associated organ damage)
- acute kidney injury (renal tubule toxicity)

Early postoperative hyperbilirubinaemia is a poor prognostic sign.

Renal protection strategies

- haptoglobin (6000 IU)
- urinary alkalinisation (500-1000mL of 1.26% bicarbonate)
- diuresis (20mg frusemide, 0.5mL/kg mannitol 20%)

Ultrafiltration with conventional filters will not remove free haemoglobin.

HEMOLYTIC TRANSFUSION REACTION

Ann Ngui | David Borshoff

1. Stop transfusion and replace fluid infusion tubing.
2. Check label and recipient identity.
3. Treat hypotension by increasing pump flows and titrating vasoactive agents.
4. Monitor with ABG (Hb, K^+ and metHb), unconjugated bilirubin, LDH and haptoglobin.
5. Promote urine output using IV fluids and diuretic therapy.
6. Start urinary alkalinisation.
7. Inform transfusion services and plan strategy for subsequent red cell transfusions and potential coagulopathy.
8. Return all products to blood bank and take fresh blood and urine samples for analysis.
9. Consider alternative diagnoses for haemolysis [23].

HEMOLYTIC TRANSFUSION REACTION

Ann Ngui | David Borshoff

Acute haemolytic transfusion reaction is a consequence of incompatible red blood cell transfusion. Binding of complement-fixing antibodies to donor RBCs leads to RBC lysis, activation of complement and coagulation pathways, and cytokine release. It may rarely be caused by non-ABO incompatibility. Recipient RBC lysis may occur after transfusion of ABO-incompatible fresh frozen plasma or its components.

Diagnostic signs during bypass

- hypotension
- pulmonary oedema
- difficultly weaning
- bleeding
- hyperkalaemia
- elevated LDH
- anaemia
- methaemoglobinaemia
- haemoglobinuria

Falling haemoglobin level, a rise in serum LDH and haemoglobinuria strongly suggest haemolysis. This can be confirmed by a direct antibody test.

Coagulopathy with consumption of fibrinogen and elevation of D-dimers suggests the development of DIC.

5 Drug dose

frusemide 0.5-1mg/kg IV
mannitol 20% 0.5-1g/kg IV
hydrocortisone 100mg IV

TRALI (transfusion related acute lung injury) occurs within 6 hours of transfusion. Protein-rich oedema fluid causes bilateral lung infiltrates in the absence of raised left atrial pressures, with a $PaO_2/FiO_2 \leq 300$.

6 Urinary alkalinisation

1-2mL/kg 8.4% $NaHCO_3$ over 5 min *then* 250-1000mL/hr of isotonic 1.26% $NaHCO_3$ (150mL 8.4% $NaHCO_3$ in 850mL sterile water = 1.26%).

HEPARIN RESISTANCE

Warren Pavey | Steve Same

1. Confirm the correct drug and dose was given.

2. Aspirate from administration line to confirm patency and intravascular position.

3. Give another 100mg of fresh heparin into functioning IV line.

5. If ACT still low, give an additional 100mg heparin and consider an alternative machine using in-date cuvettes when repeating.

6. Review all causes of resistance and treat accordingly.

7. Consider giving 500-1000 IU ATIII concentrate and additional heparin up to a total dose of 1000mg.

8. Proceed with a lower target ACT if acceptable for procedure but monitor at increased frequency (20 minutely).

9. Consider using 1mg/kg bivalirudin bolus followed by an infusion at 2.5mg/kg/hr and monitor ecarin clotting time.

HEPARIN RESISTANCE

Warren Pavey | Steve Same

Definition
Inadequate ACT after standard heparin dose.
ACT < 400s after 300 IU/kg
ACT < 480s after 400 IU/kg *or*
Heparin sensitivity index < 1.3

Heparin Sensitivity Index is a measure of heparin resistance and can be calculated:

$$\text{Heparin Sensitivity Index} = \frac{\text{post heparin ACT} - \text{baseline ACT}}{\text{heparin loading dose (IU)}}$$

6 Common causes of heparin resistance

Delivery issue: wrong drug/dose, extravascular, not flushed, misdirected to isolated cardiotomy reservoir
Antithrombin III deficiency: hereditary, pre-op heparin or enoxaparin, decreased production (liver disease) or increased loss (DIC, endocarditis, DVT/PE, ECMO, VAD)
Increased heparin binding: platelets, medications, factor VIII, fibrinogen
Recent protamine

With pre-op heparin/enoxaparin therapy, the thrombin/ATIII complex is cleared rapidly by the RES and ATIII levels drop 5-7% per day.

Management
ATIII deficiency responds to exogenous supplementation. Heparin binding will respond to increased heparin dosing.

ACT level and cardiopulmonary bypass
Target ACT depends on procedure, circuit and ACT measurement device. The presence of blood stasis (reservoir), pericardial shed blood salvage to circuit, valvular procedures or the lack of circuit coating increases the required ACT level.

An ACT of 300s prevents visible clots in circuit and may be adequate for commencement of cardiopulmonary bypass in an emergent situation when utilizing a heparin-bonded circuit.

ACTs of 400s or 480s are common targets.

Ecarin clotting time
Used to target bivalirudin concentration of 15mcg/mL.
If ecarin clotting time unavailable target ACT > 600s.

HITTS

Heparin-Induced Thrombotic Thrombocytopenia Syndrome

Kristine Wardle | Paul Sadleir

1. Stop heparin, defer procedure and come off bypass if practical.
2. Urgently source alternative anticoagulants.
3. Scan for clot within circuit [21], avoid stasis, maintain circuit flow and give volume.
4. Monitor for oxygenator failure [32].
5. Monitor lactate for evidence of organ malperfusion.
6. Do not give platelets.
7. Use sodium citrate as anticoagulant for cell salvage.
8. Send blood for platelet factor 4/polyanion antibody assay and platelet activation test.
9. Prepare for significant postbypass bleeding.
10. Avoid postoperative warfarin until platelet count recovered.

HITTS

Heparin-Induced Thrombotic Thrombocytopenia Syndrome

Kristine Wardle | Paul Sadleir

Heparin-Induced Thrombotic Thrombocytopenia Syndrome (HITTS) is a prothrombotic disorder caused by IgG mediated antibodies to complexes of platelet factor 4 (PF4) and heparin.

Diagnosis
The detection of HITTS antibodies plus one of the following.

- unexplained drop in platelet count by 30-50%
- venous or arterial thrombosis
- skin lesions at heparin injection site
- anaphylactoid reactions

The antibodies bind to the PF4-heparin complexes on the platelet surface inducing activation. The activated platelets increase the release and surface expression of PF4, creating a positive feedback loop in which further release of PF4 promotes further platelet activation.

Warfarin can induce a paradoxical, hypercoagulable state usually within 3 to 10 days of therapy initiation, associated with inadequate heparin overlap, and thought to be due to an imbalance between anticoagulant and procoagulant pathways. The anticoagulants protein C and protein S have a shorter half-life than other vitamin K–dependent factors (II, IX, and X), resulting in a deficiency of both proteins early in treatment. This increases the chance of thrombosis and subsequent skin necrosis.

2 Alternative anticoagulants

Direct thrombin inhibitor (half-life 25 min)
Bivalirudin 1mg/kg IV, followed by 2.5mg/kg/hr. Use additional boluses of 0.5-1mg/kg to maintain ACT 2.5 x baseline or > 600s or APTT ~ 200s.
Cease the infusion 15 min prior to planned separation.
Use ecarin clotting times to monitor to target blood concentration 15mcg/mL.

Factor Xa inhibitor (half-life 18-24 hours)
Danaparoid 7500 units with 1500 units in prime to achieve a level of at least 1 unit/mL during CPB. ACT does not correlate with anti-Xa activity.

Antiplatelet agent plus heparin
Tirofiban 10mcg/kg bolus with infusion 0.15mcg/kg/min followed by heparin 400 IU/kg (*Restore* protocol).

Minimise stagnant blood in circuit and cardiotomy suction, and continually flush cardioplegia circuit.

MASSIVE AIR EMBOLISM

Steve Same | David Borshoff

1. Stop bypass, clamp lines and alert team.
2. Place head down.
3. Give 100% O_2 and set heater-cooler to 20°C.
4. Identify and correct source of embolus, then reprime circuit.
5. Suction on vents once correct positioning in race verified.
6. Use air volume to guide treatment.
   - < **10-20mL** aspirate heart/aorta, re-start bypass, initial carotid compression
   - > **10-20mL** retrograde cerebral perfusion
7. Re-establish strategic hypothermic bypass for 45 min.
8. Maintain 100% O_2 and gently rewarm to 35.5°C.
9. Use needle venting and retrograde cardioplegia to de-air coronaries.
10. Consider post-operative hyperbaric therapy.

MASSIVE AIR EMBOLISM

Steve Same | David Borshoff

4 Sources of embolism

- reversed tubing (inversion) in roller pump or reversed roller pump direction
- obstructed oxygenator gas outlet (unlikely with modern oxygenator design)
- inattention to reservoir level resulting in emptying
- unexpected resumption of cardiac activity with open left cardiac chamber
- open right heart chamber with ASD or VSD
- inadequate de-airing of heart prior to cross-clamp removal

Factors supporting retrograde cerebral perfusion

- air aspirated from aorta
- embolic volume exceeds 10-20mL
- RWMAs or air seen on TOE
- surgeon visualises air in coronaries
- rapid fall in cerebral oximetry reading

6 Retrograde cerebral perfusion

1. Remove aortic cannula and cannulate SVC above Azygous – snare or clamp below.
2. Check arterial line free of air. Fill with saline if required.
3. Perfuse 1-2 L/min for CVP 20-25mmHg for 1-3 min.
4. When aortic opening looks free of air start intermittent carotid artery occlusion for a total of 2 min to flush vertebral arterial system.
5. Recannulate aorta.
6. Re-establish hypothermic bypass at 25°C for 45 min.

7 Strategic hypothermic bypass measures: 100% O_2 , hypothermia, haemodilution to Hct 25%, higher flows (2.6L/min/m2) and moderate hypertension (70mmHg).

These measures reduce $CMRO_2$, increase absorption rate and decrease cerebral emboli residence time.

10 Additional treatments

Prophylactic anticonvulsants: leviteracitam 500mg bd.
Hyperbaric therapy: 6 ATM up to 48 hours post insult if neurological deficit and/or macroscopic air on CT head.
Pharmaceutical neuroprotection has little supporting evidence.

Estimating embolism volume based on tubing size

3/16″	15mL/m	3/8″	65mL/m
1/4″	30mL/m	1/2″	115mL/m

PROTAMINE REACTION

Steve Same | David Borshoff

1. Cease protamine infusion.

2. Give 100% O_2.

3. Titrate vasoconstrictor and consider IV fluid bolus.

4. If poor response use TOE to help determine reaction type.

5. Use specific treatment for reaction type:

Type 3 reaction

- dobutamine 250mg/50mL at 5mL/hr
- vasopressin 20 IU/50mL at 1-10mL/hr
- inhaled nitric oxide at 20-40ppm
- consider return to bypass

Non-Type 3 reaction

- Type 2a/2b: treat as for anaphylaxis 19
- Type 2c: treat for pulmonary oedema using PEEP
- consider return to bypass or ECMO

6. Prepare to reheparinise and re-institute CPB if resistant to initial therapy.

7. Re-attempt slow IV protamine (250mg over 30 min) for Type 1 and Type 3 reactions or use alternatives.

PROTAMINE REACTION

Paul Rodoreda | Paul Sadleir

Horrow classification of protamine reactions	
Type 1	Non-IgE dependent mast cell histamine release producing a mild, transient reduction in systemic vascular resistance. It is related to both dose and speed of giving (< 3min). Can usually continue at a much slower rate after stabilisation.
Type 2a	IgE-mediated anaphylaxis [19]
Type 2b	Anaphylaxis-like syndrome but in the absence of IgE-antibodies to protamine. May involve anti-protamine IgG antibodies, or the generation of anaphylatoxins by protamine-heparin complex activation of the complement cascade.
Type 2c	Delayed non-cardiogenic pulmonary oedema (potentially florid).
Type 3	Pulmonary vasoconstrictive syndrome mediated by protamine-heparin complex generation of thromboxane A2. Characterised by intense pulmonary vasoconstriction, right ventricular failure and dilatation, systemic hypotension and decreased left atrial pressures.

Post-reaction management

- mast cell tryptase at 1 and 4 hours
- allergy center referral

6 In severe reactions rapid re-establishment of bypass may be required [43].

7 Protamine alternatives

- spontaneous reversal of heparin
- rPF4 (5mg/kg) or give platelets
- hexadimethrine bromide

SICKLE CELL DISEASE

+ RBC abnormalities

Paul Sadleir | Ross Baker

1. Use 100% O_2 sweep gas and keep SvO_2 > 80%.
2. Target Hct 20-30% and HbS < 10%.
3. Consider exchange transfusion if HbS > 30%.
4. Establish bypass flow gradually using oxygenated prime.
5. Once on bypass drain venous blood to autologous transfusion bag and replace with donor RBCs + crystalloid to achieve targets.
6. Maintain MAP 60-70mmHg.
7. Avoid acidosis, hypothermia < 32°C and dehydration.
8. Avoid cold blood cardioplegia or consider off-pump surgery.
9. Do not use cell salvage and minimise cardiotomy suction.
10. Keep peripheries warm and minimise low-flow periods.

SICKLE CELL DISEASE

+ RBC abnormalities

Paul Sadleir | Ross Baker

Preparation

If cardiac surgery urgent, a pre-op HbS < 30% should be targeted using transfusion of donor RBCs. Aim for minimal fasting, good hydration and anxiolysis. Although hydroxyurea increases the concentration of HbF (which can decrease incidence and severity of sickle episodes), the long lead time for effect limits its use in the urgent scenario.

Goals of therapy in sickle cell disease are to keep HbS levels < 30% (preferably below 10%) and haemodilution to maintain Hct at 20-30%. Use exchange transfusion once on bypass to target HbS level.

Homozygous sickling occurs at PaO_2< 40mmHg or SpO_2 < 85%.

> Hypothermia, low flow, hypoperfusion, dehydration, acidosis and low SpO_2 all predispose to sickling. SvO_2 should be kept > 80%.

Gradual increase in flow and minimal suction reduce trauma to already fragile RBCs.

Sickle Cell Crisis

Should a crisis develop:

- exchange transfuse to HbS < 5%
- treat haemolysis 23
- use appropriate factors for dilutional coagulopathy
- maintain ACT > 600s to prevent thrombosis
- treat vaso-occlusive complications
- rewarm
- maintain urine flow at 0.5-1ml/kg/hr

Other red cell abnormalities

The following principles apply to other red cell abnormalities such as **G6PD** and **Hereditary Spherocytosis**.

- minimise cardiopulmonary bypass time
- use off pump technique if practical
- minimise red cell trauma
- maintain urine flow
- avoid hypothermia, hyperglycaemia and acidosis

G6PD deficiency specifically requires avoidance of volatiles, diazepam, methylene blue, vitamin K, paracetamol and lignocaine. A leukodepletion filter may reduce the incidence of lung injury after cardiopulmonary bypass.

Hereditary Spherocytosis may require pre-emptive splenectomy.

Always prepare for haemolysis during the perioperative period.

CIRCUIT LEAK

Mark Schneider | Steve Same

1. Turn flow down and locate the source of leak.

2. Troubleshoot while continuing bypass or if conditions allow separate from bypass to resolve the issue.

3. Add volume to reservoir ≥ 500ml above level sensor.

4. Assess leak rate and treat according to component affected.

   low-pressure component
   - *reservoir defect:* pack with bone wax or change-out
   - *split pump head inflow tubing,* clamp and replace

   high-pressure component
   - *pump head or oxygenator:* change-out
   - *aortic line:* replace

5. Check cardioplegia circuit and change out if necessary.

6. Return to normal flow bypass.

CIRCUIT LEAK

Mark Schneider | Steve Same

1 Common sources of leak

- misthreaded, loose or unattached caps, luerlocks or tubing
- split tubing
- cracked reservoir/filter/pump head
- cardioplegia circuit

Low pressure leaks carry the risk of air entrainment.
High pressure leaks risk exsanguination.

2 Hard plastic components can sometimes be temporarily sealed using bone wax until off bypass. Ensure sterile caps, connectors and alcohol wipes immediately at hand.

If the patient is warm, ventilated and the heart is beating, separating from bypass will instantly resolve the issue.

3 Adding volume to 500ml above level sensor provides a buffer against losses from ongoing leak or component change-out.

ELECTRICAL FAILURE

Ken Williams | Paul Sadleir

1. If main pump failure notify team and call for help.

2. Clamp arterial and venous lines.

3. If conditions permit, manually ventilate and hand crank to separate from bypass.

4. Look for immediately reversible causes
   - CPB console switch off
   - AC plug disengaged or switched off
   - AC power supply failure

5. Start manual systemic perfusion using hand crank if issue not immediately corrected.

6. Move AC wall plug to different power source.

7. Minimise battery load to conserve function.

8. Locate alternative light source to monitor reservoir level.

9. Re-establish essential monitoring with battery-operated devices and aneroid manometer.

10. If electrical *supply* failure activate internal disaster response.

11. Discuss abandoning or shortening procedure with team.

12. Consider a portable generator to run heater-cooler.

ELECTRICAL FAILURE

Ken Williams | Paul Sadleir

Electrical failure may be due to CPB console fault or failure of supply to the CPB machine wall socket. Wall socket failure requires failure of metropolitan power supply (white outlets), back-up generator failure (red outlets) and uninterrupted power supply (UPS) failure (blue outlets). UPS is battery back-up and will have limited duration.

5 Manual perfusion

- Unclamp arterial and venous lines.
- Requires 2 operators, one to crank and one to monitor venous return.
- Operate pole-mounted hand-crank at 60-100rpm.
- Maximum 15 min per operator to prevent exhaustion.
- If intra-arterial pressure monitoring not available, intermittently palpate aorta to confirm efficacy of hand-cranking.
- If functioning SvO_2 monitor, crank to maintain saturations over 70%.

7 CPB console battery

Typical battery is 17 amp hours and may be expected to provide 20min at 400W or 90min at 160W. Typical power consumption of arterial pump, sucker, vents and light is 400W.

Minimise the load on the console battery based on typical values.

- 3-panel console 45W, 6-panel console 90W
- 150mm roller-pump 160W, 85mm roller-pump 80W
- centrifugal pump and control panel 84W

Consider hand-cranking suckers early to save power but beware reversed rotation and massive air embolism.

A battery powered light should be available for reservoir level and emergency responses. Hand cranks should be available for each roller-pump head.

FAILURE TO OXYGENATE

Mark Schneider | Steve Same

Low aortic line pO_2

1. Turn blender to 100% O_2, confirm flow and no CO_2 added.
2. Confirm gas supply is securely connected to oxygenator.
3. Check vaporiser is seated with filling port closed.
4. Check transmembrane pressure gradient.
5. Inspect oxygenator/circuit for clots and add heparin if ACT low [21].
6. Inspect cardioplegia circuit for cold agglutinins [22].
7. Trial 10L/min gas flow for 10s to clear any condensation.
8. Switch to alternative gas supply (O_2 cylinder with regulator).
9. If PaO_2 remains < 100mmHg or SvO_2 < 50% change out oxygenator [40].

Normal aortic line pO_2 / low SvO_2

1. Recalculate target flow and confirm current flow is adequate.
2. Exclude any high-volume shunts in circuit or patient.
3. Check ABG to exclude artifact.
4. If anemic increase Hct to 26%.
5. Prevent shivering and reduce O_2 requirements.
6. Consider lower perfusate temp or reduce speed of rewarming.
7. Close all in-circuit shunts and increase flow to 2.8L/m2/min.
8. Consider Malignant Hyperthermia [17].

FAILURE TO OXYGENATE

Mark Schneider | Steve Same

If aortic line pO_2 is low there is either failure to supply oxygen to the oxygenator or failure of the oxygenator.

If aortic line pO_2 is normal but SvO_2 is low, there is failure of oxygen delivery to the tissues or increased O_2 consumption.

O_2 tissue delivery is impaired in anaemia, cyanide poisoning, methaemoglobinaemia. It can also result from an inappropriate low flow or reduced effective flow from either circuit or patient shunts.

O_2 consumption is increased in sepsis, malignant hyperthermia, thyroid storm.

Supportive evidence for oxygenator failure

- high transmembrane pressure gradient
- clots in oxygenator or circuit
- precipitates in cardioplegia circuit
- blood or plasma leak into gas line from oxygenator
- persistent hypoxaemia despite alternative O_2 supply

4 Indications for oxygenator change-out

- arterial blood pO_2 < 100mmHg
- transmembrane pressure gradient > 250mmHg
- pre-membrane pressure > 500mmHg
- failure to rewarm despite multiple attempts 09
- water to blood fluid leak 33

O_2 flow is confirmed by spinning rotameter or digital readout.
Artifact in SvO_2 reading can be due metHb or methylene blue administration.
Lower perfusate temperature reduces oxygen demand.

HEAT EXCHANGER LEAK

Ken Williams | Paul Sadleir

1. Inform team and turn heater-cooler off.
2. Clamp or turn off heater-cooler hoses.
3. Place in head down position.
4. Move bubble detector to post-oxygenator line.
5. Consider coming off bypass and oxygenator change-out if
   - macrobubbles
   - significant water absorption or haemolysis
   - significant blood loss
6. Treat consequences of haemolysis.
7. Send blood and exchange fluid for culture and start antibiotics.
8. Plan alternative rewarming strategy.
9. Report incident and save circuit for analysis by manufacturer.

HEAT EXCHANGER LEAK

Ken Williams | Paul Sadleir

Oxygenator design favours a blood-path to heater-cooler (water) path leak. Modern heater-coolers circulate fluid by drawing fluid to the unit (subatmospheric) rather than pushing fluid (positive pressure). Nonetheless, a fluid path leak may present as an unexplained increase in circuit volume, hyponatraemia and haemolysis. There is likely to be a potential for air to also cross into the oxygenator in this situation. Urgent oxygenator change-out is indicated.

6 Potential leak consequences

- unexplained increase in circuit volume
- hyponatraemia, hypokalaemia
- anaemia
- haematuria/urine discoloration
- air embolism
- unexplained acidosis
- blood stained fluid in heater-cooler lines
- microbial contamination
- inability to rewarm

Anaemia, hyperkalaemia, hypernatraemia and urine discoloration (tea colored) are all attributable to haemolysis or dilution.

Heater-cooler hoses are turned off or clamped rather than disconnected as a fluid-filled interface is less likely to result in air embolism.

8 If the patient is hypothermic the cardioplegia circuit can be adapted as an alternative rewarming strategy [09].

Leak testing prior to priming circuit
Circulate through oxygenator heat exchanger for 10 min and inspect circuit for fluid leakage. The process should be repeated for the cardioplegia heat exchanger.

A described alternative is to connect a ½″ dead-end tube to the heat exchanger outlet. A manometer adapted to ½″ tubing is then attached to the inlet side and pressurised to 250mmHg for 30 seconds. There should be no observed pressure drop.

LVAD HYPOTENSION

Anton Van Niekerk | Paul Sadleir

1. Lie patient flat and confirm BP with doppler and manual BP.
2. Check controller power light is on and delegate assistant to contact local VAD coordinator to engage system monitor.
3. Plug in power module to wall power while troubleshooting and check all connections, power cables and driveline.
4. Assess rhythm and impeller function by auscultation.
5. If pump function not detectable replace controller with back up and check all connections again.
6. Review controller alarm indicators for pump speed, power, flow and pulsatility index to help direct therapy.
7. If low flow give a 250mL fluid bolus.
8. Assess cardiac and circulatory status using TTE.
   - volume
   - aortic regurgitation
   - pericardial effusion or tamponade
   - inflow cannula/outflow graft obstruction
   - suction event

LVAD HYPOTENSION

Anton Van Niekerk | Paul Sadleir

Causes of LVAD hypotension	
Pump failure	**Circulatory failure**
pump speed set too high obstruction of pump or cannula • compression or obstruction of inflow cannula or outflow graft • thrombosis • suction event	hypovolaemia right ventricular failure arrhythmias aortic regurgitation pericardial effusion / tamponade cardiac arrest

Using a manual BP cuff and doppler the first sound heard is the MAP – normal range is 65-90mmHg non-pulsatile (90-110 pulsatile, depending on device).

The local VAD coordinator should be contacted to engage system monitor if its available.

The controller regulates pump speed and provides information about speed, flow, pulsatility index, and LVAD power.

Pulsatility index reflects cardiac output contribution by heart compared to LVAD. Changes in pulsatility index can indicate obstruction of the inflow cannula, a decrease in volume status, arrhythmias, or increased pulmonary artery pressure suggestive of right heart failure.

6 LVAD parameter abnormalities	
High power **Low-pulsatility index** **Flucuating pump speed**	pump thrombosis or hypotension
High power **High pulsatility index**	fluid overload
Low power **Low pulsatility index** **Unchanging speed**	hypertension or inflow/outflow obstruction
Low power **Normal or high pulsatility index**	suction event

SUDDEN ARTERIAL PUMP FAILURE

Warren Pavey | Paul Sadleir

1. Check main pump RPM and if line clamps activated.

2. Check display for alarm triggered pump stop.

3. Reverse any inadvertent switch off.
   - bypass machine
   - drive console

4. Turn off, reseat and restart decoupled magnetic pump head.

5. Troubleshoot main pump drive.
   - check connections back from mechanical drive
   - restart with minimal power if suspect internal overload
   - check roller pumps not over occluded or jammed

6. If no immediate correctable cause found:
   - clamp arterial and venous lines
   - declare emergency
   - hand-crank to manually perfuse
   - ventilate and separate from bypass if conditions permit

6. Resume bypass with backup drive console.

SUDDEN ARTERIAL PUMP FAILURE

Warren Pavey | Paul Sadleir

Presentation	
Complete Power Loss	electrical supply or battery failure
Patient	falling line pressure falling systemic BP
Machine & Circuit	rise in reservoir level dark venous blood reduced flow rate/RPM decrease in cerebral oximetry
Wet End	unusual noise from pump blood leak visible clot
Magnetic Drive	noise or smoke from drive

2 Alarm triggered pump stop

- level sensor
- bubble indicator
- line monitor positive pressure
- vacuum assisted negative pressure
- roller pump shield lift

Back up drive console

Change out magnetic drive head and control unit *or* use a second machine alongside.

- place pump head into magnetic drive
- use level sensor from new machine
- attach pressure cut-out sensors

If no immediate cause identified, clamping arterial and venous lines can prevent exsanguination or embolism.

Consider inserting arterial tubing into spare 150mm roller pump but *beware operating an occlusive pump without a pressure triggered stop-link*.

EMERGENCY PROCEDURES

Contents

36 CPB PROTOCOLS

SECTION ONE | SECTION TWO

EMERGENCY PROCEDURES

ECMO PROTOCOLS

AORTIC LINE

change

Warren Pavey | Steve Same

1. Turn flow off. Clamp arterial and venous lines.

2. Clamp either side of leak and disinfect.

3. Cut out affected segment.

4. Rejoin with ⅜ - ⅜" Luer lock connector.

5. Unclamp pump side and use Luer lock to aspirate from segment or attach wide bore purge line to reservoir.

6. Once debubbled, clamp purge line.

7. Unclamp patient side and re-establish bypass.

8. Cable-tie connections.

PUMP HEAD

inflow tubing change

Warren Pavey | Steve Same

1. Turn flow off. Clamp arterial and venous lines.

2. Clamp out, disinfect and cut out tubing defect.

3. Rejoin with ⅜" connector directly or in a tube segment.

4. Unclamp reservoir side and manipulate air back to reservoir.

5. Reprime pump head 42.

6. Unclamp pump head side and recirculate until debubbled.

7. Unclamp venous and arterial lines and re-establish bypass.

EMERGENCY PUMP SET UP STAGE 1

Paul Sadleir | Steve Same

1. Open correct circuit pack, tighten all connections and caps.
2. Lock reservoir and oxygenator on reservoir stand.
3. Complete the four critical attachments (FLOP).
4. Start CO_2 gas priming of circuit.
5. Insert cardiotomy suction tubing in roller-pump race and attach to reservoir.
6. Clamp reservoir outlet and oxygenator cardioplegia outlet.
7. Add heparinised prime to reservoir, unclamp outlet, prime pump head, de-air and attach to centrifugal drive.
8. Turn on bypass machine and increase flow to de-air oxygenator and A-V loop.
9. Turn CO_2 off and turn oxygen on.
10. Prime arterial line pressure monitor and attach to transducer.
11. Pass lines to operating table and connect cardiotomy sucker to table suction tubing.
12. Perform the five critical checks before commencing CPB.

EMERGENCY PUMP SET UP STAGE 1

Paul Sadleir | Steve Same

The aim is to rapidly establish bypass and assumes an event equivalent to a cardiac arrest or a deteriorating patient. Establishment of cardiopulmonary bypass is critical to maintain organ perfusion and will reduce myocardial oxygen consumption by reducing wall tension. Subsequent pump set up for administration of cardioplegia and cardiac venting can follow.

3 **Four critical attachments (FLOP)**

- **F**low probe
- **L**evel sensor
- **O**xygen tubing attached to oxygenator
- **P**ressure monitor (circuit arterial line)

6 Clamping the cardioplegia outlet on the oxygenator allows deferral of attaching and priming the cardioplegia circuit to minimise time to establishing extracorporeal flows. If cardioplegia is needed urgently, cold crystalloid cardioplegia can be administered through an IV giving set with pressure bag.

7 To hasten process consider bypassing the non-integrated arterial filter if present.

12 **Five critical checks prior to starting CPB**

- level and bubble sensors on
- oxygen flowing and CO_2 off
- de-aired oxygenator and A-V loop
- ACT > 400s
- recirculation lines clamped

EMERGENCY PUMP SET UP STAGE 2

Paul Sadleir | Steve Same

1. Attach temperature probes, connect and start heater-cooler.
2. Correctly orientate vent tubing in roller pump race.
3. Attach LV and aortic root vents to reservoir and table lines.
4. Attach cardioplegia (CPL) circuit to clamped blood limb of oxygenator, connect CPL purge line to reservoir and open.
5. Attach plasmalyte to crystalloid CPL limb, prime beyond mixing connection with the blood limb and then clamp.
6. Unclamp blood limb of oxygenator and prime CPL circuit via purge line.
7. Insert both blood and crystalloid CPL limbs into roller pump race and occlude.
8. Replace plasmalyte with cardioplegia solution.
9. Prime and attach CPL pressure monitor.
10. Attach heater-cooler hoses and temperature probe to CPL heat exchanger.
11. Attach CPL circuit to table lines and recirculate.
12. Perform 5 critical checks before X-clamp.

EMERGENCY PUMP SET UP STAGE 2

Paul Sadleir | Steve Same

These are steps to be taken in the urgent case immediately after bypass is established but before placement of X-clamp.

2 Care must be taken to avoid avoid inverting and risk massive air embolus.

5 Priming cardioplegia circuit

Depending on the filters present in the crystalloid cardioplegia limb, plasmalyte priming may be possible using gravity. Alternatively, applying bag pressure to overcome the resistance created by fine particle filters may be required.

Bubble traps should be inverted to de-air. Once the crystalloid limb has been primed and clamped, both limbs should be inserted into the roller pump, occlusion checked, all proximal clamps removed and the circuit primed by running the CPL roller pump forward. A second stage of de-airing will occur when cardioplegia solution is recirculated.

12 Five critical checks prior to X-clamp

- all clamps removed and purge lines closed
- de-aired cardioplegia circuit
- pressure monitor primed and functioning
- CPL heat exchanger hoses connected, set and functioning
- correct cardioplegia solution

OXYGENATOR (OXGR) CHANGE-OUT

Preparation

Mark Schneider | Steve Same

1. Call for assistance.
2. Rewarm to separate from bypass or cool to 25°C.
3. Gather equipment and place towel under failed OXGR.
4. Place *new* OXGR near failed unit and confirm circuit lines reach.
5. Do not move failed OXGR.
6. Attach *new* OXGR purge line and sampling manifold line to reservoir and open.
7. If *new* OXGR has short tubing to inlet and outlet, insert ⅜" connectors.
8. Leave cardioplegia and oxygen tubing attached to failed OXGR.
9. Disinfect planned tubing cuts.
10. Add extra 500mL to reservoir and set sweep gas to 100% O_2.
11. Perform final orientation check of *new* OXGR.
12. Proceed to replacement 41.

OXYGENATOR (OXGR) CHANGE-OUT

Preparation

Mark Schneider | Steve Same

2 Ideally, rewarming to allow separation from bypass is the preferred option. If the clinical context prohibits, cooling to 25°C is the best alternative for change-out.

This is a two person procedure.

3 Equipment

1. IV pole with OXGR holder attached
2. replacement OXGR, either custom pack or cut from *new* circuit, complete with sampling manifold line and oxygenator purge line
3. two ⅜-⅜" tubing connectors (for pre- and post-membrane circuit tubing unless exposed tubing barbs present on new OXGR)
4. two ¼-¼" connectors (for OXGR cardioplegia line and OXGR recirculation line)
5. 8 cable ties and a cable-tie tensioner
6. 8 extra clamps
7. heavy sterile scissors
8. alcohol wipes
9. towel

Temperature of 25°C will provide 14 min of *safe* circulatory arrest. OXGR change procedure will require 3 min.

Positioning replacement OXGR

No attempt should be made to remove the failed OXGR as this wastes time and adds complexity to the procedure. Leaving the failed OXGR in place provides a reference for the correct connection of lines and also acts as a line holder as they are sequentially transferred.

11 The *new* OXGR should be positioned next to the failed OXGR and **orientated to ensure correct blood flow direction.** The easiest and most secure way is to attach the *new* OXGR to an IV pole on wheels via a spare holder clamp. This can then be positioned at an appropriate height and moved into a position to check pre and post-membrane lines will reach. If a spare OXGR holder is not available, a satisfactory alternative is to bed the *new* OXGR into towels on the floor next to the failed OXGR.

OXYGENATOR (OXGR) CHANGE-OUT

Replacement

Mark Schneider | Steve Same

1. Announce: *ready to separate from bypass.*
2. Turn off pump. Clamp arterial and venous lines.
3. Double clamp pre and post-membrane circuit from failed OXGR close to unit with at least 3cm between clamps.
4. Cut between clamps close to OXGR end.
5. Connect circuit to *new* OXGR inlet and outlet using ⅜" connectors.
6. If high-volume recirculation line directly from failed OXGR, transfer to *new* OXGR and open.
7. Unclamp *new* OXGR pre-membrane line allowing gravity to fill.
8. De-air post-membrane arterial line.
9. When *new* OXGR filling slows, turn pump on and increase flow to de-bubble.
10. Move gas supply tubing and cardioplegia line to *new* OXGR.
11. Prime first few inches of cardioplegia line and reclamp.
12. Perform 4 final checks and re-establish bypass.
13. Unclamp cardioplegia line, recirculate and de-bubble.
14. Transfer sampling manifold, temperature probe, heater-cooler hoses and gas scavenging line.

OXYGENATOR (OXGR) CHANGE-OUT

Replacement

Mark Schneider | Steve Same

8 The arterial line is de-aired by lowering and using gravity, tilting the OXGR to assist.

12 **Final checks prior to re-establishing bypass**

- oxygenator and arterial line debubbled
- recirculation lines re-clamped
- O_2 gas supply attached
- level and bubble alarms on
- sufficient volume in reservoir

Once transfers and positioning are complete, cable-tie all hoses.

PUMP HEAD REPRIME

air in pump head

Mark Schneider | Steve Same

1. Inform team and turn pump off. Clamp arterial and venous lines.
2. Identify and correct cause of deprime.
3. Add 500-1000mL volume to reservoir.
4. Take pump head out of magnetic drive and float any air back to the reservoir.
5. Open dependent vent *distal* to pump head allowing forward flow and the movement of remaining air out.
6. After re-priming, close vent and reinsert pump head into drive.
7. Open circuit recirculation shunt, start pump and recirculate.
8. Once de-bubbled, slow pump, close recirculation shunt and confirm level and bubble sensors active.
9. Confirm sufficient reservoir volume.
10. Unclamp arterial and venous lines and re-establish bypass.

PUMP HEAD REPRIME

air in pump head

Mark Schneider | Steve Same

Air/foam can be displaced from the pump head with this technique within 30 seconds. The entire repriming and de-bubbling process can be completed within 90 seconds.

2 Causes of pump deprime

- emptying of reservoir
- vortexing
- reservoir tilt or displacement
- excessive vent suction
- circuit breach between reservoir and pump

5 Venting air

In order to vent air beyond the pump head there must be an open vent allowing flow from the reservoir through the pump head to a blood salvage bag at a lower height. Open to the pre-membrane pressure transducing line, AP transducing line or cardioplegia blood limb.

Blood drained into the blood salvage bag can be returned to the reservoir after the incident is resolved.

RAPID RE-ESTABLISHMENT OF BYPASS

Warren Pavey | Paul Sadleir

1. Use 4mg/kg heparin to fully reheparinise patient.

2. Add 100mg heparin to reservoir.

3. Re-attach level, bubble and flow sensors.

4. Attach heater-cooler to oxygenator and CPL circuit.

5. Manage lines according to current status.

6. Check the ACT.

7. Confirm there is no clamp on the CPL blood limb.

8. Perform the critical pre-CPB checks.

RAPID RE-ESTABLISHMENT OF BYPASS

Warren Pavey | Paul Sadleir

5	Action according to line status
Primed, on table	Reattach to new arterial / venous cannulas and go on bypass.
On table, venous line unprimed	Confirm venous line clamped (close to reservoir) and refill from table end.
On table, arterial *and* venous lines unprimed	Attach arterial to venous with ⅜″ to ½″ connector. Add volume to reservoir and recirculate until bubble free. Clamp, cut and connect to appropriate cannula.
Off table, still attached to pump, oxygenator not drained	Open new heart lung pack and cut out AV loop close to oxygenator and reservoir using sterile scissors. Drain old venous line back to reservoir, ensure pump is off and then allow arterial line level to drop back to reservoir level. Clamp and cut out used AV lines, connect new AV loop and pass to table. Cut out the prebypass filter to prevent red cell damage. Ensure adequate reservoir volume, connect the cut ends of the AV lines, recirculate and then connect to appropriate cannula.
Lines unsterile or oxygenator drained	Rapidly clear the pump. Replace with pre-assembled second pump if available. If not, go to Emergency Pump Setup 38 39. Re-establish bypass.

Disconnected sensors
level and bubble sensors
flow sensor
temperature monitor

6* In emergencies waiting for the ACT result should not delay bypass if an appropriate heparin dose has been successfully administered.

8 Pre-bypass checks

- sensors on
- oxygen flowing and attached to oxygenator
- oxygenator and AV loop de-aired
- *ACT > 400s

RESERVOIR CHANGE-OUT

Steve Same | Paul Sadleir

1. Cool to 25°C if leak rate permits.
2. Have equipment and assistants ready.
3. Position *new* reservoir on IV stand next to the old at appropriate height above ground.
4. Clamp *new* reservoir outlet tubing and place connector for pump head inlet.
5. Transfer IV filling line from old reservoir to rapid filling port of the *new* and add 500mL of prime.
6. Turn off vents and transfer from old to *new* reservoir.
7. Turn off purge and sampling lines.
8. Minimise old reservoir level by filling patient and draining remaining volume into collection bag.
9. Announce: *ready to separate from bypass.*
10. Turn pump off. Clamp arterial and venous lines.
11. Disinfect, double clamp and cut pump head inflow close to old reservoir and connect to *new* outlet.
12. Cut clamped venous line and connect to *new* reservoir.
13. Perform final reservoir check and re-establish bypass.
14. Connect purge lines and sampling manifold to *new* reservoir.

RESERVOIR CHANGE-OUT

Steve Same | Paul Sadleir

25°C allows 14 min of *safe* circulatory arrest.

Clear instructions, appropriately skilled assistants and good equipment preparation are essential.

Equipment

1. IV pole with reservoir holder attached
2. replacement reservoir (custom pack or cut from new circuit)
3. ⅜" - ⅜" connector
4. 5 extra clamps
5. heavy sterile scissors
6. alcohol wipes

Securing replacement reservoir

Removing defective reservoir is difficult and the best solution may be to position the new reservoir immediately adjacent to the old. Security is critical. A spare emergency reservoir holder should always be available and attached to an IV pole on wheels, allowing a replacement reservoir to be safely manoeuvred and secured.

Make sure all clamps and appropriate connectors are ready and in place.

4 When clamping the *new* reservoir outlet tubing, always leave a reasonable length to connect to the pump head inlet.

13 **Final reservoir checks**

- sensors on
- sufficient reservoir volume
- vents connected
- recirculation lines closed

UNEXPECTED CIRCULATORY ARREST

Paul Sadleir | Steve Same

1. Anticipate safe arrest duration based on nasopharyngeal temp.
2. Treat anaemia to improve tissue oxygen stores pre-arrest.
3. Consider selective cerebral perfusion (CP) at 500-1000mL/min.
4. Monitor antegrade CP with right radial pressure and target 20-40mmHg.
5. Monitor retrograde CP with SVC pressure < 20mmHg.
6. Use cerebral oximetry to detect cerebral malperfusion.
7. Regularly call out arrest duration to assist decision-making.
8. Perfuse intermittently with 2 min of full pump flow every 20 min at 18°C or every 10min at 25°C if procedure allows.
9. Consider a period of strategic hypothermic bypass [27].

UNEXPECTED CIRCULATORY ARREST

Paul Sadleir | Steve Same

Assumed safe duration of circulatory arrest		
Temp °C (esophageal)	**CMR (% of baseline)**	**Duration (min)**
15	16	31
20	24	21
25	37	14
30	56	9
37	100	5

The spinal cord has a lower metabolic rate (1/4 brain) allowing 20 min ischaemic time at 37°C or 50 min at 32°C. Antegrade cerebral perfusion results in blood flow only to the proximal spinal cord.

3 Selective cerebral perfusion

Antegrade cerebral perfusion (ACP)

ACP during circulatory arrest allows comparable outcomes at moderate hypothermia as deep hypothermia without ACP. Ischaemic threshold in pigs is 6mL/kg/min.

- Cardioplegia circuit heater-cooler set to 25°C.
- Recirculate circuit with cold blood only prior to commencing.
- Monitor efficacy of cerebral perfusion with bilateral frontal cerebral oximetry and right radial arterial line.
- ACP at 8-12mL/kg/min for right radial pressure of 20-40mmHg.
- Sweep gas 100% O_2 and flow rate guided by in-line ABG.
- Venous line must be unobstructed.

Retrograde cerebral perfusion (RCP)

- Cardioplegia circuit heater-cooler set to 25°C.
- Recirculate circuit with cold blood only prior to commencing.
- Cannulate SVC with cardioplegia cannula or place within pre-existing SVC cannula.
- Clamp the IVC and consider upper limb tourniquets.
- Internal jugular venous pressure should not exceed 25mmHg.
- Check for air in cannula/line prior to commencing.
- Commence RCP at 500mL/min.
- Sweep gas 100% O_2 and flow rate guided by in-line ABG.

6 Cerebral oximetry

Cerebral oximetry may be useful to detect selective CP malperfusion (cannula migration, lack of collateral circulation) or intolerance to circulatory arrest duration. Animal studies suggest increased time from oximetry plateau to reperfusion is associated with increased neurological deficit. Safe arrest duration is influenced by haematocrit and temperature. A suggested minimum oximetry saturation is 45%.

ECMO
Contents

ECMO
AIR IN CIRCUIT

Paul Sadleir | David Borshoff

1. Clamp access and return lines.

2. Stop pump.

3. Place head down and give 100% O_2.

4. If venous air use left lateral tilt and aspirate from right heart catheter.

5. Ventilate patient and start advanced life support.

6. Locate source of air and prevent further entrainment.

access line
- unsealed central line
- migrated access cannula
- rupture

pump head/oxygenator
- plastic housing breaches, fractures, cracks
- unsealed access points
- rupture

7. Remove any remaining air.

8. Release clamps and re-establish ECMO flow.

ECMO
AIR IN CIRCUIT

Paul Sadleir | David Borshoff

6 If air is in access line (negative pressure side of circuit) check all central lines are sealed, access cannula has not migrated or decannulated and there is no rupture or breach.

If air is in pump-head or oxygenator, check for any breaches/fractures/cracks of plastic housing and systematically check all access points.

Membrane rupture mandates circuit change-out.

If arterial air embolism, consider cooling to reduce O_2 consumption and facilitate reabsorption of embolised air.

7 Air removal

- **Access line**
  Attach 50mL syringe to line Luer lock, manipulate line air to lock location and aspirate.
- **Return line**
  Treat as per access line but briefly unclamp return line to also aspirate air from return cannula.
- **Pump-head/oxygenator air**
  - Take out of drive, lower and manipulate air into access line for aspiration.
  - Aspirate all oxygenator membrane ports, and briefly unclamp return line to reprime circuit from patient.
  - Replace oxygenator/pump-head in drive, release access line clamp, and turn to 3000RPM with vents open for 2 min to purge.

ECMO
BLEEDING

Paul Rodoreda | Paul Sadleir

1. Check ACT.
2. Apply pressure to external site of bleeding.
3. Confirm appropriate heparin infusion rate and composition.
4. Stop or decrease infusion if:
   - ACT > 180s
   - APTT > 90s
   - anti-Xa > 0.5 IU/mL
5. Transfuse RBCs, platelets and factors to target levels.
6. Consider tranexamic acid infusion.
7. Treat any *acquired* Von Willebrands disease.
8. Lower APTT target or stop anticoagulation.
9. Consider circuit change-out if PFHb > 0.1g/dL.
10. Address any underlying surgically correctable cause.

ECMO
BLEEDING

Paul Rodoreda | Paul Sadleir

Significant clotting deficiencies should be corrected prior to the administration of heparin, cannulation and initiation of ECMO.

Major bleeding
Defined as > 20mL/kg/24 hour period, or significant retroperitoneal, intracranial or pulmonary haemorrhage.

5 Coagulopathy treatment

Result	Treatment
INR > 1.5	FFP
fibrinogen < 2g/L	cryoprecipitate or fibrinogen concentrate
platelets < 100,000/mm3	platelets
Hb < 8g/dL	RBCs

Tranexamic acid may be considered but use cautiously in hypercoagulable state.

7 Evidence suggests acquired Von Willebrands disease is very common on ECMO and treatment using VW factor may be of benefit.

Consumptive coagulopathy may develop as a consequence of circuit intravascular clot and haemolysis, and is suggested by a rise in plasma free haemoglobin. Circuit change-out may be necessary.

Off-label use of recombinant factor VIIa has been described but risks catastrophic circuit thrombosis.

ECMO **CIRCUIT CHANGE-OUT**

Ken Williams | Paul Sadleir

1. Delegate second operator to prime and de-air new circuit on back up machine or hand crank.
2. Check ACT > 200s.
3. Turn sweep gas to 100% O_2.
4. Apply defibrillation pads.
5. Double clamp access and return lines close to new unit.
6. Disinfect and divide between clamps, add connectors and cable tie.
7. Clamp and divide access and return lines close to old unit.
8. Pass old divided lines to the second operator.
9. Squeeze tubing to expel air and fill with sterile saline, connect to new circuit and cable tie.
10. Confirm O_2 on, increase RPM to 1000, unclamp venous and arterial lines and establish full flow.
11. Connect heater-cooler to new circuit.

ECMO **CIRCUIT CHANGE-OUT**

new circuit set up

Ken Williams | Paul Sadleir

This procedure should be practiced using simulation every 3 months.

Indications for circuit change-out

- transmembrane pressure > 60mmHg
- outflow PaO_2 < 150mmHg
- pumphead noise or large thrombus
- heat exchanger rupture
- haemolysis

There is little time to complete this before cardiac arrest if patient is completely ECMO dependent. Its advisable to have a second primed circuit ready whenever ECMO is in use.

New circuit set up

1. Insert HLS Advanced module on to Cardiohelp drive (or crank).
2. Attach 3 Luer lock/ J loops to pre and post-membrane apical ports and post-membrane return port.
3. Connect interface cable for integrated sensors.
4. Attach flow and bubble sensors to the arterial and return lines.
5. Connect gas supply and set sweep flow to 100% oxygen.
6. Fill the priming bag with 1.5L of plasmalyte.
7. Remove quick action couplings and connect to main circuit.
8. Turn on console, activate *Global Override*.
9. Calibrate integrated pressure sensors (time permitting).
10. Open clamps and prime circuit by gravity. Aspirate all Luer lock connections.
11. Run pump at 3000rpm for 2 min.
12. Check circuit primed and debubbled.
13. Pump off.

7 Double clamping close to old unit prevents loss of volume and RBCs.

ECMO
CIRCUIT RUPTURE

Warren Pavey | Paul Sadleir

Initial actions

1. Assign roles to patient resuscitation or circuit troubleshooting.
2. Try immediate temporizing measures but if catastrophic, clamp out ruptured section to prevent exsanguination or air embolism.
3. If partial decannulation, attempt re-insertion and monitor for air embolism 47.
4. Consider early set up and priming of replacement circuit.

Pre pump head circuit breach (negative pressure)

1. Clamp access and return lines and turn pump off.
2. If air embolus suspected, place head down and aspirate right ventricle if lines are in place.
3. Ventilate patient and support circulation.
4. Re-stablish access with new cannula or replacement of breached segment.
5. De-air old circuit if salvageable or prime new circuit and connect.

Post pump head circuit breach (positive pressure)

1. Close any open tap and apply pressure to circuit breach.
2. Give volume if significant blood loss.
3. If bleeding uncontrolled, clamp lines and stop pump.
4. Repair the breach with new cannula or segment cut out.
5. Consider circuit replacement if repair not possible.

Central decannulation

1. Declare emergency. Call for surgeon and sternotomy trolley.
2. Prepare for massive catastrophic bleeding. Activate MTP.
3. Ventilate patient, volume resuscitate and start cardiac surgery advanced life support (CALS).
4. Consider circuit change-out as potential for thrombosis in stagnant circuit.

ECMO
CIRCUIT RUPTURE

Warren Pavey | Paul Sadleir

Non-catastrophic circuit rupture may be temporised using sterile occlusive dressings, bone wax, or manual pressure while a replacement circuit is set up and primed.

Access breach

A breach of the access side of the circuit will result in air entrainment, rapid de-priming of the ECMO circuit and the resultant loss of ECMO support. Massive air entry will result in rapid deprime of the pump with loss of forward flow. Because of this, air tends not to enter the patient.

If air does enter the venous system, treatment should be in accordance with 27.

Return breach

A breach on the arterial or return side of the circuit produces high-pressure blood loss that can rapidly lead to exsanguination, loss of ECMO support and associated haemodynamic collapse.

If air does enter the patient's arterial circulation consider cooling.

The speed of decline depends on time to detection, size of defect or rupture and the underlying cardiac and respiratory reserve of the patient.

Central decannulation can be catastrophic and requires immediate surgical and perfusionist involvement.

CALS = Cardiac Advanced Life Support.

Unexpected decannulation and circuit rupture is life threatening. Patient survival depends on team training and the speed of response. Three monthly simulation training for these scenarios is an integral part of any ECMO program.

ECMO
CLOTTING IN CIRCUIT

Paul Sadleir | Steve Same

1. Increase APTT to 60-70s or ACT > 200s.

2. Avoid or remedy any low flow periods (< 2L/min).

3. Immediately treat any reduction in pump flow 53.

4. Check transmembrane pressure (TMP) and post-oxygenator PaO_2.

5. Change-out circuit if TMP > 100mmHg.

6. Treat associated hyperfibrinolysis or thrombocytopenia.

ECMO
CLOTTING IN CIRCUIT

Paul Sadleir | Steve Same

Signs	
Oxygenator thrombosis	acute increase in TMP decrease in circuit flow for same pump RPM worsening of gas transfer (50% decrease in post-oxygenator PaO_2 or < 200mmHg)
Pump head thrombosis	abnormal noise from pump head haemolysis (increased PFHb > 3g/dL) thrombocytopenia

The circuit must be regularly checked for clot formation which typically develops within the pump head and on the inflow side of the oxygenator. Areas of stasis (eg. clamped tubing bridge) should be checked. Flow rates below 2L/min for prolonged periods must be avoided. Small clots may be seen in the pump head or on the inflow side of the oxygenator. This does not always adversely affect oxygenator function and may not warrant oxygenator changeout.

Thrombus-induced hyperfibrinolysis may result in D-dimer levels > 25-30mg/L.

Standard ECMO anticoagulation

Confirm platelet count > $80x10^9$/L and use 70 IU/kg heparin bolus.
Initial target ACT > 200s or APTT 60s (40-50s in patients at increased risk of bleeding).
Maintain ACT 150-180s with heparin infusion rates of 10-50 units/kg/hr.

Treat heparin resistance with 500-1000 IU antithrombin III concentrate or FFP.

HITTS should be considered if severe thrombocytopenia 5-10 days after heparin initiation or immediately on re-exposure.

ECMO
HAEMOLYSIS

Paul Sadleir | Steve Same

1. Check pump flow setting < 4000rpm.

2. Reduce pump speed if excessive negative access line pressure.

3. Review access side of circuit and treat access insufficiency.

4. Examine pump-head and oxygenator for fibrin/clots.

5. Exclude water to blood heater-cooler leak.

6. Use echo to check cannula position and patency.

7. Consider circuit change-out.

8. Consider plasma exchange transfusion.

ECMO
HAEMOLYSIS

Paul Sadleir | Steve Same

Plasma free haemoglobin should be measured daily or when clinically indicated. Values of 0.05-0.1g/dL are viewed as acceptable but if > 0.1g/dL, the underlying cause must be identified and rectified. This may require inserting a second access cannula or circuit change-out.

Causes	Effects
shear stress old red cell transfusion line chatter/negative line pressures blood clots/cannula obstruction high flows/pressure changes	renal failure jaundice hyperkalaemia haematuria vascular/organ dysfunction

Sources of haemolysis

Circuit-induced

- circuit at least one week old
- rising D-dimer level
- decreasing fibrinogen level
- increased blood product requirement

Oxygenator thrombosis-related

- increased transmembrane pressure
- visible clots in oxygenator (pre > post)
- low post-oxygenator PaO_2

Pump head thrombosis-related

- rise in PFHg
- reduction in flow rate for RPM

ECMO
LOW FLOW

Ken Williams | David Borshoff

1. Check correct RPM setting.
2. Review monitors to confirm flow and exclude sensor error.
3. Examine access and return lines for kinks or obstruction.
4. Confirm cannula positions and inspect for clot using echo.
5. Ensure patient adequately sedated and paralysed.
6. Check head position neutral if using IJV access.
7. Exclude underlying pathology
   - haemorrhage/hypovolaemia
   - tension pneumothorax
   - tamponade
   - raised intraabdominal pressure
8. If access line *suck down* or *chatter*, reduce RPM and then smoothly increase again to re-establish target flow.
9. Add up to 1L volume, repeat step 7 and consider a second access cannula.
10. If access side of circuit functional, consider diagnosis based on pressure combinations.

ECMO
LOW FLOW

Ken Williams | David Borshoff

Monitored line pressure, blood pressure, SvO_2, lactate and urine output can confirm low flow status. If all are normal, suspect flow probe malfunction.

Normal pump flow rate for VA ECMO is 60-80mL/kg/min or 2.2L/min/m^2. Target blood flow in combination with native cardiac function must provide adequate systemic oxygen delivery, allow some native cardiac and pulmonary artery flow (to avoid stasis and clot formation in the cardiac and pulmonary vasculature) as well as adequate decompression of the right and left cardiac chambers.

VV ECMO target flow rates must provide adequate arterial oxygenation while allowing non-injurious lung ventilation.

A drop in flow *with stable RPM* will be due to either decreased preload or increased resistance to return flow.

Causes of decreased flow with stable RPM	
Decreased preload	**Increased resistance to return flow**
hypovolaemia or bleeding obstruction to venous return (tamponade, pneumothorax, abdo comp syndrome) kink or obstruction in access line	increased SVR clots in line/cannula kink or obstruction in return line/cannula altered cannula position

Hypovolaemia is a common cause of line chatter.
CVP readings for monitoring trends may be helpful.
Concealed bleeding may be retroperitoneal, into the groin or gastrointestinal.

10 Transmembrane pressures (Δ_P)	
Low/Normal Δ_P	When associated with **elevated return line or patient pressures** is due to high SVR or return line/cannula obstruction. Treatment may include vasodilators for elevated patient BP and TOE examination of cannula for position and clot.
Low/Normal Δ_P	When associated with **reduced return line or patient pressures** is due to inappropriate low RPM, hypovolaemia, magnet decoupling or air in circuit. For decoupling reduce RPM and then slowly restart, or stop flow, clamp access line, remove and then re-engage pump-head and smoothly restart while unclamping. For air in line see 42.
Increased Δ_P	Usually indicates thrombus or fibrin deposition. May be due to period of low flow, poor anticoagulation, hypercoagulability or prolonged use (> 30 days with coated cannula).

ECMO **LOW PaO_2**

Veno Arterial ECMO

Ken Williams | David Borshoff

1. Confirm O_2 supply connected and 100% O_2 sweep gas.
2. Give 100% O_2 to patient lungs until issue resolved and ensure adequate ventilation if arterial line pulsatility evident.
3. Check for adequate flow (2.4L/min/m^2) and venous drainage 07.
4. Exclude haemo/pneumothorax.
5. Check for clots or air and oxygenator blood flow > 2L/min.
6. Change-out circuit if oxygenator issue unresolved.
7. Check for blood in exhaust gas port.
8. Review heater-cooler for obstruction or water/blood leak.
9. Exclude seizures, sepsis or hyperthermia, and reduce the O_2 demand with paralysis and/or cooling to 35°C.
10. Consider changing to central VA ECMO or VAV ECMO.

ECMO **LOW PaO_2**

Veno Arterial ECMO

Ken Williams | David Borshoff

Oxygenator failure

The pre and post-oxygenator pO_2 should be measured. If post membrane pO_2 < 200mmHg consider semi-elective change-out.

If air is seen in the oxygenator it may be possible to vent but if not, or there is blood in the exhaust gas port indicative of membrane rupture, change-out circuit.

Seizures, sepsis and hyperthermia will all increase oxygen demand. Treating the underlying condition, cooling to 35°C and ensuring complete muscle paralysis will help reduce O_2 utilization.

Differential hypoxia (Two-Circulation Syndrome)

Retrograde flow from a femoral arterial cannula can mix with blood ejected from the heart. The mixing point depends on the degree of cardiac function. The poorer the function the closer the mixing point to the aortic root. If cardiac function returns, the mixing point moves distally.

> If the lungs are not ventilated adequately, or there is significant pulmonary pathology when cardiac function returns, poorly oxygenated blood will be ejected into the aortic arch with the associated risk of hypoxic brain damage.

This can be corrected by changing to central VA ECMO or VAV ECMO.

Simultaneous VAV ECMO and decreasing ECMO flow can result in decreased afterload of the left ventricle. It promotes a decrease in left atrial pressure resulting in improvement of pulmonary oedema.

5 Check ACT > 200s and if air is present attempt to vent.

7 Blood detected in exhaust gas port is indicative of membrane rupture.

ECMO **LOW PaO_2**

Veno Venous ECMO

Paul Sadleir | David Borshoff

1. Confirm sweep gas 100% O_2 and connected to oxygenator.
2. Increase ECMO circuit flow to 2/3 patient cardiac output.
3. Check oxygenator output PaO_2 > 200mmHg.
4. Increase sweep gas flow to equal ECMO circuit flow.
5. Optimise lung function using FiO_2, PEEP, iNO ± prone position.
6. Cool to 35°C and transfuse to target Hb > 10g/dL.
7. Check access pO_2, treat significant recirculation and consider additional access cannula.
8. Treat a hyperdynamic circulation by reducing inotropes and adding beta blockade.
9. Trial an increase in ECMO circuit flow rate if access PaO_2 low.

ECMO **LOW PaO_2**

Veno Venous ECMO

Paul Sadleir | David Borshoff

An acceptable PaO_2 during veno venous ECMO requires a functioning oxygenator, adequate ECMO flow rates relative to patient cardiac output, absence of recirculation and appropriate Hb. Failure to oxygenate *extracorporeal* flow is excluded by checking oxygenator outlet pO_2.

Hyperdynamic circulation
ECMO circuit flow rate should be at least 2/3 patient cardiac output to adequately supplement mixed venous oxygen content. This can be difficult to achieve in the setting of a hyperdynamic circulation. Cooling and beta blockers help reduce cardiac output and oxygen demand.

Access insufficiency may resolve with volume supplementation or an additional access cannula.

Recirculation
Recirculation is indicated by an inappropriately high access blood pO_2 relative to the patient mixed venous or arterial pO_2. Return flow is directed back to the oxygenator rather than the tricuspid valve. This may result from the return and access being in close proximity, or a two lumen IJV cannula being mispositioned proximally in the innominate vein. Recirculation is more likely with smaller cannulas or high ECMO flow rates.

Additional access
If there is recirculation, low circuit flow (relative to cardiac output), line chatter or evidence of haemolysis, adding a second access cannula may be beneficial.

The PERFUSION CRISIS MANUAL

Cardiopulmonary Bypass
Extracorporeal Membrane Oxygenation

The Perfusion Crisis Manual is a practical quick reference handbook in aviation checklist format, giving step-by-step guidance for management of cardiopulmonary bypass and ECMO crises encountered in the OR or Intensive Care.

- 55 crisis management protocols covering major perfusion emergencies.
- An emergency procedure section containing checklists for rapid set ups.
- A separate section specific for ECMO troubleshooting.
- A color coded, tabbed layout enabling fast navigation.
- A must for every operating room, perfusionist and trainee.

Dr Paul Sadleir is a cardiac anesthesiologist and medical perfusionist in Perth, Western Australia. He is qualified in perioperative echocardiography, a graduate in professional writing and a contributing author for the University of Melbourne Medical Perfusion course. With his clinical experience and writing background, Dr Sadleir is an in-demand educator of anaesthesiologists and perfusionists across Australia.

Dr Steve Same has worked in cardiothoracic anaesthesia and medical perfusion since1984. He was instrumental in the establishment of cardiac services for one of the largest private cardiothoracic units in Australia and was the department director for over 20 years. He has been directly involved in more than 16000 cardiac cases, chaired numerous committees and remains dedicated to furthering the role of perfusion in patient management.

Dr David Borshoff is an anesthesiologist in Perth, Western Australia. He has worked in cardiothoracic, general and paediatric anaesthesia, holds a recreational pilot's licence and maintains an interest in human factors, cognitive aids and patient safety. He is also the author of The Anaesthetic Crisis Manual and editor of The Resuscitation Crisis Manual.

Visit **leeuwinpress.com.au**
to purchase this manual.

LEEUWIN PRESS