

Right Ventricular Failure

Protocol

For completion by Author			
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Document Statement

Acute right ventricular failure (failure of the sub-pulmonic ventricle) can be defined as a rapidly progressive syndrome with systemic congestion resulting from impaired RV filling and/or a reduced RV flow/output.

Right ventricular failure has a relatively low incidence amongst non-congenital cardiac surgery patients but is a recognised cause of early graft failure in orthotopic heart transplant. Many cardiac procedures cause a degree of right ventricular dysfunction with clinically significant effects occurring in up to 20% of patients. When right ventricular failure occurs, it presents a clinical challenge due to the often limited evidence based therapeutic treatment options available to and is correspondingly associated with a high degree of morbidity and mortality. Severe right ventricular heart failure is reported in 0.1% of patients undergoing cardiac surgery with mortality reported to be between 70 - 75%.

The objective of this document is to provide information regarding the assessment and treatment options for the patient with right ventricular failure (sub-pulmonic ventricle) particularly in the post-operative phase though some management strategies can be extrapolated and employed in the treatment of non-post-operative RV failure.

1. Roles and Responsibilities

Medical Director

The above Director is responsible for implementation of this policy.

The Drug and Therapeutics Committee

The above Committee is responsible for development and approval of this policy.

Liverpool Heart and Chest Hospital NHS Trust Staff are responsible for co-operating with the development and implementation of Corporate policies as part of their normal duties and responsibilities.

Temporary or Agency Staff, Contractors, Students or Others will be expected to comply with the requirements of all Trust policies applicable to their area of operation.

Medical staff will be responsible for the prescribing of therapy including parenteral, inhaled and oral, using the LHCH in-patient prescriptions appropriately (the Electronic Prescribing System should be used for all in-patient prescribing including intravenous continuous therapy). Mechanical support will not be prescribed but will be documented via EPR.

Nursing staff will be responsible for administration of therapy in accordance with the Medicines Policy and may support mechanical therapy after insertion by medical staff with perfusion support. Nursing staff will also be responsible for the onward communication information regarding their patients.

Pharmacists will be responsible for ensuring appropriate support for the medical and nursing teams using treatments of RV failure.

2. Controlled Document Standards

All staff involved with RV failure management will be expected to comply with current legislation e.g. Medicines Act 1968 and the trust's Medicines Policy, together with their code of professional practice ensuring safety of patients and other staff at all times.

3. Procedure

Glossary of terms

RV – right ventricle

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CPB – cardiopulmonary bypass
 RCA – right coronary artery
 RV FAC – right ventricular fractional area change
 PFO/ASD – patent foramen ovale/atrial septal defect
 CI – cardiac index
 TR – tricuspid regurgitation
 PR – pulmonary regurgitation
 TAPSE – tricuspid annular plane systolic excursion
 MAP – mean arterial pressure
 iNO – inhaled nitric oxide
 ECMO – extra-corporeal membrane oxygenation
 VAD – ventricular assist device
 RVAD – right ventricular assist device

Factors that increase the risk of acute right ventricular (RV) failure post-operatively include:

- Pre-operative RV dysfunction
- Pulmonary vascular disease i.e. high RV afterload
- Congenital heart disease
- Supraventricular Tachycardias
- CPB time >150 minutes
- Air embolism to the right coronary artery or injury to the RCA following surgical procedures which require RCA mobilization.

Diagnosis of acute right ventricular failure

- Right ventricular fractional area change (RV FAC) less than 35% on transesophageal echocardiography (mid oesophageal 4 chamber view) or transthoracic echocardiography (apical 4 chamber view)
- Right ventricular distension with flattening of the intraventricular septum or ventricular interdependence
- On transoesophageal echocardiography - interatrial septum bowing to the left +/- right to left shunting through patent foramen ovale (PFO) or atrial septal defect (ASD)
- Worsening tricuspid regurgitation compared to baseline
- Failure to wean of cardiopulmonary bypass or severe haemodynamic instability due to right ventricular failure
- Elevated central venous pressure
- New liver dysfunction
- Thermodilutional Cardiac Index (CI) <2L/min/m² on pulmonary artery floatation catheter (PAFC, also known as a Swan-Ganz catheter) in the absence of more than moderate tricuspid or pulmonary regurgitation

Minimum monitoring requirements for haemodynamically unstable RV failure include:

- Arterial line
- Central venous line
- ECG
- Invasive blood pressure monitoring

RV Failure Treatment Algorithm

Management of RV failure should include preload optimization; afterload reduction; and contractile support once any primary pathology is corrected.

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Step 1:

- Optimise respiratory and metabolic parameters (PaO₂, PaCO₂, pH, temperature- correct hypoxia, hypercapnia and acidosis. It may be advisable to adjust the ventilatory settings to reduce positive end expiratory pressure (PEEP) to <8 cmH₂O, tidal volume 6-8mls/kg and plateau pressure <30cmH₂O whilst maintaining acceptable oxygenation and CO₂ levels to optimize RV filling/afterload. Permissive hypercapnoea should not be undertaken whilst RV failure is present.
- Consider atrial or sequential pacing. Due to the lower contractile performance of the RV, increasing the heart rate may increase RV output significantly where bradycardia has been a particular issue.
- Optimise analgesia to reduce sympathetic tone

RV function should be reassessed at this point with central venous pressure (CVP) monitoring, cardiac output, arterial gas monitoring, echocardiographic assessment (FAC, TAPSE, RV size, tricuspid regurgitation and atrial septal movement/flow) and liver function tests

Step 2:

- Optimise fluid status - consider coming off CPB 'empty'. In the majority of patients with reduced RV contractility attempting to reduce the RV preload and therefore RV dilatation will lead to reduced RV oxygen demand and will increase RV contractility. CVP should be not be above 12 cmH₂O. Less commonly, fluid boluses maybe needed and this should be in the form of a 250mls bolus and guided by a CVP- aiming for 8-12 cmH₂O.
- Improve coronary perfusion pressure aim for mean arterial pressure (MAP) >65mmHg or higher if suspected RCA embolus
- Diuretics should be used in those with signs of overload or venous congestion.

RV function should be reassessed at this point with CVP monitoring, cardiac output, arterial gas monitoring, echocardiographic assessment (FAC, TAPSE, RV size, tricuspid regurgitation and atrial septal movement/flow) and liver function tests

Step 3:

- Maintain MAP with noradrenaline - max 0.3mcg/kg/min to limit pulmonary vascular resistance (PVR) increase. Consider early addition of intravenous vasopressin in order to decrease dose of noradrenaline. Once the MAP has been stabilized with ongoing RV dysfunction, RV inotropy should be improved with phosphodiesterase 3 inhibitors, such as milrinone or enoximone. Because they cause a significant drop in systemic vascular resistance (SVR), they should be used in combination with noradrenaline in order to augment the perfusion pressure to the right ventricle.
- Enoximone – loading 0.5 - 1mg/kg and maintenance 5 - 20 mcg/kg/min (max 24mg/kg/day)
 - Enoximone has a long half-life: so effects of reducing the dose are seen after 4 – 6 hours
- Milrinone – loading 50mcg/kg over 10 minutes and maintenance 375 - 750 nanograms/kg/min (max 1.13mg/kg/day)
 - Milrinone has a short half-life, so effects of dose change will be seen relatively quickly.

RV function should be reassessed at this point with CVP monitoring, cardiac output, arterial gas monitoring, echocardiographic assessment (FAC, TAPSE, RV size, tricuspid regurgitation and atrial septal movement/flow) and liver function tests

Step 4:

- Consider intravenous adrenaline 0.01 - 0.07 mcg/kg/min as this may improve cardiac output without severely increasing PVR. In refractory hypotension adrenaline maybe particularly helpful.

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RV function should be reassessed at this point with CVP monitoring, cardiac output, arterial gas monitoring, echocardiographic assessment (FAC, TAPSE, RV size, tricuspid regurgitation and atrial septal movement/flow) and liver function tests

Step 5:

- Inhaled nitric oxide (iNO) increases cardiac output by reducing PVR and is the preferred option in theatres for acute RV failure with high PVR. It has a synergistic effect with inhaled iloprost and can improve RV function. iNO has a very quick onset of action and maybe used with greatest ease and speed in theatre cases. iNO delivered by face mask, nasal cannula or endotracheal tube is commenced at 20ppm and weaned gradually as it can trigger rebound increase in PVR. Weaning needs to be done cautiously to avoid rebound pulmonary hypertension with the highest risk being during weaning the last 5ppm.
- Nebulised iloprost initiated in most cases at 5mcg diluted with 0.9% saline to 5mls every 3 hours. Iloprost can be used synergistically with iNO and can be started on the ward and would be preferred over iNO in the later postoperative phase. Iloprost can also be given IV if desired.
- Sildenafil improves cardiac output by balancing pulmonary and systemic vasodilation and also has a synergistic effect with nitric oxide. Sildenafil 20 mg TDS PO should only be initiated in case of rebound pulmonary hypertension after iloprost has been stopped. Sildenafil should not be started without discussion with specialists at the pulmonary hypertension unit (Royal Hallamshire Hospital, Sheffield).

A separate policy exists outlining the administration of inhaled nitric oxide and iloprost.

RV function should be reassessed at this point with CVP monitoring, cardiac output, arterial gas monitoring, echocardiographic assessment (FAC, TAPSE, RV size, TR and atrial septal movement/flow) and liver function tests

Step 6:

At this point in the algorithm, consideration should be made towards mechanical support and discussion with a cardiac transplant unit.

- IABP can be considered and may improve coronary perfusion if there is LV (systemic ventricular dysfunction which maybe worsening RV function or non-treatable RCA ischaemia)
- Protek Duo cannula (percutaneous ventricular assist device (VAD)) improves oxygenation and alleviates pressure on the right side of the heart. This acts as RV extra-corporeal membrane oxygenation (ECMO) and can inserted percutaneously.
- ECMO and RVAD have been used successfully in RV failure refractory to pharmacological interventions and early referral to an ECMO center should be considered.

*Methylene blue should be avoided as it increases PAP and PVR to unacceptable levels.

Available Mechanical support at LHCH

LHCH currently offers central veno-arterial ECMO (V-A ECMO) in cases of unexpected failure to wean from CPB and not primary or peripheral ECMO. ECMO maybe instituted following an ECMO MDT for these cases and RV failure constitutes a common cause of failure to wean from CPB and ECMO usage in LHCH.

The use of Protek Duo percutaneous RV ECMO has been approved in principle by the Clinical Audit and Effectiveness Group at LHCH and offers specific support for RV failure. Currently, its active use has not begun pending further training and this may be an option for isolated RV failure in more settings.

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Based on the EUROMACS-RHF score and patients who should be considered for the Protek system should have Cardiogenic shock/failure to wean from CPB plus at least one of the following:

- Need for ≥ 3 inotropes
- Severe RV dysfunction on echo
- RA to PCWP ratio > 0.54
- CPB time > 100 minutes

Insertion

Insertion is recommended primarily from the internal jugular vein or the femoral vein if needed. Prior to insertion, there should be confirmation that the access point is patent and that clotting/platelet indices and function are normal or as normal as the situation allows (insertion may occur during emergency RV failure with concomitant effects on the liver/clotting).

At the insertion point, an ultrasound guided puncture should be made and a haemostatic sheath 6fr and above inserted. Once the sheath has been inserted and flushed, access to the main pulmonary artery is required and this can be achieved using a balloon tip catheter or a diagnostic cardiac catheter with or without wire support (0.035 J wire or Terumo). It is recommended that this be done using a fluoroscopic screening with or without TOE support.

Once the catheter is confirmed in a stable distal PA position, a stiff wire such as a 0.035 Amplatz super stiff or Lundeqvist wire is uncovered over the catheter in the distal PA position. Once this is completed, the haemostatic sheath can be removed and subsequently dilators used to dilate the puncture point up to the required 29Fr sheath size. It is recommended, a reasonable incision is made in the skin to aid sheath passage.

Next after flushing all the ports of the Protek, the Protek can be advanced over the stiff wire to the main PA. There is X-ray visible braiding around the RA side holes and distal lumen to guide positioning. The dilator may need to be pulled back sometimes to allow the catheter to make the turn into the PA if the anatomy is difficult. Once in a stable main PA position, the wire and dilator can be removed and the RA lumen (marked proximal) be connected to the inflow portion of our Levitronix centrimag pump and PA lumen (marked distal) to the outflow after flushing and priming the pump. Oxygenation can be added dependent on respiratory function. The cannula should then be sutured in place once positioning on fluoroscopy and TOE is confirmed.

Commencement of RV support

Once the Protek has been connected to centrifugal pump and oxygenator as required, Heparin should be given intravenously aiming for a target ACT of 180-220 or an APTT of 65-80 (non-ratio time used) if there are no bleeding concerns. Heparin can be held till chest closure and if needed till drain output subsides to acceptable levels

The pump can be turned on and the rpm levels gradually increased till further rpm increase does not increase flow through the circuit. Maximum flow achievable is 4.5l/min via a 29fr Protek. Ideally, a cardiac index of greater than 2.2 should be aimed for with use of the Protek.

Cardiac index can be calculated using available means or using the following calculations:

Cardiac index: Cardiac output/BSA

Cardiac output:
$$\frac{VO_2 (VO_2=125 \times BSA)}{(Arterial\ oxygen\ sat - Mixed\ venous\ oxygen\ sat) \times Hb (Divide\ by\ 10) \times 13.4}$$

Practical setup and management of the system

Currently, a fully integrated pump system which is portable is available from Livanova but is not yet supported and sold in the UK. At the set-up point, a perfusionist will connect and commence the pump run with

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guidance/support from the senior ICU staff. It is envisaged due to the simple function of the device with training once the pump is set up, it can be monitored by ICU nursing staff but till then it will be monitored and adjusted the perfusion team.

Sub-pulmonary Ventricular failure in ACHD

In the majority of cases, management of acute RV failure is as illustrated above and is more common in the ACHD population due to the underlying anatomical problems and multiple previous interventions. There are some specific points which should be remembered:

- In some cases the sub-pulmonary ventricle will be a morphological left ventricle. Failure of this ventricle is uncommon but depends to be more preload dependent and so is less likely to respond to low filling pressures. Ventricular pacing is less deleterious than in a sub pulmonary right ventricle and so can be used more readily to augment cardiac output.
- In ACHD there are many conditions where the functional subpulmonary ventricle maybe relatively hypoplastic/borderline in size (e.g. Ebstein's anomaly) and management here can be challenging - augmenting the cardiac output should be managed as above though the size of the hypoplastic subpulmonary ventricle may limit the response.
- In tetralogy of Fallot/pulmonary atresia especially in later repairs, there can be development of restrictive physiology with significant issues with RV forward flow and reversal in vena caval flow. This phenomenon is not normally seen in acquired RV physiology. These patients will need a higher CVP to compensate for the increased ventricular stiffness, vena caval flow reversal and diastolic pulmonary forward flow. Reducing PEEP if ventilated will also help. There is evidence that direct adrenergic agents due a change in inotrope receptor make up in these patients are less/negative in their efficacy,

Adjunctive treatments that can be considered for RV failure

Given the limited number of treatments specifically for RV dysfunction, there are small scale trials or expert recommendations for other optional treatments:

- Ranolazine has been shown to have potentially beneficial effects on CO/ RV metabolism and function and pulmonary hypertension. It can be considered as adjunct if patients have exhausted all other options.
- Ascitic drainage if there is ascites may help with abdominal compartment syndrome and therefore improve RV preload
- Septostomy, can be considered in specific situations to improve RV function and cardiac output. The systemic ventricular function must be adequate to cope with the increased volume and this will come at the cost of oxygenation. This may be of particular use in the ACHD cohort.

4. Policy Implementation Plan

This policy will be disseminated via the Drug and Therapeutics Committee.

5. Monitoring of Compliance

Audit of the policy should be conducted every 3 years through morbidity and mortality incidents pertaining to RV failure.

6. References

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8. Endorsed By:

Name of Lead Clinician / Manager or Committee Chair	Position of Endorser or Name of Endorsing Committee	Date
Dr Nigel Scawn	Drug & Therapeutics Committee	15/9/2021
Dr Dennis Wat	Drug & Therapeutics Committee	15/11/2023

9. Record of Changes

Section No	Version No	Date of Change	Description of Amendment	Description of Deletion	Description of Addition	Reason