

V Reunión. Estado del Arte en INSUFICIENCIA CARDIACA AVANZADA PRÁCTICA CLÍNICA Y MODELOS ORGANIZATIVOS V Meeting. State of the Art in **ADVANCED HEART FAILURE** CLINICAL PRACTICE AND ORGANIZATIONAL MODELS

"Short and long-term mechanical circulatory support: Management in intensive care"

Dr Ana Hurtado Consultant Intensivist Harefield Hospital, UK



Right device for the right patient





why are you doing this? what do you want to achieve?

"bridge to decision" (non-durable)

"bridge to recovery" (non-durable and durable)

"bridge to transplant" (non-durable and durable)

"destination therapy" (durable)





when do we do it?

Refractory circulatory shock, on maximal medical therapy resulting in organ hypoperfusion

Emergency

Semi-elective

Elective





"exit strategy" and treatment objectives

before AMCS device use, which may include RV support as a bridge to recovery, a bridge to LVAD, biventricular VAD, a total artificial heart, or a bridge to orthotopic heart transplantation.





Short term support

IABP

ECMO

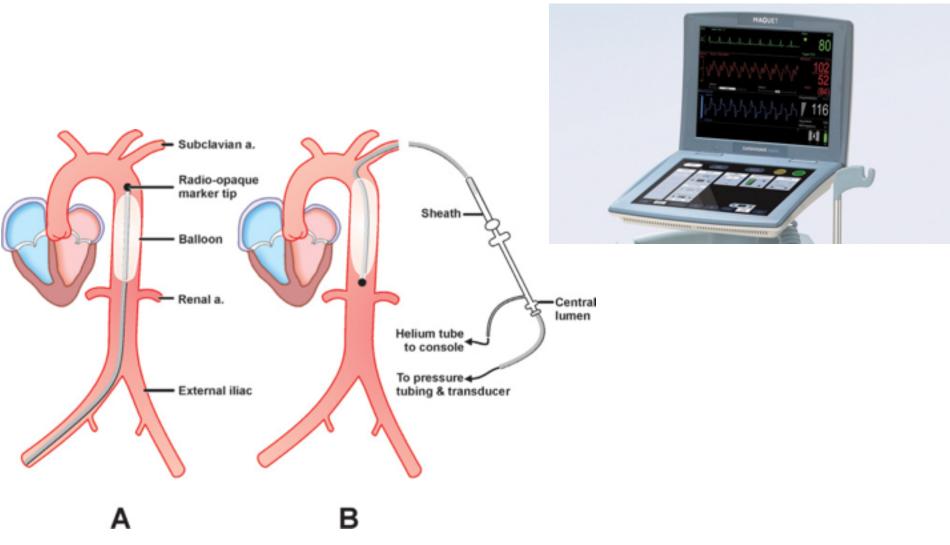
Impella

Univentricular/ Biventricular assist devices TandemHeart CentriMag RotaFlow Biomedicus





IABP







IABP

Implantation (cath lab, bedside by echo or CxR)

Synchronisation (auto/ECG/Arterial waveform) 1:1

Anticoagulation : internal protocols -UF Heparin (APTT 60-80/Heparin anti Xa 0.2-0.3)





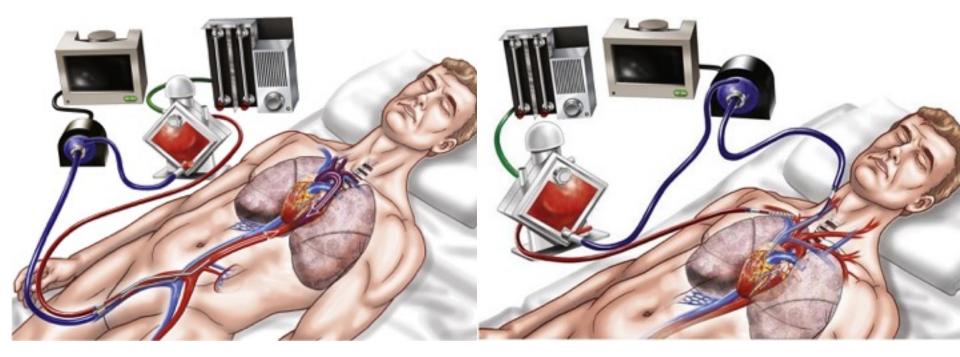
ECMO

Peripheral VA ECMO percutaneous cutdown reperfusion cannula Central VA ECMO VAV ECMO **VVA**





ECMO

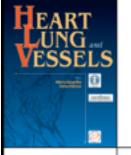






Mechanical circuit complications	Patient complications
Cannula and tubing	Renal
Wrong size	Capillary leak syndrome
Bleeding	Loss of auto regulation
Malposition	Fluid retention
Clotting	Hematological
Dissection	Hemolysis'
Decannulation	Thrombo-occlusive disorders
Bladder	Coagulopathy
Inadequate return	Neurological
Hypovolemia	Intracranial bleed
Increased intra-thoracic pressure	Sinus thrombosis
Venous cannula occlusion	Cerebral Infarction
Capillary leak Syndrome	Seizures
Air embolism	Cardiovascular
High FiO,	Myocardial stunning
Inlet obstruction	Sub-endocardial ischemia
Gas - blood leak	Poor capillary refilling
Pump	Hypoxia re-perfusion injury
Pump failure	Pulmonary
Loss of occlusion	Pulmonary fibrosis
Oxygenator -	Pneumonitis
Thrombosis - Membrane/Inlet/	Consolidation
Outlet port	Pulmonary hypertension
Fluid in Gas phase	
Failing oxygenator -	
Decreased O ₂ /CO ₂ transfer	
Widened pre- and post-membrane gradient	
Increased hemolysis	
Coagulopathy	
Heat exchanger	
Corrosion and leak	
Hemolysis, dilution and electrolyte imbalance	
Sepsis	
Hyponatremia, Hemolysis and seizures	

Royal Brompton & Harefield NHS



ORIGINAL ARTICLE

Heart, Lung and Vessels. 2015; 7(4): 320-326

320

Pitfalls in percutaneous ECMO cannulation

L. Rupprecht¹, D. Lunz², A. Philipp¹, M. Lubnow³, C. Schmid¹

¹Departmentof Cardiothoracic Surgery; ²Departmentof Anesthesiology; ³Department of Internal Medicine II/Pneumology, University Medical Center Regensburg, Regensburg, Germany

Table 1 - Conspicuous events and complications during and after percutaneous cannula placement in 159 patients with venous arterial extracorporeal membrane oxygenation.

Event	Consequence	Incidence
Mild limb ischemia	Clinical control	9.4%
Difficult puncture Bleeding during cannulation Vessel perforation during cannula placement	Multiple attempts Blood transfusion Surgical revision	8.8% 5.7% 1.9%
Upper body hypoxia	Cannula relocation (Subclavian artery)	8.8%
Vascular (femoral) complication	Surgical revision/ contralateral cannula relocation	7.5%
Mild bleeding after cannula removal	No revision	3.8%
Significant bleeding after cannula removal	Surgical revision	3.1%
Cannula dislocation	Cannula reinsertion	0.6%
Wound infection	Wound debridement	0.6%



Limb ischaemia

Femoral arterial cannulation

- Prevention: check vessel size prior to cannula insertion
- Monitoring/regular inspection and palpation of pulses/Doppler
- INVOS monitoring (regional oxygen sat)









ECMO circuit complications

Clots in circuit (turbin, oxygenator) Air in circuit Motor failure Oxygenator failure Tubing rupture (rare) Blood loss from circuit (faulty tap) Hemolysis Infection (rare)

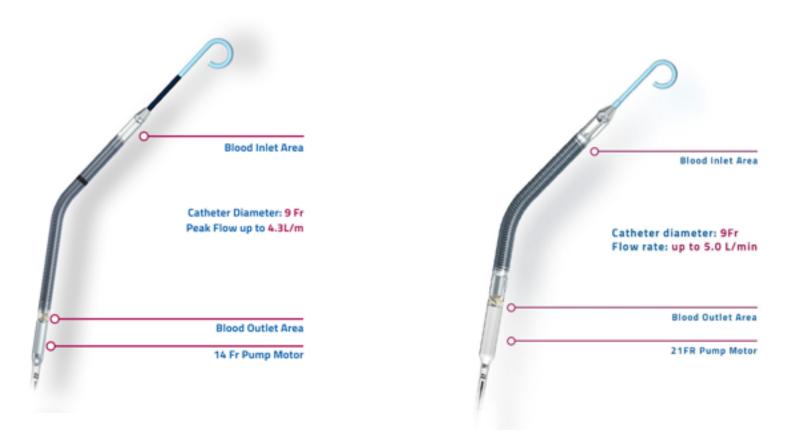






Impella CP[®]

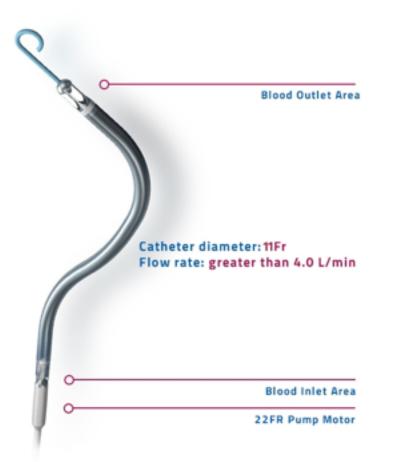
Impella 5.0[®]



Royal Brompton & Harefield NHS



Impella RP[®]







Impella®

Position

Anticoagulation: UF Heparin (APTT 60-80, AntiXa 0.2-0.3)

Aortic regurgitation

Thrombosis

Haemolysis: LDH, Plasma Free Hb, Bil, D-dimers

Weaning Impella

Gradual and continuous decrease in pump rate every 12h Discontinuation when P1 tolerated at least for 2h Echo: weaning trial Ao VTI>12cm, LVEF >25%, Lat MV peak syst velocity>6cm/s at P1 Weaning trial every 24h Impossible weaning >2weeks: long term support/transplant



Uni/Biventricular assist devices

TandemHeart CentriMag RotaFlow Biomedicus





CentriMag®



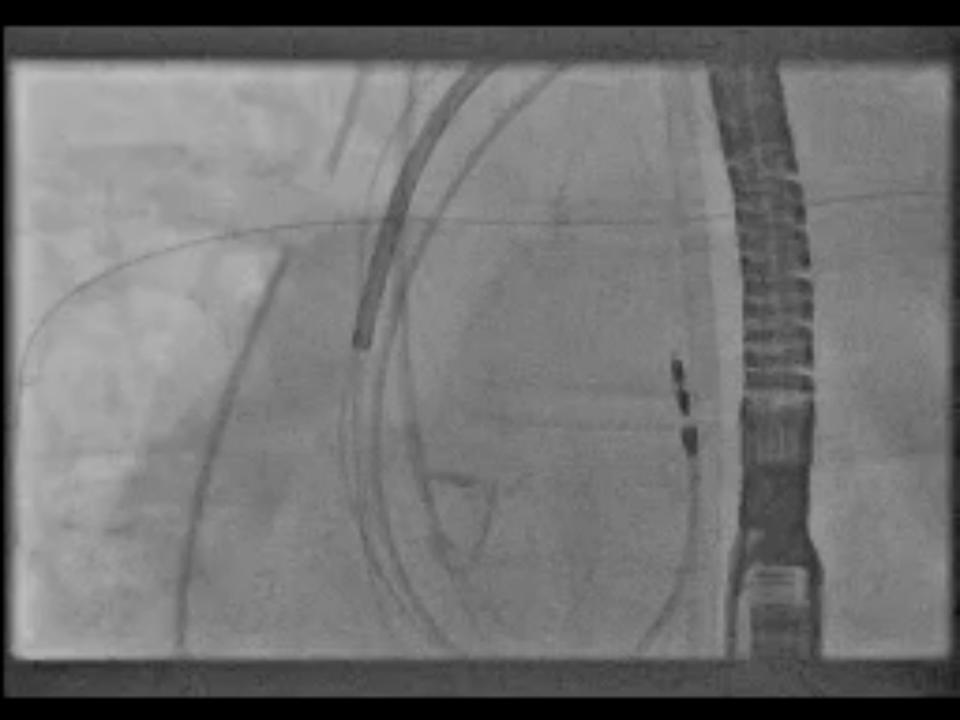




Protek Duo® cannula









Long term support

HVAD

Heartmate 3

Berlin Heart> EXCOR

AVAD

Total artificial Heart: SynCardia



HVAD/Heartmate 3/AVAD





Royal Brompton & Harefield **NHS Foundation Trust**





Berlin Heart/EXCOR



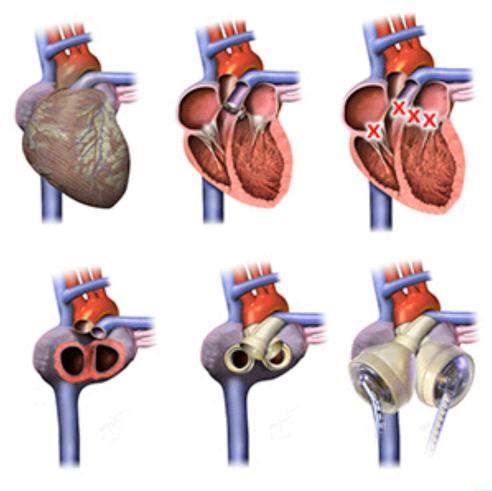






Total Artificial Heart









Harefield experience

	AVAD	Berlin Heart	Circulite	Circulite Synergy	C-Pulse	HeartAssist5	Heartware	Heartware Bivad	Heartware RVAD	HM II	Impella	TAH
2011-2012				2		4	12		1	11		
2012-2013			9				16			- 4		ľ
2013-2014			3				24	1				
2014-2015				1	1		42					5
2015-2016							37			1		3
2016-2017	2	1					23					5
2017-2018	1	1					32				3	5
2018-2019							5					1
Total	3	2	12	3	1	4	191	1	1	16	3	19





which problems are we going to face? (general, device specific, and evolving)

Anticoagulation Bleeding Thrombosis **RV** failure Haemolysis **Device** specific Infection





39 yo female High BMI (36) Recent diagnosis of DCM, likely postpartum (normal coronaries) Presented at local hospital (20/06/2017) on cardiogenic shock Echo: LV dilated, severely impaired, LVEF 10-15%, RV dilated and severely impaired Started on inotropes: **Dobutamine (5mcg/kg/min)** Initial improvement.

Discussed with Harefield Hospital and transferred conventionally





Right catheterisation study

	Dobutamine 5mcg/kg/h	Milrinone		
PVR	4.07	3.49		
PA	46/36/40	47/33/39		
PCWP	31	27		
TPG	9	12		
СО	2.2	3.4		
CI	1.08	1.68		



Dobutamine was changed to Milrinone 0.3 mcg/kg/min

Developed VT storm > transferred to ITU Electrolytes replaced, Amiodarone



Already on Milrinone (0.3mcg/mk/min), **Adrenaline** commenced due to hypotension



Lactate rising, worsening metabolic acidosis and LFTs





Discussed with patient Decision for **awake peripheral VA ECMO**

Performed in cath lab under fluoroscopy, USS guided percutaneous cannulation:

25F multistage right femoral vein 17F arterial cannula left femoral artery (no reperfusion cannula)

Started ECMO at 3.5lpm and inotropic support maintained





Prevention: check vessel size prior to cannula insertion

Monitoring/regular inspection and palpation of pulses/Doppler

INVOS monitoring (regional oxygen sat)









Awake, self ventilated ECMO at 3.5lpm Adrenaline 0.05mcg/kg/min Milrinone 0.3mcg/kg/min

Intermittently stopped ejecting, aortic valve closed Signs of **pulmonary oedema** with increased work of breathing Inotropes increased (milrinone 0.4mcg/kg/min, adrenaline 0.15mcg/kg/min)

but

Intubated few hours later...



Pulmonary oedema

Table 1. Hemodynamic Effects of Mechanical Circulatory Support Devices^{3,10-12}

Device	Flow	Left Ventricular Preload	Left Ventricular Afterload	Mean Arterial Pressure	
Intraaortic balloon pump	0.5 L/min	Slight decrease	Slight decrease	Slight increase	
Impella	Up to 5 L/min	Decrease	No change	Increase	
TandemHeart	Up to 5 L/min	Decrease	Increase	Increase	
Extracorporeal membrane oxygenation	Up to 6 L/min	Decrease	Increase	Increase	



MDT (Intensive Care, Cardiologist, Transplant surgeons)

- -Heart Tx not an option as High BMI and High PVR
- -Needs biventricular support, short term RVAD unlikely to work
- -Needs implantable system to allow mobilisation, rehabilitation and weight loss
- Therefore option is only realistically TAH SynCardia





SynCardia implanted 27/06/2017

Needed VV ECMO post implant due to hypoxia secondary to pulmonary oedema

Renal failure post TAH implant (>70% patients develop AKI) requiring RRT

30/06/2017: VV ECMO explant (3 days of support) and tracheostomy performed 2 weeks to wean from ventilator and being mobile

15/07/2017: Discharged to ward Renal function recovered

Discharged home December 2017





Case 1

- Pulmonary oedema postimplantation
- Need of temporary VV ECMO support
- **Bleeding**: Correct coagulation post implant
- Frequently Chest scented post implant during first 24h
- UF Heparin: APTT 60-80/Anti Xa 0.2-0.3
- Aspirin early when bleeding complications resolved





Selwa



https://www.bbc.co.uk/news/av/uk-england-london-42755505/the-ilford-woman-who-carries hereiton articles add



Case 2

50 yo male
Idiopathic DCM
08/06/2017: HVAD implanted bilateral thoracotomies and femoral CPB
Possible anaphylaxis when come off bypass
Admitted ITU
TOE post-implant: Reasonable RV function, moderate TR, Poor LV, Mild AR



Harefield recipe

- 1. Milrinone 0.2-0.5
- 2. Adrenaline
- 3. iNO at 20ppm
- 4. Noradrenaline +/- Vasopressin

Support the right ventricle!!







Case 2

Deterioration overnight

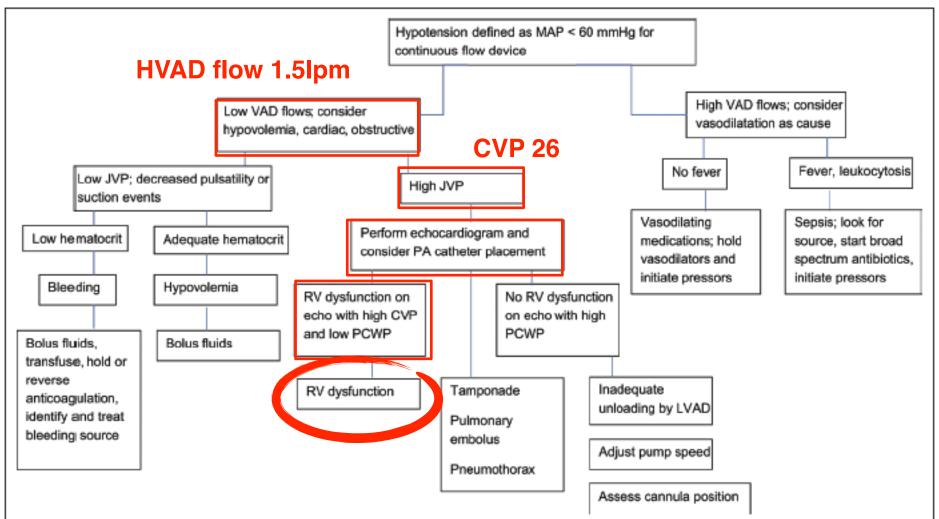
Hypotensive

Worsening metabolic acidosis

LVAD flow dropped to 1.5lpm



Hypotension LVAD





RV failure

- CVP 25
- Low LVAD Flow
- Hypotension
- Metabolic acidosis
- Echo showed severely dilated and impaired RV







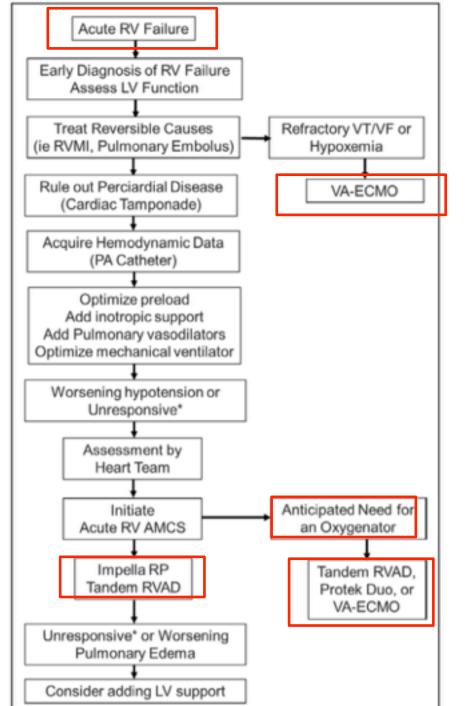


Mechanical Circulatory Support Devices for Acute Right Ventricular Failure Navin K. Kapur, Michele L. Esposito, Yousef Bader, Kevin J. Morine, Michael S. Kiernan, Duc Thinh Pham and Daniel Burkhoff

Circulation. 2017;136:314-326 doi: 10.1161/CIRCULATIONAHA.116.025290 Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231 Copyright © 2017 American Heart Association, Inc. All rights reserved. Print ISSN: 0009-7322. Online ISSN: 1524-4539











RV Failure

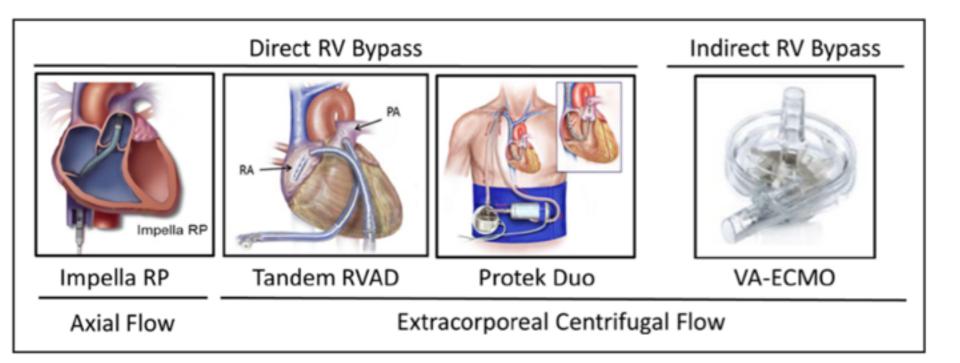




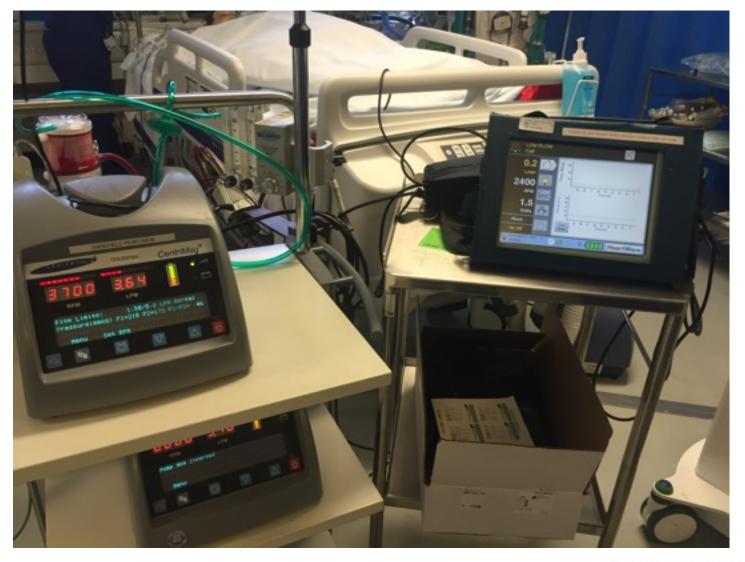
Table 3.Clinical Studies Evaluating the Utility ofAcute Mechanical Circulatory Support Systems forRight Ventricular Failure

Device	Patient Population	Outcomes	Study
Impella RP	18 Patients (15 Impella RD, 3 Impella RP) AMI, 39% (n=7) PCCS, 22% (n=4) Post-OHT, 17% (n=3) Post-LVAD, 11% (n=2) Myocarditis, 11% (n=2)	30-d Survival, 72% 1-y Survival, 50% Hemodynamic effects: increased CI, decreased RA pressure	Cheung et al ⁸³
	30 Patients Post-LVAD (n=18) PCCS/AMI (n=12)	30-d Survival, 73.3% Hemodynamic effects: increased CI, decreased RA pressure	Anderson et al ⁶⁵
TH-RVAD	46 Patients Postvalve surgery, 32% (n=15) AMI, 25% (n=12) Post-OHT, 11% (n=5) Post-LVAD, 11% (n=5) Post-CABG, 7% (n=3) Chronic HF, 7% (n=3) Myocarditis, 7% (n=3)	In-hospital mortality, 57% Hemodynamic effects: increased MAP, CI, and PA O ₂ saturation; decreased RA and PA systolic pressures No change in number of vasopressors/ inotropes	Kapur et al ^{at}
	9 Patients Sepsis, 11.1% (n=1) PCCS, 22.2% (n=2) IWMI, 66.7% (n=6)	In-hospital mortality 44% Hemodynamic effects: increased MAP, CI, RV stroke work; decreased RA pressure	Kapur et al ^{sz}
VA-ECMO	179 Patients PCCS, 39% (n=70) AMI, 26% (n=46) Primary graft failure, 10% (n=17) ADHF, 13% (n=24)	In-hospital mortality, 38.6% (n=69) Hemodynamic effects: decreased RA pressure and mean PA pressure	Truby et al ⁸⁵





Peripheral VA ECMO inserted





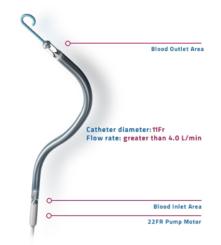


Case 2

Temporary solution as no flow on LVAD> high risk of thrombosis

Impella RP[®] placed a few hours after pVA ECMO

ECMO removed later on that day







HVAD Impella RP ICD TOE Drains ET tube CVC Vas-cath







Ongoing severe haemolysis...

ab Flowsheet	10/08/2017 08:00	11/08/2017 08:00	12/08/2017 08:00	13/08/2017 08:00	14/08/2017 08:00	15/08/2017 08:00	16/08/2017 08:00
Glucose	8.3	8.0	6.9	7.0	8.6	6.8	6.8
Total Bilirubin	85	80	196	332	240	190	231
Direct Bilirubin							
ALP	39	46	53	81	81	94	120
GGT				23			
ALT	28	27	26	38	36	34	35
Aspartate Transaminase							
Total protein	39	40	41	43	46	48	47
Albumin	23	25	27		27	26	24
Creatine Kinase	2373	2814					
Calcium	2.16	2.32	2.36	2.42	2.38	2.39	2.41
Calcium Corrected	2.50	2.63	2.63	2.67	2.65	2.68	2.73
Inorganic Phosphate	1.43	0.90	0.86	1.13	1.05	0.61	1.02
Magnesium	0.93	1.00	0.89	1.17		0.93	0.97
Amylase	107	109	117	89	62	66	8
CRP	89	136	148	204	211	188	17(
LDH	1429			3439		1396	1171
Troponin I							
Random Urine Sodium							
Plasma Haemoglobin	3.7			2.6	3.6	3.4	

Impella RP removed



Case 2

On Milrinone, adrenaline, sildenafil to facilitate iNO wean

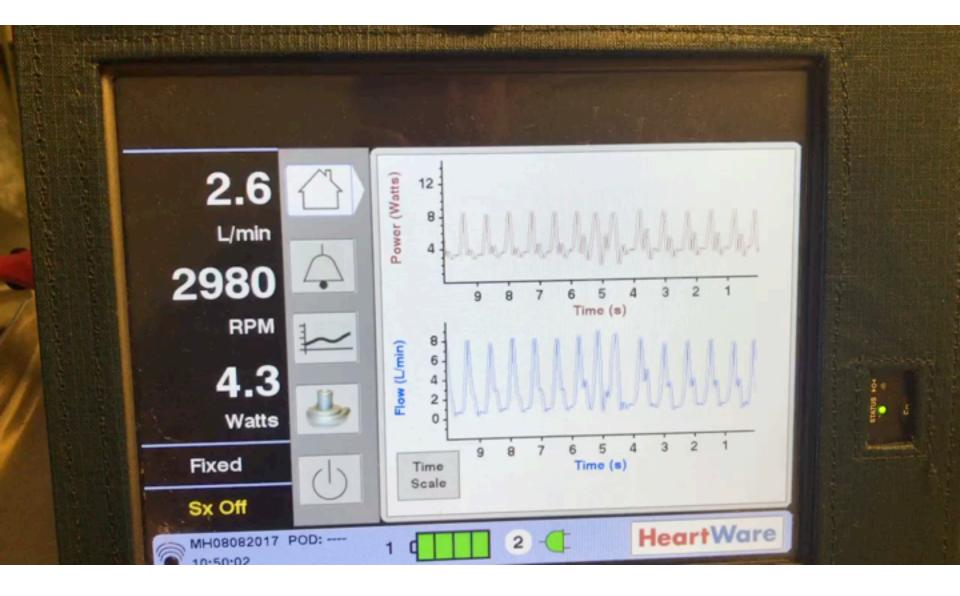
16/08/2017: tracheostomy

Resp wean

23/08/2017: LVAD flow dropped...









COL showed large left pleural effusion and pericardial collection



Royal Brompton & Harefield NHS NHS Foundation Trust



Case 2

Cardiac tamponade and re-exploration in theatre

Improved since then Heparin re-started 12h after re-exploration> Anti Xa 0.2-0.3

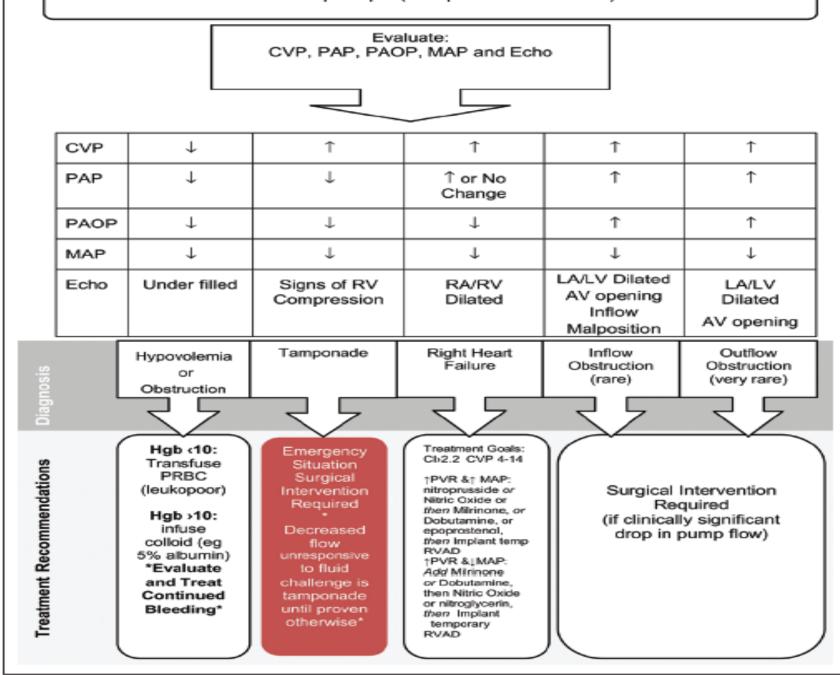
Wean off ventilator Recovered renal function

Discharged to ward

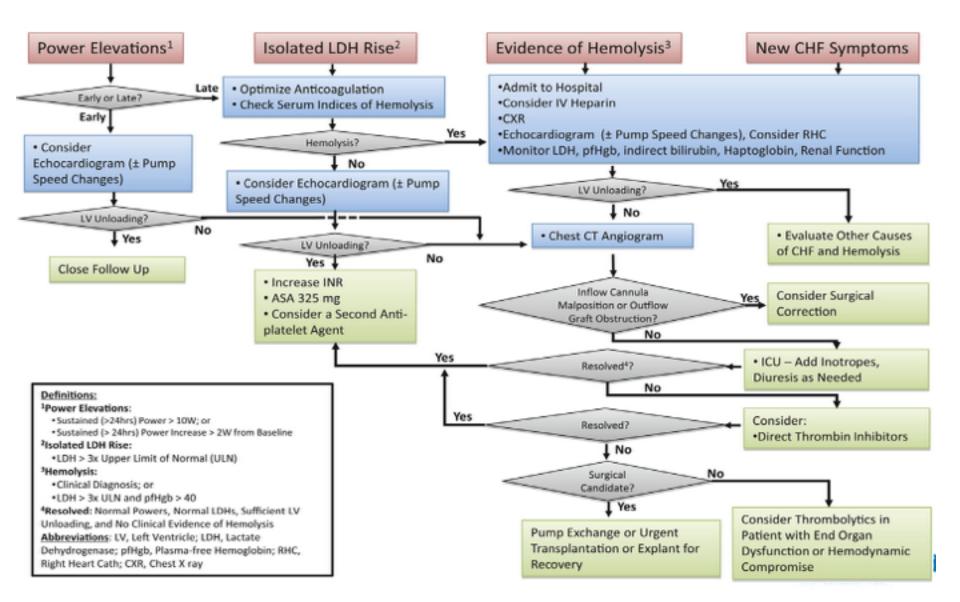




Low Pump Output (not speed or rate related)



Thrombosis post-LVAD





Thrombosis

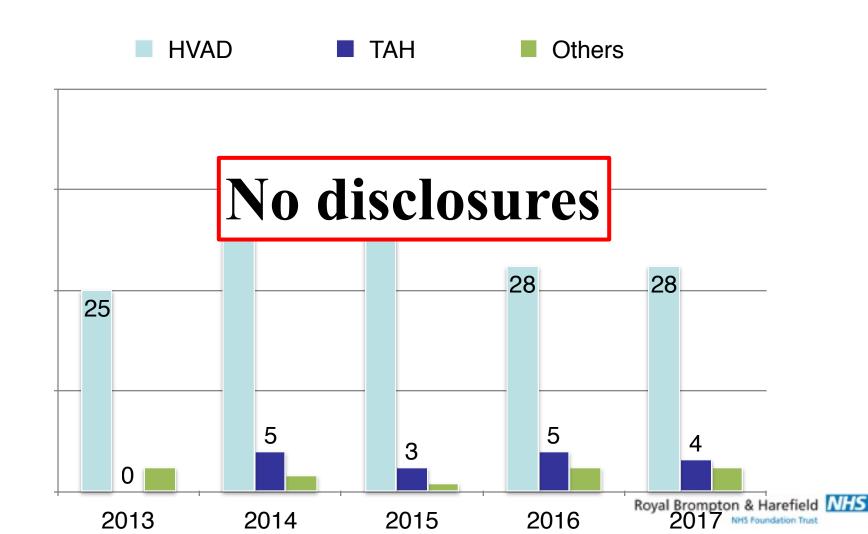
Sustained increase in LVAD power Signs of heart failure Cardiogenic shock Blood test: increasing LDH/plasma Free Hb Auscultation of pump

Tea-colored urine: severe haemolysis

- LDH >x3 upper limit
- Plasma free Hb >40mg/dL (>0.3)
- Low Haptoglobin level



Harefield experience





Thrombosis

- 1. CXR
- **2.** CT
- **3. Echocardiography:** dilated ventricle, severe MR, frequent aortic valve opening

-<u>"ECHO Test/ Ramp speed test":</u> Serial echocardiography recording of LV end-diastolic diameter (LVEDD) with increasing LVAD speed

– If **VAD thrombosis**, LVEDD fails to decrease in response to increasing LVAD speed

<u>Echo + LDH :</u> specific and sensitive test for the dx of VAD thrombosis with flow obstruction



Thrombosis

If suspected VAD thrombosis not discharge or readmit to ITU, high possibility of rapid progression

Treatment:

- IV Unfractioned Heparin
- Diuretics/inotropic support
- Aspirin high dose 325mg/d
- 2nd antiplatelet therapy (Tirofiban)
- Thrombolytics: Alteplase (systemic, intraventricular)
- <u>Pump exchange</u>

Reason to be moved into high-priority transplant waiting status, because of the increased mortality associated with this complication.



Conclusions

VA ECMO

Imperfect but viable option

Improvement in cannula design

Eliminate need for back flow cannulation (peripheral)

Less thrombotic risks/heparin

Technological solutions to:

minimise after load

increase/promote AV opening

minimise LV distension/minimally invasive LV venting strategies



Conclusions

Improve durability of short term VADs ?Impella/ access/position/haemolysis More options for long term biventricular support Long term RVAD Anticoagulation ?targets





Thank you







Harefield experience

HVAD mortality ~5-7% at D60

BTT main indication

DT not funded by NHS > number will increase

Private LVAD increasing

TOE in Intensive Care

- **1.Assist in fluid management**
- 2.Diagnose cardiac tamponade
- **3.Assess LV decompression**
- **4.Guide fixed speed selection**
- **5.Identify RV dysfunction**
- 6.Identify valvular pathology/ AV opening
- 7.Diagnose "suction events"/volume status/collections
- 8.<u>Guide therapy</u>



RV failure

When medical treatment fails....

Mechanical support

The need for an RVAD is associated with worse outcomes, but elective implantation of an RVAD correlates with better long-term survival than does an emergency implantation.

RVAD implant at the time of LVAD implant also improves survival to transplant compared with delayed RVAD insertion

Table 1. Hemodynamic Formulas to Assess Right Ventricular Function

Hemodynamic Formulas to Assess RV Function					
Cardiac filling pressures	RAP/PCWP	>0.63 (RVF after LVAD) ¹³ >0.86 (RVF in acute MI) ³⁰			
PA pulsatility index (PAPi)	(PASP-PADP)/RAP	<1.85 (RVF after LVAD) ³¹ <1.0 (RVF in acute MI) ³²			
Pulmonary vascular resistance	mPAP-PCWP/CO	>3.6 (RVF after LVAD) ¹⁵			
Transpulmonary gradient	mPAP-PCWP	Undetermined ³³			
Diastolic pulmonary gradient	PADP-PCWP	Undetermined ^{33,34}			
RV stroke work	(mPAP-RAP)× SV×0.0136	<15 (RVF after LVAD) ¹⁵ <10 (RVF after acute MI) ³⁵			
RV stroke work index	(mPAP–RAP)/SV index	<0.3–0.6 (RVF after LVAD) ^{13,31}			
PA compliance	SV/(PASP-PADP)	<2.5 (RVF in chronic heart failure) ³⁶			
PA elastance	PASP/SV	Undetermined ³⁷			



RV failure

PA catheter +/- TOE

Goal CVP<15mmHg

– CVP >15: furosemide, RRT or decrease LVAD flow
– CVP <10: fluid boluses

Reduce PVR (avoid hypoxia, hypercarbia, and acidosis) **RV dysfunction +/- high PVR**: adrenaline, milrinone early

- <u>If high PVR (>3 Wood Units</u>): pulmonary vasodilator (iNO, sildenafil, epoprostenol, iloprost)

- <u>If low SVR (<800 dyn/s/cm-5</u>): inotropic support + vasopressors to increase perfusion of the RV (MAP-CVP)



Options

- 1. RVAD CentriMag (Levitronix)
- 2. Percutaneous RVAD > Protek Duo[®]
- 3. Impella RP[®]
- **4. ECMO**
- **5. TAH**

Requirement of an RV assist device (RVAD) or >14 consecutive days of intravenous (IV) inotropic support, has an estimated prevalence of 13% to 44% and is associated with significant morbidity and mortality



Anticoagulation

- Anticoagulation therapy is required
- Starting anticoagulation too early is a common mistake
- Adequate haemostasis should be achieved before anticoagulation is initiated
- Modification of the anticoagulation regimen may be required in the face of bleeding.

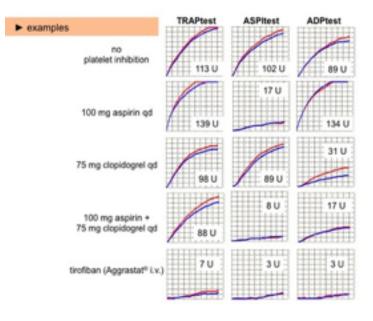
Titrating Anticoagulation

-Unfractionated Heparin > infusion APTT/Anti Xa every 6h

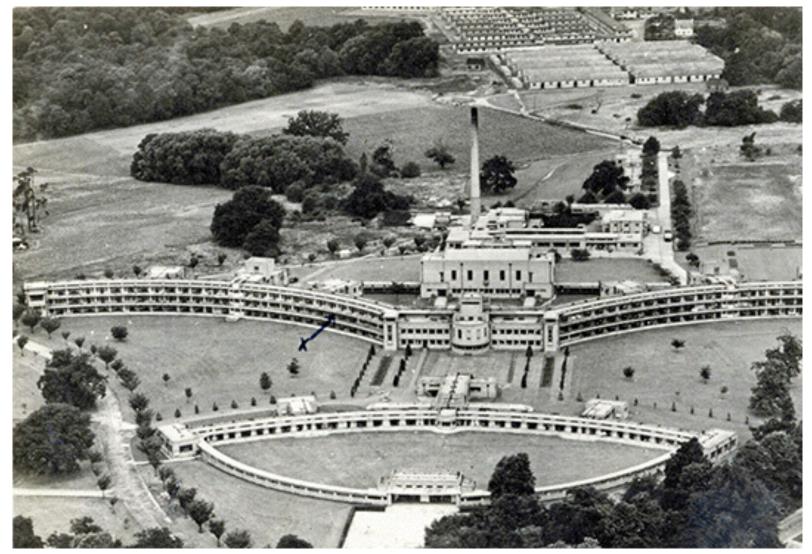
-Aspirin > We titrate aspirin dose

Multiplate[®]platelet function analysis









a.hurtadodoce@rbht.nhs.uk