Devices for Cardio-Renal Failure
The Future of HF and Shock Therapies

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Cardio-Renal Syndrome

Type 1: acute cardio-renal syndrome
Acute HF leading to AKI
Altered cardiac and/or renal haemodynamics might be of particular importance

Type 2: chronic cardio-renal syndrome
Chronic HF leading to progressive and permanent CKD
Accelerated renal cell apoptosis and replacement fibrosis might be of particular importance

Type 3: acute reno-cardiac syndrome
AKI causing acute HF
Salt and water imbalance, uraemia-induced effects and neuro-hormonal dysregulation might be key in this setting

Type 4: chronic reno-cardiac syndrome
CKD leading to chronic HF and CKD progression
CKD-induced myopathy might be of particular importance in this setting

Type 5: secondary cardio-renal syndrome
Systemic insult (e.g. in severe sepsis and/or septic shock)
Microcirculatory dysfunction, altered innate and adaptive immune responses and cytokine release, and other effects result in simultaneous organ injury

Schefold and von Haeling Nat Rev 2016
How Heart Failure Promotes Renal Dysfunction

Nephronic factors

- NSAIDs
- Thiazides
- Tubular and glomerular injury
- MRA
- Loop diuretics
- Interstitial fibrosis
- Increased renal interstitial pressure
- Decreased urine output
- Increased renal venous pressure
- Reduced RBF, intravascular volume depletion
- Cortex Medulla

B Glomerular factors

- Low filtration gradient
- Increased renal interstitial pressure
- Hypertension
- Diabetes
- Atherosclerosis
- NSAIDs
- RBF ↓ and GFR ↓
- ACEi/ARB
- RBF ↑, FF ↓ and GFR ↓

Eur H J 2014
Diuretic Resistance and Renal Autoregulation

1. Reduced CO → Reduced Effective Circulating Volume
2. NSAIDs or ACE-I / ARB overdosing
3. Aggressive afterload reduction with impaired renal autoregulation

Renal Autoregulation:
- Myogenic Response
- Tubulo-interstitial Feedback
Impact of increased Renal Arterial Pressure

<table>
<thead>
<tr>
<th>Renal Artery Pressure (mm Hg)</th>
<th>Renal Vein Pressure (mm Hg)</th>
<th>Renal Blood Flow (cc/min)</th>
<th>Intrarenal Venous Pressure (mm Hg)</th>
<th>Over-all Renal Resistance mmHg/cc/min</th>
<th>Intrarenal Resistance mmHg/cc/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>80</td>
<td>0</td>
<td>100</td>
<td>8</td>
<td>$\frac{80}{100} = 0.80$</td>
<td>$\frac{80-8}{100} = 0.072$</td>
</tr>
<tr>
<td>180</td>
<td>0</td>
<td>139</td>
<td>80</td>
<td>$\frac{180}{139} = 1.29$</td>
<td>$\frac{180-80}{139} = 0.72$</td>
</tr>
</tbody>
</table>

Hinshaw and Worthen Circ Res 1961
Increased Renal Venous Pressure Reduces GFR

Mullens and Tang JACC 2009
Increased Ureteral Pressure Decreases RBF

Renal Preload (Arterial)

Renal Afterload (Venous)

Renal Afterload (Non-vascular)

Hinshaw and Worthen Circ Res 1961
Ultrafiltration in Decompensated Heart Failure with Cardiorenal Syndrome

- Ultrafiltration (N=92)
- Pharmacologic therapy (N=94)

Creatinine Increase (mg/dl)

Weight Loss (lb) vs. Weight Gain (lb)

Creatinine Decrease (mg/dl)

P=0.003

Bart and Braunwald et al NEJM 2012
Impact of Acute MCS Devices on Renal Function

Increased Renal Preload
- IABP
- Impella
- VA-ECMO

Reduce Renal Afterload
- Impella RP
- Tandem RVAD
- VA-ECMO

Neuromodulation/Ablation
Minimal Impact of IABP on Renal Blood Flow

Unless your IABP is out of position

Swartz and Pennington Ann Thor Surg 1992
Signal of Increased RBF with Impella

No change in RBF with IABP
Signal of increased RBF with Impella 2.5

Renal Blood Flow

Baseline  IABP  Impella®  Combined  No support, 10 min  No support, 60 min

mL/min

310  261  303  289  245  218

p < 0.05

Moller-Helgestadt Int J Card 2015
Emerging Devices for Cardio-Renal Syndrome 1

Cardiac Goals of Therapy:
1. Reduce LV Afterload
2. Reduce LV Stroke Work
3. Increase Cardiac Output

Renal Goals of Therapy
1. Increase Renal Blood Flow
2. Increase Urine Output
3. Avoid RV / Pulmonary Loading
Emerging Aortic Platforms for CRS Type 1

Potential Advantages of Aortic Pumps
1. Extracardiac positioning
2. Reduce LV Afterload & Work
3. Enhance Systemic Perfusion
4. Increase Renal Perfusion & UOP
Emerging Aortic Platforms for CRS Type 1

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Advantages of CF-Aortic Pumps
1. Cardiac-Renal Uncoupling
2. Less dependent on LV function
3. Ambulatory/Outpatient Viability
The Tufts Cardiovascular Center for Research and Innovation

The CVCRI Innovation Engine

MCRI  
BENCH  
Basic Science

SIRL  
Translational Lab

CVCRI  
BEDSIDE  
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Discovery to Development  
Knowledge to Practice

Clinical Excellence  
Preclinical Testing  
Fundamental Discoveries  
Preclinical Trials  
Novel Clinical Trials
Micro-Axial Pump
Stentless Anchoring Tines
18Fr Catheter Delivery System
Improved Hemodynamics With a Novel Miniaturized Intra-aortic Axial Flow Pump in a Porcine Model of Acute Left Ventricular Dysfunction

Farshad Raissi Shabari,* Joggy George,* Michael P. Cuchiara,† Robert J. Langsner,† Jason J. Heuring,† William E. Cohn,* Benjamin A. Hertzog,† and Reynolds Delgado*
Retractable Propeller
Retractable Stent Cage
13Fr Catheter Delivery System
Emerging Devices for Cardio-Renal Syndrome 1

Procyrion

SECOND HEART ASSIST
REVERSING CARDIO-RENAL DYSFUNCTION

Magenta Medical

Paragate Medical

Revamp Medical

White Swell

Renal Preload (Arterial)

Renal Afterload (Venous)

Renal Afterload (Non-vascular)
Thank you

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To Learn More about Acute MCS & Hemodynamics

Interventional Heart Failure

August 24-25, 2017: Barcelona, Spain