Case Study: Heart Transplantation

Kim Anderson, MD, FRCPC

Dr. Anderson is an adult cardiologist specialized in advanced heart failure, transplantation and mechanical circulatory support. She obtained her medical diploma in 2007 at the Université de Montréal. She trained in internal medicine and adult cardiology also in Université de Montréal. She then completed a 2 years clinical fellowship in Heart Failure and Transplantation in Toronto at University Health Network, followed by a Master in Clinical Epidemiology and Health Care Research at University of Toronto. She is now an Assistant Professor at the Halifax Infirmary Hospital affiliated with Dalhousie University here in Halifax, in the Heart Failure and Transplantation group.

References:
## Disclosure

**Kim Anderson, MD, FRCPC**

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Consultant/Speaker Fees:
- Novartis
Heart Transplant Case
2017 CST-Astellas Canadian Transplant Fellows Symposium

Kim Anderson, MD FRCPC
QEII-Halifax Infirmary Hospital, Dalhousie University
Nova Scotia
Clinical case: Mrs. H

- 56 yo W. BG B, 68 kg
- Dilated cmp with biV failure, most likely post-myocarditis. Dx 2005
- Embolic myocardial infarction (LV thrombus) 08-2010
- Appropriate choc for VF May 2013
- RHC 1 month pre-Tx: RA 2, RV 18/0, PA 19/2/9, wedge 1, MVO2 66%
- cPRA 2013: 99/54%-Listed status 4s
- No end organ significant dysfunction
Heart transplant 2014

- Virtual cross-match negative
- OR at 7h00, CVICU at 13h40 same day
- Ischemic time: 164 minutes
- Solumedrol 1g on call to OR then 0.5g at cross-clamp
- Thymoglobulin 75 mg IV started at 15h30
Mrs. H Donor

- 50 yo, 65 kg, 165 cm
- Risk factors for coronary artery disease +
- Angiogram: very minor OM origin disease, not palpable at time of Tx
- « Good looking donor heart »
- Small area LAD with some plaque seen by surgeon at time of Tx (but lumen OK on angio)
• Surgery and immediate post-op:
  – Intra-op TEE: BiV function OK
  – Bleeding + and vasoplegia
  – Milrinone 0.25, epi 0.07 to 0.09, levo 0.06
  – AV pace 100 bpm
  – PA 24/10, RA 5-10, CO 2.7/CI 1.8, MAP 65 (90/60),
    urine output 30 cc/h, lactate 6.7 (17h00)
  – Extubated at 16h00 same day then on 5L O2
The retrospective cross-match

FLOW XM Auto:
  T Flow -, B Flow –

FLOW XM Allo:
  T Flow -, B Flow +
No, there is no DSA (ouf!)

STAT typing of her HLA antibodies with serum post-transplant comparison to HLA typing of donor:

NO DSA
Acute rejection... or what else?

- Acute RV dysfunction?
- Pulmonary hypertension?
- Bradyarhythmia/ sinus node dysfunction?
- Surgical complication?
- Myocardial infarction?
  - Underestimated CAD of donor?
  - Air or thrombi emboli?
  - Plaque rupture?
- Marginal donor (a « bad heart »)?
Primary graft dysfunction (heart)

ISHLT CONSENSUS

Report from a consensus conference on primary graft dysfunction after cardiac transplantation

• No discernable cause for graft dysfunction
• LV or RV or BiV dysfunction
• ≤ 24 h after the transplant surgery

Kobashigawa. JHLT 2014;33.
Pre-concensus survey\textsuperscript{1}:

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Primary Graft Dysfunction in Heart Transplantation, Results of Pre-conference Online Survey (47 centers participating) January 2013–March 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Total number of transplant patients at all participating centers was 9,901 with 733 patients thought to have PGD—rate 7.4%</td>
<td></td>
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<tr>
<td>• 30-day mortality was 30% and 1-year mortality was 34.6%</td>
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<tr>
<td>• Most common causes of death for 30-day mortality: Multiorgan failure (70%), graft failure (20%), and sepsis (10%)</td>
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<tr>
<td>• Definition parameters for PGD:</td>
<td></td>
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<tr>
<td>◦ 79% of centers felt that LVEF ( \leq 40% ) was a criteria of PGD</td>
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<tr>
<td>◦ 68% of centers felt that a time frame of within 24 hours should be used to define PGD</td>
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<tr>
<td>◦ 70% of participating centers felt that mechanical support is a mandatory criteria for the definition of PGD</td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{1}Kobashigawa. JHLT 2014;33.  \textsuperscript{2}Mark. Transplantation 2010;90

• Death or reTx: 2.5%
• 23.4% of all deaths 90 days post-Tx\textsuperscript{2}
## Definitions - severity

### Table 6  Definition of Severity Scale for Primary Graft Dysfunction (PGD)

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>1. <strong>PGD-Left ventricle (PGD-LV):</strong></td>
<td><strong>Mild PGD-LV:</strong> One of the following criteria must be met:</td>
</tr>
<tr>
<td></td>
<td><strong>LVEF ≤ 40%</strong> by echocardiography, or</td>
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<tr>
<td></td>
<td><strong>Hemodynamics with RAP &gt; 15 mm Hg, PCWP &gt; 20 mm Hg, CI &lt; 2.0 L/min/m²</strong> (lasting more than 1 hour) requiring low-dose inotropes</td>
</tr>
<tr>
<td></td>
<td><strong>Moderate PGD-LV:</strong> Must meet one criterion from I and another criterion from II:</td>
</tr>
<tr>
<td></td>
<td>I. <strong>One</strong> criteria from the following:</td>
</tr>
<tr>
<td></td>
<td><strong>Left ventricular ejection fraction ≤ 40%,</strong> or</td>
</tr>
<tr>
<td></td>
<td><strong>Hemodynamic compromise with RAP &gt; 15 mm Hg, PCWP &gt; 20 mm Hg, CI &lt; 2.0 L/min/m², hypotension with MAP &lt; 70 mm Hg</strong> (lasting more than 1 hour)</td>
</tr>
<tr>
<td></td>
<td>II. <strong>One</strong> criteria from the following:</td>
</tr>
<tr>
<td></td>
<td>i. <strong>High-dose inotropes</strong> — Inotrope score &gt; 10⁶ or</td>
</tr>
<tr>
<td></td>
<td>ii. <strong>Newly placed IABP</strong> (regardless of inotropes)</td>
</tr>
<tr>
<td></td>
<td><strong>Severe PGD-LV</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Dependence on left or biventricular mechanical support including ECMO, LVAD, BiVAD, or percutaneous LVAD. Excludes requirement for IABP.</strong></td>
</tr>
<tr>
<td>2. <strong>PGD-right ventricle (PGD-RV):</strong></td>
<td>Diagnosis requires either both i and ii, or iii alone:</td>
</tr>
<tr>
<td></td>
<td>i. <strong>Hemodynamics with RAP &gt; 15 mm Hg, PCWP &lt; 15 mm Hg, CI &lt; 2.0 L/min/m²</strong></td>
</tr>
<tr>
<td></td>
<td>ii. <strong>TPG &lt; 15 mm Hg</strong> and/or <strong>pulmonary artery systolic pressure &lt; 50 mm Hg</strong>, or</td>
</tr>
<tr>
<td></td>
<td>iii. <strong>Need for RVAD</strong></td>
</tr>
</tbody>
</table>

Kobashigawa. JHLT 2014;33.
Pathogenesis - from donor to recipient
Brain death of the donor:

++ NE released
→ Ca\(^{2+}\) overload
→ Autophagy, apoptosis, necrosis

Donor resuscitation (IV catecholamines)

Beta-receptor signaling desensitization

→ Contractility

↓ T3, cortisol & insulin

Older donor:
- CAD unrecognized
- LVH (HTN)
- ↓ cardioprotective mechanisms
Hypothermic organ preservation:

- Lactic acidosis
  - ↑ Intracellular Ca
  - MPTP
  - Pro-apoptotic factors
    - Mitochondrial swelling & necrosis
      - ↓ Contractility

- Na⁺/K⁺ ATPase stopped
  - Cellular swelling
  - Progressive ischemic injury

- Incomplete ↓ in metabolism
In the recipient:

- Reperfusion
- O₂ free radicals
- Enzymes disruption

- Reperfusion
- Ca²⁺
- MPTP

- MCS (VAD)
- Ischemic time

- Systemic inflammatory response
- Peripheral vasodilatation
- «Hostile environment »
## Autopsy evaluation

<table>
<thead>
<tr>
<th>Pathologic diagnosis</th>
<th>Autopsy results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rejection</td>
<td>7 (%)</td>
</tr>
<tr>
<td>Reperfusion injury/ischemia</td>
<td>48 (%)</td>
</tr>
<tr>
<td>Possible freeze injury</td>
<td>7</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
<td>3.4</td>
</tr>
<tr>
<td>Myocyte necrosis</td>
<td>28</td>
</tr>
<tr>
<td>Antibody-mediated rejection (C4D staining; CD68)</td>
<td>3.4</td>
</tr>
<tr>
<td>Multifocal edema and/or hemorrhage</td>
<td>14</td>
</tr>
<tr>
<td>Aortic tear</td>
<td>3.4</td>
</tr>
<tr>
<td>Infection</td>
<td>3.4</td>
</tr>
</tbody>
</table>

Kobashigawa. JHLT 2014;33.
Mrs. H:
Hemodynamic support and IS

- Epi, norepi and isoproterenol 48h
- Milrinone 7 days
- Plex x 5
- Thymoglobuline 225
- Solumedrol 1.5g total then pred 40mg OD
- Cellcept 1g bid+ Tac 2g bid
Pharmacologic management of PGD

- Low-dose inotropes
- Pulmonary vascular vasodilators
- Nitric oxide
- Specific treatment for PGD? or standard of care after cardiac transplantation?
Mechanical management of PGD

Right, Left or Both ventricles?

With oxygenator or not?

Central or peripheral cannulation?
Mrs. H: Graft function evolution (Echo)

<table>
<thead>
<tr>
<th>Days post-Tx</th>
<th>LV function</th>
<th>RV function</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;20%</td>
<td>Mild to moderate dysfct</td>
</tr>
<tr>
<td>3</td>
<td>40-59%</td>
<td>Mild to moderate dysfct</td>
</tr>
<tr>
<td>7</td>
<td>40-59%</td>
<td>Low normal</td>
</tr>
<tr>
<td>10</td>
<td>Normal</td>
<td>Low normal</td>
</tr>
</tbody>
</table>
Biopsy #1 (1 week post-Tx)

- No cellular rejection
- Subendocardial areas of recent ischemic (reperfusion injury) damage
  - Likely related to the ischemic interval and reperfusion
Conclusions

• Recognize primary graft dysfunction in heart transplant recipient
  • Uni or BiV dysfunction
  • ≤ 24h post-Tx
  • No other discernable cause

• Rate ± 7% (2.5-25%), mortality ± 30%

• Understand the pathophysiology of this entity
  • Ischemic-reperfusion
  • Multiple risk factors
    (donor, recipient, surgery and organ preservation)
BACK-UP SLIDES
Adult Heart Transplants

Relative Incidence of Leading Causes of Death

(Deaths: January 2006 – June 2012)

Since only leading causes of death are shown, sum of percentages for each time period is less than 100%
Cardiac Size and Sex-Matching in Heart Transplantation

Size Matters in Matters of Sex and the Heart

Robert M. Reed, MD,* Giora Netzer, MD, MSCE,*† Lawrence Hunsicker, MD,‡
Braxton D. Mitchell, PhD,§∥ Keshava Rajagopal, MD, PhD,¶ Steven Scharf, MD, PhD,*
Michael Eberlein, MD, PhD#

Baltimore, Maryland; and Iowa City, Iowa

[1] Predicted left ventricular mass (g)

\[ \text{Predicted left ventricular mass} = a \cdot \text{Height}^{0.54} \cdot \text{Weight}^{0.61} \]

where \( a = 6.82 \) for women and \( 8.25 \) for men; and

[2] Predicted right ventricular mass (g)

\[ \text{Predicted right ventricular mass} = a \cdot \text{Age}^{-0.32} \cdot \text{Height}^{1.135} \cdot \text{Weight}^{0.315} \]
A-Survival by difference in weight
B-Survival by difference in predicted heart size
D-Survival by gender matching

Reed. JACC Heart Fail. 2014;2.
These « others » antibodies...

A Reevaluation of the Role of IgM Non-HLA Antibodies in Cardiac Transplantation
Smith, John D.¹; Hamour, Iman M.¹; Burke, Margaret M.²; Mahesh, Balikrishnan³; Stanford, Rachel E.¹; Haj-Yahia, Saleem¹ Magdi H.³; Banner, Nicholas R.¹; Rose, Marlene L.³,⁵

- Retrospective 616 patients HTx, UK 1 center (91-2003)
- 59/616 non-HLA IgM+ and no HLA-Ab
- 25/59 died ≤ 1 year
  - 6 from PGD; 10% mortality associated to PGD

55.9% survival 1st year vs 75.8% no antibody

These « others » antibodies...

**TABLE 3.** Multivariate analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter estimate</th>
<th>Standard error</th>
<th>P</th>
<th>Hazard ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgM non-HLA</td>
<td>0.8583</td>
<td>0.2281</td>
<td>0.0002</td>
<td>2.359</td>
</tr>
<tr>
<td>Recipient age(^a)</td>
<td>0.02908</td>
<td>0.00863</td>
<td>0.0008</td>
<td>1.030</td>
</tr>
<tr>
<td>Immunosuppression 2 (is better than 1)</td>
<td>−0.3926</td>
<td>0.1820</td>
<td>0.031</td>
<td>0.675</td>
</tr>
<tr>
<td>NYHA class IV (is worse)</td>
<td>0.3357</td>
<td>0.0996</td>
<td>0.0007</td>
<td>1.399</td>
</tr>
<tr>
<td>Male recipient, female donor (is worse)</td>
<td>0.3517</td>
<td>0.1620</td>
<td>0.030</td>
<td>1.421</td>
</tr>
<tr>
<td>Ischemic time</td>
<td>0.00261</td>
<td>0.00125</td>
<td>0.037</td>
<td>1.003</td>
</tr>
<tr>
<td>Donor age(^{a,b})</td>
<td>−0.05600</td>
<td>0.02840</td>
<td>0.049</td>
<td>0.523</td>
</tr>
<tr>
<td>Donor age(^{a,b,c})</td>
<td>0.00104</td>
<td>0.0004349</td>
<td>0.017</td>
<td>1.127</td>
</tr>
</tbody>
</table>

Is AMR possible without DSA?

- 17 HTx recipients with late AMR
  - 15 DSA +
  - 2 no DSA but anti-vimentin IgG+ ¹

- 37 HTx recipients with AMR
  - 15/37 no DSA
  - 19 of AMR tested for MICA Ab; 3/19 DSA- MICA-²

¹ Rose. Human Immunology 2013.
² Zhang. Transplantation 2011;91.
Characterization of immune responses to cardiac self-antigens myosin and vimentin in human cardiac allograft recipients with antibody-mediated rejection and cardiac allograft vasculopathy

Dilip S. Nath, MD, Haseeb Ilias Basha, MD, Venkataswarup Tiriveedhi, MD, PhD, Chiraag Alur, BS, Donna Phelan, BA, Gregory A. Ewald, MD, Nader Moazami, MD, and Thalachallour Mohanakumar, PhD

Table 2 Characteristics of AMR\(^+\) Patients

<table>
<thead>
<tr>
<th>Pt</th>
<th>Symptoms</th>
<th>Abnormal echocardiography</th>
<th>Inotropes</th>
<th>Histology</th>
<th>Immunopathology</th>
<th>CD4</th>
<th>CD68</th>
<th>DSA</th>
<th>Anti-MYO</th>
<th>Anti-VIM</th>
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<tbody>
<tr>
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Pt, patient.

Nath. JHLT 2010;29.
Biomarkers of PGD

- The role of biomarkers in PGD remains controversial.
- Brain death -> Biomarkers↑
- Can we use them to define whether a heart is acceptable or not?
- Can they predict long-term outcomes in recipients with regard to PGD?
What to choose?

- A **composite of the biomarkers** has been proposed as a better method of predicting PGD.
- Combination of **NT-proBNP and cTnT** \((n=63)\) for an early diagnosis of LV systolic dysfunction, ↑**sensitivity** \((0.78–0.9 \text{ VS } 1.0)^1\)
- Combination of **PCT and cTnT** in donors is a better predictor of early graft dysfunction in recipients than lone biomarkers.
- More research is needed in this area to try different combinations.

2. Potapov et al; Int J Cardiol 2003
Levosimendan *not available in Canada

- a calcium sensitizer
- PGD - despite inotropic support with **epinephrine** > 0.1 µg/kg/min & **milrinone** > 0.3 µg/kg/min.
- 30-day survival rate was 93%.
- Subsequent 3-year follow-up of this study showed a significantly lower 1-year and 3-year survival rate.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-levosimendan</th>
<th>24 hours</th>
<th>48 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection fraction, %</td>
<td>27 ± 4</td>
<td>38 ± 8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>45 ± 10&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Cardiac output, liter/min</td>
<td>5.2 ± 0.6</td>
<td>6.2 ± 0.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.9 ± 0.6</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>67 ± 10</td>
<td>80 ± 9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>83 ± 9&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>MPAP, mm Hg</td>
<td>31 ± 7</td>
<td>24 ± 6</td>
<td>23 ± 6</td>
</tr>
<tr>
<td>Epinephrine,&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.3 ± 1.2</td>
<td>0.4 ± 0.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.3 ± 0.4&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Norepinephrine,&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.0 ± 0.8</td>
<td>0.3 ± 0.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.15 ± 0.2&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>mg/hours</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milrinone,&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.5 ± 0.6</td>
<td>0.9 ± 0.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.8 ± 0.9&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Iloprost,&lt;sup&gt;c&lt;/sup&gt;</td>
<td>54 ± 10</td>
<td>43 ± 11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>32 ± 20&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

MAP, mean arterial pressure; MPAP, mean pulmonary arterial pressure.

<sup>a</sup>Value of p < 0.05 for comparison to the pre-levosimendan figures.

<sup>b</sup>Maximum dose used on the respective day.

<sup>c</sup>Cumulative dose used on the respective day.

Weis et al; J Heart Lung Transplant 2009
90 patients
54 – ECMO
8 - assist devices
  2 biventricular assist devices
  6 centrifugal RV assist
  28 were on maximal inotropes alone
Medically treated – 46% survival
Mechanical support -25% survival
ECMO - 50% survival

1 year survival:
  PGD 37%
  No PGD 78%
28 patients - acute RV failure
   - 13 pt - ECMO
   - 15 pt - RVAD

Patient survival was similar

Graft survival was markedly improved (7 compared with 1)

Retransplantation was less often required
   (1 compared with 6)

Weaning rates were significantly higher
   (10 compared with 2)
• 38 patients from 2003 to 2008
• CentriMag device for PGD survival was 50% at 30 days and 32% at 1 year.
• Earlier implantation of the device after transplant appeared to correlate with improved survival.
• All survivors were supported with the device for no more than 30 days.
Organ preservation

• How can we reduce the incidence of PGD and increase graft and patient survival?
  – Reduction of the ischemic time
  – Better preservation of older donor hearts (potentially)
Ex vivo perfusion

- Potentially avoid the limitation of cold storage by providing warm blood perfusion to the donor organ.
- Better preservation of the endothelium integrity
- Reduce the incidence of coronary artery vasculopothy (CAV)?
- Increase the donor heart utilization and weeding out sub-optimal organs
Ex vivo perfusion

• PROCEED II is a global clinical trial that compares standard cold storage of donor hearts to warm oxygenated blood perfusion using the Organ Care System.
• 92 patients (43 OCS and 49 SOC patients)
• Total cross clamp-time:
  – OCS group $5.4 \pm 1.4$ hours↑
  – SOC group $3.4 \pm 1.1$ hours
• Total ischemic time:
  – OCS group $1.8 \pm 0.4$ hours↓
  – SOC group $3.4 \pm 1.1$ hours
• No statistical differences in: 30-day patient survival, 30-day graft survival, all reported SAEs, reported cardiac SAEs, early graft dysfunction, and episodes of rejection.
• Development of more effective donor management and donor heart preservation strategies may reduce the incidence of PGD.

S. Ullah, Perfusion 2006
Effect of intraoperative blood cardioplegia

• Single cold flush preservation is the gold standard
• Reliable protection as long as total ischemic time of the heart does not exceed 3 to 4 hours and donor age is < 40 years.
• Clinical reality today often challenges these limits due to changes in the donor population and altered non-regional organ allocation.
Improve preservation-intraoperative blood cardioplegia

- Group 1: standard filtrated cold University of Wisconsin (UW) single flush perfusion - served as the historical control.

- Group 2: after initial UW preservation, additional Buckberg cold blood cardioplegia was administered antegrade by aortic root after completion of each anastomosis or at least every 20 minutes during implantation.

- Group 3: preservation was as in Group 2, but starting with graft implantation, perfusate of extracorporeal circulation blood cardioplegia was leucocyte-depleted by $40 \times 10^{-6}$ by inline filtration.
Improve preservation-intraoperative blood cardioplegia

• PGD:
  – Group 1 5.2%
  – Group 2 4.1%
  – Group 3 0% ($p < 0.05$ Group 3 vs Group 1).

• epinephrine use & IABP support significantly ↓ in Group 3 (0.2 vs 0.3 vs. 0.3 $\mu$g/kg/min and 2% vs 10% vs 17% in Group 3 vs Group 2 vs Group 1, respectively; $p < 0.05$).

• Need for permanent pacemaker implantation before hospital discharge was ↓ in Group 3 (0% vs 2.0% vs 5.5% in Group 3 vs 2 vs 1, respectively; $p < 0.05$).
# Size and sex mismatch

## Table 2: Survival by Quantiles of Difference in pHM

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted Models</th>
<th>Adjusted Models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Survival 1 yr</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HR (95% CI)</td>
<td>HR (95% CI)</td>
</tr>
<tr>
<td></td>
<td>p Value</td>
<td>p Value</td>
</tr>
<tr>
<td>1 (most-undersized donor)</td>
<td>1.27 (1.13–1.43)</td>
<td>1.25 (1.02–1.54)</td>
</tr>
<tr>
<td>2</td>
<td>1.10 (0.98–1.24)</td>
<td>1.14 (0.95–1.36)</td>
</tr>
<tr>
<td>3</td>
<td>0.96 (0.85–1.09)</td>
<td>1.02 (0.85–1.23)</td>
</tr>
<tr>
<td>4 (best fit)</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>5</td>
<td>1.00 (0.88–1.13)</td>
<td>1.06 (0.89–1.26)</td>
</tr>
<tr>
<td>6</td>
<td>1.06 (0.94–1.19)</td>
<td>1.03 (0.86–1.24)</td>
</tr>
<tr>
<td>7 (most-oversized donor)</td>
<td>1.08 (0.96–1.22)</td>
<td>0.95 (0.78–1.16)</td>
</tr>
<tr>
<td>MR/MD</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>MR/FD</td>
<td>1.32 (1.22–1.43)</td>
<td>1.00 (0.85–1.17)</td>
</tr>
<tr>
<td>FR/FD</td>
<td>1.17 (1.06–1.29)</td>
<td>1.26 (1.08–1.47)</td>
</tr>
<tr>
<td>FR/MD</td>
<td>1.17 (1.06–1.30)</td>
<td>1.62 (1.36–1.92)</td>
</tr>
<tr>
<td>FR/MD†</td>
<td>1.00 (0.88–1.14)</td>
<td>1.28 (1.04–1.57)</td>
</tr>
</tbody>
</table>

Reed. JACC Heart Fail. 2014;2.
PLEX in PGD; reasonable if any suspicion of AMR

Steroid Pulse Therapy Combined with Plasmapheresis for Clinically Compromised Patients after Heart Transplantation


Chou. Transplantation Proceedings 2012;44.
PLEX in PGD; reasonable if any suspicion of AMR

PLEX in PGD; also done in other organs Tx

Comparison of the Molecular Adsorbent Recirculating System and Plasmapheresis for Patients With Graft Dysfunction After Liver Transplantation


- 31 OLT with graft dysfunction 2002-2007; MARS vs PLEX
- 90 days survival not different: 53% vs 56%
- Cause of graft dysfunction:
  - MARS: PGD 2, Rejection 7, Ischemia 5, HBV 1
  - PLEX: PGD 3, Rejection 6, Ischemia 7

Lee. Transplantation Proceedings 2012;42.