The authors would like to acknowledge the surgery were being treated with targeted therapy. Months post surgery. Only 17% of patients at 12 months post 10 years was 74%. There was a significant functional and hae-

Conclusion The conditional survival of a subset of the cohort at 10 years was 74%. There was a significant functional and hae-

Acknowledgements The authors would like to acknowledge the pulmonary hypertension centres in the UK. “This research was supported by the National Institute for Health Research (NIHR) Cambridge Biomedical Research Centre”.

Abstract S47 Table 1. Median treatment effects on PVR and CI

<table>
<thead>
<tr>
<th>PVR, dyn·sec/cm²</th>
<th>Cl, L/min/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>(relative benefit* to placebo expressed in %)</td>
<td>Macitentan 3mg p-value</td>
</tr>
<tr>
<td>All</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Treatment Naive</td>
<td>-19.9 (-34.2,0.8)</td>
</tr>
<tr>
<td>Treated</td>
<td>-34.4 (-45.6,22.3)</td>
</tr>
<tr>
<td>FC UI</td>
<td>-35.2 (-49.2,21.6)</td>
</tr>
<tr>
<td>FC IMV</td>
<td>-21.8 (37.8,8.0)</td>
</tr>
</tbody>
</table>

Median (95% CI) placebo-corrected change from baseline and Wilcoxon test p-values, *based on the log of Month 6/baseline values

only chance of cure. Data on the long term survival after PEA are limited.

Method All patients who have undergone a PEA for CTEPH at Papworth hospital were included between January 1997 and November 2012. Patients who had a re-do operation were excluded. Pre- and post-operative data on haemodynamics, exercise capacity, functional class and targeted PAH therapies taken were obtained from our PH database and from other UK PH centres. The long-term survival of patients who followed for follow-up at 3 months post PEA was determined using the NHS spine summary care record tracking system. Overseas patients were censored when last seen.

Results 880 patients underwent PEA over the 15 year period. The mean age was 57 (range 15–84) and 53% were male. The majority (89%) were in WHO functional class 3 or 4 prior to surgery with an average mean pulmonary artery pressure (mPAP) of 47 mmHg and PVR of 795 dynes. 65% of patients were taking at least 1 targeted therapy as a “bridge to surgery”. Post surgery the majority of patients (86%) were in WHO functional class 1 or 2 at the 12 month follow-up with only 17% taking targeted therapy. There was a reduction in the average mPAP to 27 mmHg and PVR to 308 dynes by 12 months. The 10 year conditional survival post PEA of the first 314 patients from the cohort (Freed et al. J Thorac Cardiovasc Surg, 2011;141:383–7) was 74%.

Conclusion The conditional survival of a subset of the cohort at 10 years was 74%. There was a significant functional and haemodynamic improvement in the majority of patients at 12 months post surgery. Only 17% of patients at 12 months post surgery were being treated with targeted therapy.

Acknowledgements The authors would like to acknowledge the pulmonary hypertension centres in the UK. “This research was supported by the National Institute for Health Research (NIHR) Cambridge Biomedical Research Centre”.
physiological index of myocardial reserve and thus at inefficient ratios, may predispose to reduced exercise capacity.

**Methods** Using RV conductance catheterisation and contemporaneous incremental cardiopulmonary exercise testing, we evaluated Ees/Ea against peak VO₂ in twenty patients with pulmonary vascular disease. Ees/Ea was compared with haemodynamic predictors of exercise capacity obtained from standard right heart catheterisation.

**Results** Resting Ees/Ea, absolute peak VO₂ and predicted peak VO₂ were 0.86 ± 0.40, 19.6 ± 6.7mL/kg/min and 88 ± 23% respectively. Univariable predictors of absolute peak VO₂ were patient gender, NYHA class, mean right atrial pressure, mean pulmonary artery pressure, cardiac index, conductance RV stroke volume and Ees/Ea (all p < 0.10). On bivariate analysis, the predictive value of Ees/Ea improved following adjustment for RV stroke volume (p = 0.03) but not for mean RA pressure (p = 0.21). Only Ees/Ea related linearly to percent predicted VO₂ (R² = 0.32, p = 0.01). RV diastolic decay (Δdp/dtmin ) showed good correlation with O₂ pulse evolution (r = 0.62, p < 0.01) although no single haemodynamic parameter differentiated absolute peak VO₂ above and below its median value.

**Discussion** VA coupling is a marker of RV energetic efficiency and adds to the debate on the multifactorial determinants of exercise capacity in PH. Ees/Ea was comparable to other predictive haemodynamic parameters of exercise capacity and may represent the 'recruitable' myocardial reserve, important for maintaining cardiac output at increased metabolic demand. Ees/Ea may be a potential therapeutic target given the unclear relationship between pulmonary haemodynamics and patient symptoms.

**Abstract S48 Table 1. Univariate predictors of exercise capacity expressed by absolute and predicted VO₂.**

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Univariable analysis</th>
<th>Univariable analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β</td>
<td>P Value</td>
</tr>
<tr>
<td>Age</td>
<td>-0.16</td>
<td>0.50</td>
</tr>
<tr>
<td>Gender</td>
<td>0.53</td>
<td>0.02</td>
</tr>
<tr>
<td>BSA</td>
<td>-0.33</td>
<td>0.16</td>
</tr>
<tr>
<td>NYHA Class</td>
<td>-0.62</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Swan Ganz</td>
<td>-0.60</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mean RAP (mmHg)</td>
<td>-0.42</td>
<td>0.06</td>
</tr>
<tr>
<td>mPAP (mmHg)</td>
<td>-0.56</td>
<td>0.01</td>
</tr>
<tr>
<td>Cardiac index (L/min/m2)</td>
<td>0.60</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Conductance</td>
<td>-0.12</td>
<td>0.61</td>
</tr>
<tr>
<td>RVS/ Sw</td>
<td>0.39</td>
<td>0.09</td>
</tr>
<tr>
<td>Ees (mmHg/ml)</td>
<td>0.06</td>
<td>0.79</td>
</tr>
<tr>
<td>Ea (mmHg/ml)</td>
<td>-0.29</td>
<td>0.22</td>
</tr>
<tr>
<td>Cao (ml/mmHg)</td>
<td>0.33</td>
<td>0.16</td>
</tr>
<tr>
<td>Ees/Ea</td>
<td>0.45</td>
<td>0.04</td>
</tr>
</tbody>
</table>

β values represent standardised Beta coefficients.

The exercise response in pulmonary hypertension (PH) has characteristic features, including decreased peak oxygen consumption (VO₂-peak), increased ventilatory inefficiency (VE/VCO₂ slope) and widened alveolar-arterial oxygen-gradient (AaG). We wished to evaluate if the AaG at peak exercise predicted those patients likely to have PH who would subsequently require catheter studies.

**Methods** We performed a retrospective analysis of patients referred to Hammersmith Hospital between Feb 2008 and Feb 2012 for investigation of Pulmonary Hypertension (PH) who underwent cardiopulmonary exercise testing (CPX) with testing of AaG using arterial blood gas analysis at peak exercise. Patients found to have alternative cardiac or respiratory diagnoses were excluded. Patients given diagnoses of Pulmonary Arterial Hypertension or Pulmonary Hypertension due to Left Heart Disease and with temporally coincident data from CPX and RHC (within 3 months) were included. Patients without cardiorespiratory diagnoses were healthy controls. The VE/VCO₂ slope and AaG were compared to the diagnosis of PH and the trans-pulmonary pressure gradient (TPG), the difference between mean pulmonary artery pressure (mPAP) and pulmonary capillary wedge pressure (PCWP) or left ventricular end diastolic pressure (LVEDP) where available.

**Results** Using logistic regression to predict a diagnosis of PH, AaG had an odds ratios of 2.98 (p < 0.01) and receiver operating characteristic curve for sensitivity and specificity had area under the curve (ROC-AUC) of 0.92. An AaG cut-off of 2.5kPa had 90% sensitivity and 80% specificity. Similarly, VE/VCO₂ had an odds ratio of 1.21 (p < 0.01) and ROC-AUC 0.85 for predicting PH. Combining AaG and VE/VCO₂ had ROC-AUC of 0.94 for diagnosing PH without significant interaction between AaG and VE/VCO₂. For predicting a TPG >12mmHg, AaG had an odds ratios of 4.54 (p < 0.01) and ROC-AUC of 0.95. VE/VCO₂ had an odds ratio of 1.10 (p < 0.01) and ROC-AUC 0.74 for predicting TPG>12mmHg.

**Conclusion** CPX has become part of the diagnostic workup of patients with PH. AaG measured at peak exercise has a high sensitivity and specificity in predicting patients with PH, which may help determining which patients will require invasive catheter studies. The AaG provides independent information than VE/VCO₂ alone in predicting PH and may be useful in the investigation of PH.