slide performed by a researcher blinded to the groups. Data are expressed as median and IQR, and the groups were compared by the Kruskal–Wallis or Mann–Whitney U test.

Results (1) PAH specimens showed co-localisation of p65 within CD68+ macrophages in 75.4 (64.8–84.6)% of samples. Airway epithelium, neutrophils and lymphocytes were also positive for p65. (2) Pulmonary arterial medial thickness was increased in PAH compared to controls, at 33.7 (18.8–67.9)% in vessels 100–250 mm external diameter (E.D.) and 27.2 (14.8–44.2)% in vessels 250–500 mm ED, vs 17.7 (11.2–30.3)% and 14.9 (11.8–17.8)% in controls ($p<0.0001$ between groups). (3) Nuclear p65 was present in pulmonary artery endothelial cells (EC) but not other vascular cells including pulmonary artery smooth muscle cells in PAH: 53.9 (0–100)% of vessels 100–250 mm E.D. and 53.1 (0–100)% of those 250–500 mm E.D. scored EC p65 positivity in PAH compared to 7.5 (0–25.0)% in 100–250 mm ED and 4.7 (0–21.1)% in 250–500 mm ED in controls ($p<0.0001$ between groups) (Abstract P29 Figure 1).

Abstract P29 Figure 1 Percentage of positive endothelial cell (EC) nuclear p65 immunostaining subdivided according to pulmonary artery external diameter (E.D.).

Conclusion NF- κ B activation is present in macrophages and pulmonary arterial endothelial cells in pulmonary arteries of 100–500 mm ED in patients with PAH.

P30 THE CHANGING FACE OF PULMONARY HYPERTENSION: THE ROLE OF HEART AND LUNG DISEASE

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SF Crawley, MK Johnson, AJ Peacock. *Scottish Pulmonary Vascular Unit (SPVU), Glasgow, UK*

Introduction The advent of disease-targeted therapy for pulmonary arterial hypertension (PAH) has led to an increased awareness of this condition within the general cardiology and respiratory communities. More patients are being referred to specialist centres for diagnostic assessment, however there is concern that many will have pulmonary hypertension (PH) due to underlying lung disease or left heart disease.

Aim The aim of the study was to review the outcome of diagnostic admissions to the Scottish Pulmonary Vascular Unit (SPVU) before

(2003–2005) and after (2006–2009) the introduction of disease-targeted PAH therapy.

Methods 470 new patients with suspected PH were admitted between January 2003 and December 2009 for diagnostic assessment including right heart catheterisation (RHC). Demographic and haemodynamic data from these patients were retrospectively reviewed. Following RHC patients were diagnosed with:

- Group 1. PAH;
- Group 2. Pulmonary venous hypertension (PVH) due to left heart disease;
- Group 3. PH due to hypoxic lung disease (HLDPH);
- Group 4. Chronic thromboembolic PH (CTEPH);
- Group 5. PH due to unclear multifactorial mechanisms (PH Misc);
- No PH.

Results In 2003–2005, 114 patients underwent diagnostic assessment, and 112 had PH. Of these 77.7% had PAH and 18.8% had CTEPH. Only 2.7% of patients had PVH and 0.8% had HLDPH. In 2006–2009, 356 patients underwent diagnostic assessment, and 308 had PH. Of these only 51.9% had PAH and 15.9% had CTEPH. However, now 17.9% of patients had PVH, and 10.4% had HLDPH. 48 patients had No PH at the time of RHC (Abstract P30 Figure 1).

Abstract P30 Figure 1 Outcome of diagnostic assessment in the pre and post-treatment era.

Conclusion Prior to admission all referrals are screened by an SPVU consultant, but despite this there are a significant number of patients proceeding to RHC who will have PH due to left heart or lung disease. This highlights the importance of fully assessing all patients with suspected PH, as per ESC/ERS 2009 guidelines, before instituting expensive and potentially dangerous PAH therapy. We need to improve our non-invasive screening methods so that fewer patients proceed to RHC who do not have PAH.

P31 DISEASE TARGETED THERAPIES AND EFFECT ON SURVIVAL IN IDIOPATHIC, HERITABLE AND ANOREXIGEN-ASSOCIATED PULMONARY ARTERIAL HYPERTENSION (PAH)

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Y Ling, M Johnson, A Peacock. *Scottish Pulmonary Vascular Unit, Glasgow, UK*

Introduction The median survival of idiopathic PAH was 2.8 years before the availability of modern therapies. Substantial progress has been made over the past 20 years. Currently available disease targeted therapies have been shown in clinical trials to improve symptoms, exercise capacity and survival. Combination therapies