infrequently performed in the United Kingdom. We however, have persisted with video assisted thoracoscopic (VATS) LVRS and report the results of our first 200 operations.

Patients/methods Surgical candidates for LVRS were selected by a multidisciplinary team approach using radionuclide scintigraphy in all cases. After the initial 20 open cases we adopted policy of two stage bilateral VATS LVRS with the timing of the second operation determined by the patient. 160 patients have completed one-stage LVRS: median age 60 (39–73) years and 20 patients have completed two-stage bilateral LVRS: median age 59 (41–66) years. Data from a prospective database were analysed to assess the postoperative changes in pulmonary function and health status (SF 36 questionnaire) recorded at three, six and, twelve months and thence annually.

Results 30-day mortality was 6% and 25% required post-operative ventilation (9 of 50 for <24 h). Hospital stay was 14 (2–110) days. FEV_1 in both groups showed significant continuous improved at the 3-month, 6-month and 1-year postoperative review ($p \leq 0.035$). Furthermore, significant improvements ($p \leq 0.05$) in SF 36 were seen across the seven domains assessed: Physical Functioning (up to 1 year), Social Functioning (up to 1 year), Role-Physical (up to 1 year), Role-Emotional (at 5 years), Mental Health (up to 1 year), Energy/Vitality (up to 1 year), and General Health (up to 1 year). There has however been a significant reduction in the domain of Bodily Pain up to 4 years post LVRS ($p \leq 0.05$). More lasting improvements are seen in two-stage bilateral group compared to the one-stage group.

Conclusion The use of a multidisciplinary team approach and a prolonged surgical strategy can achieve durable postoperative benefits from LVRS for up to 4 years with acceptable risk.

P142 INFLAMMATORY CELLS IN THE QUADRICEPS OF COPD PATIENTS AND RESPONSE TO RESISTANCE TRAINING

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Background Physical exercise in healthy individuals leads to an acute inflammatory-cell response in skeletal muscles. In COPD, quadriceps dysfunction is an important systemic manifestation that can be offset by exercise training. However, the nature of the muscle inflammatory response in these patients to acute and chronic exercise remains unknown. We therefore measured inflammatory cell infiltration in the quadriceps of COPD patients and healthy controls, in response to a programme of lower-limb resistance training.

Methods 12 COPD patients (mean (SD) age 66.7(7.0) years, BMI 26.1(7.2) kg/m^2, FEV_1 46.4 (20.5) % predicted, 10 males) and seven healthy controls (66.7 (5.1) years, BMI 27.7(2.4) kg/m^2, FEV_1 103.4 (17.0) % predicted, five males) underwent 8 weeks of bilateral lower-limb, high-intensity resistance training, thrice weekly, on an isokinetic dynamometer (Cybex II Norm). Quadriceps muscle biopsies from the dominant thigh were obtained at baseline (T0), 24-h after the 1st exercise bout (T1), and 24-h after the last exercise session following 8-weeks training (T2). Glycol methacrylate-embedded muscle biopsies were analysed using immunohistochemistry. Inflammatory cells were identified using antibodies against neutrophil elastase (NE) and CD163 (macrophages). Dual energy x-ray Absorptiometry (DEXA) was used to determine thigh muscle mass and isometric quadriceps strength was measured on the cybex.

Results At T0, neutrophils were not detected in any of the healthy controls, but were present in six out of 12 patients. A significant increase in neutrophil and macrophage counts in both patients and controls was seen at T1 (Abstract P142 Table 1). At T2, inflammatory cell counts in both groups were close to baseline values. Isometric quadriceps strength (COPD: pre vs post -134.0 (44.0) vs 151.8 (47.9) Nm, p=0.002; Healthy: pre vs post 153.4 (42.4) vs 169.0 (43.1) Nm, p=0.04] and thigh lean mass (COPD: pre vs post 4.4 (1.2) vs 4.7 (1.2) kg, p=0.003; Healthy: pre vs post 4.5 (0.6) vs 4.7 (0.7) kg, p=0.004] increased significantly after training in both groups.

Conclusions Acute resistance exercise in COPD leads to an inflammatory-cell response in the quadriceps that is comparable to healthy controls. Regular training results in muscle adaptation characterised by a diminished inflammatory response to a bout of exercise.

Abstract P142 Table 1

	T0	T1	T2
NE (cells/mm^2 interstitial area)			
COPD (n=12)	4.1 (0.0–21.0)	137.9 (30.1–217.9)*	0.0 (0.0–15.9)‡
Healthy (n=7)	0.0 (0.0–0.0)	153.4 (13.0–305.5)†	14.2 (0.0–17.3)
CD163 (cells/mm^2 interstitial area)			
COPD (n=12)	10.4 (2.1–34.1)	90.3 (40.9–135.7)†	26.9 (12.7–72.5)
Healthy (n=7)	0.0 (0.0–27.0)	146.7 (64.3–172.2)†	0.0 (0.0–59.2)§

Values presented as medians (IQR).
Data at various time-points compared using nonparametric repeated measures ANOVA (Friedman's Test) and Dunn's post test.
*p<0.001, T1 vs T0.
†p<0.05, T1 vs T0.
‡p<0.001 T2 vs T1.
§p<0.05, T2 vs T1.

P143 NF-KAPPA B (NF-κB) AND ACTIVATOR PROTEIN-1 (AP-1) DNA BINDING IN THE QUADRICEPS OF COPD PATIENTS

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Introduction Animal work implicates NF-κB and AP-1 activation in muscle atrophy (Cai *et al, 2004 and* Costelli *et al*, 2005). Prior study of seven COPD patients with a low BMI and seven COPD patients with a normal BMI suggested that NF-κB DNA binding was increased in the former (Agusti *et al*, 2004) although others have not demonstrated NF-κB activation in COPD muscle (Plant *et al*, 2009).

Aim Evaluate NF-κB and AP-1 activation in the quadriceps of patients with stable COPD in relation to muscle atrophy.

Methods 114 COPD patients and 30 healthy age-matched controls underwent measurements of lung function and bioelectrical impedance to determine fat-free mass index (FFMI) and a percutaneous Bergstrom needle biopsy of the quadriceps. Transcription factor ELISAs were performed on nuclear extracts from quadriceps muscle to measure quantities of NF-κB P50, P65, and AP-1 c-jun subunits in muscle nuclei capable of binding DNA. Immunohistochemistry was used to determine type I and II fibre cross-sectional area (CSA) from muscle sections.