Abstract S55 Table 1 Patient characteristics, EMG para%max, measures of pulmonary function and exercise performance in thirty patients with CF

	Median	Range
Age	24	16 to 47
BMI	20.6	18.0 to 29.4
EMGpara% max	7.8	2.0 to 34.5
FEV_1 (% pred.)	63.3	16.0 to 101.3
RV/TLC (%)	40.6	21.5 to 74.0
FRC_{pleth}/TLC (%)	58.9	41.2 to 78.6
ISWT (M)	630	280 to 880
VO_{2peak} (ml/min/kg)	24.9	17.1 to 39.9

plethysmography and an incremental shuttle walking test (ISWT) with breath by breath metabolic data were also performed.

Results Patient characteristics, EMGpara%max, measures of pulmonary function and exercise performance are shown in Table 1. EMGpara%max was significantly associated with residual volume/total lung capacity ratio (RV/TLC, r = 0.724, p < 0.001), forced expiratory volume in 1 second (FEV_1% pred. r = -0.648, p < 0.001) and functional residual capacity/total lung capacity ratio (FRC_{pleth}/TLC, r = 0.625, p < 0.001). EMGpara%max showed the strongest relationship with ISWT distance (r = -0.612, p < 0.001) and peak oxygen uptake (VO_{2peak}, r = -0.665, p = 0.001). Weaker relationships were observed between ISWT distance and pulmonary function (FEV_1r = 0.518 p = 0.006, RV/TLC r = -0.451 p = 0.024, FRC_{pleth}/TLC r = -0.299 p 0.147) and VO_{2peak} (FEV_1r = 0.521 p = 0.008, RV/TLC r = -0.505 p = 0.014, FRC_{pleth}/TLC r = -0.389 p = 0.066).

Conclusion EMGpara correlates strongly with conventional pulmonary function measures in CF and has a closer relationship with exercise capacity than standard pulmonary function parameters. EMGpara%max therefore represents a promising marker of CF lung disease severity.

S56 DIFFERENCES IN FORCED OSCILLATION TECHNIQUE BETWEEN HEALTHY INDIVIDUALS, OBSTRUCTIVE SLEEP APNOEA AND OBESITY HYPOVENTILATION SYNDROME

[1]S Mandal, [1]A Vaughan-France, [2]T Dhir, [1]ES Suh, [3]P Pompilio, [3]R Dellaca, [1]N Hart. *[1]Lane Fox Respiratory Unit, Guy's and St Thomas' Hospital, London, UK; [2]King's College London, London, UK; [3]Department of Electronic Informatics and Bioengineering, Politecnico Di Milano, Milan, Italy*

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Introduction Forced oscillation technique has been used to demonstrate expiratory flow limitation (EFL, by measurement of $DX_{rs,5Hz}$) in chronic obstructive pulmonary disease, however, this technique has not been widely used in the obese population. Obese individuals breathe at lower lung volumes and are therefore likely to develop EFL. We have previously demonstrated EFL occurs in individuals with obesity hypoventilation syndrome (OHS) but wished to determine if this also occurred in those with obstructive sleep apnoea (OSA) and compare differences between these groups.

Method Subjects with established OSA, OHS and healthy volunteers were recruited from the Lane Fox Respiratory Unit and Sleep Disorders Centre, St Thomas' Hospital. Subjects underwent measurements of height, weight, spirometry and EFL (ResmonPro, ResTech, Milan, Italy).

Results Eleven healthy (HC), 8 OSA and 9 OHS subjects were recruited, age 23.6 ± 4.2, 31.4 ± 8.0 and 58.9 ± 10.4 years respectively. Body mass index (BMI): healthy subjects 17.9 ± 2.9; OSA group 41.4 ± 8.0; OHS group; 46.8 ± 9.3 kg/m^2, there were significant differences in BMI between the HC and OSA and OHS groups (p < 0.001) but no difference between OSA and OHS. Spirometry (FEV1, FVC): HC 3.54 ± 1.15, 4.35 ± 1.47, OSA 2.55 ± 0.85, 3.27 ± 1.03 OHS 2.04 ± 0.74, 2.58 ± 0.85. In both the OSA and OHS groups DX_{rs} increased with recumbency, as did the percentage of flow limited breaths (Table 1). Each group significantly increased their inspiratory resistance with the supine position compared to the upright seated position. There was a significant difference in DX_{rs} between HC and OHS only in upright, 45° and supine positions (p < 0.05). There was also a difference in the percentage of EFL breaths between HC and OHS in the 45° and supine positions and between OSA and OHS in the 45° position (p < 0.05).

Conclusion Patients with obesity and sleep disordered breathing experience EFL, which was more evident in the OHS group compared to the OSA group. This may be a consequence of their higher BMI impacting their lung volumes to a greater extent. Furthermore, the impact of position was greater in the OHS group suggesting that EFL may be a contributing factor in the development of hypercapnic respiratory failure in these individuals.

S57 AEROBIC TRAINING AND DETRAINING IN COPD AND HEALTHY CONTROLS

B Popat[1], L Latimer[1], L Houchen-Wolloff[1], C Bolton[2], [1]M Steiner. *[1]NIHR Respiratory BRU, Glenfield Hospital, Leicester, UK; [2]Nottingham Respiratory Research Unit, Nottingham, UK*

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Introduction Deconditioning is a key cause of exercise intolerance in COPD patients. Exercise training provides an effective method of improving aerobic exercise performance in this group. There is poor understanding on the trajectory of changes

Abstract S56 Table 1 Differences in expiratory flow limitation, as demonstrated by ΔX_{rs} between healthy controls, OSA and OHS

	Healthy controls			Obstructive Sleep Apnoea			Obesity Hypoventilation Syndrome		
	Upright Seated	45°	Supine	Upright Seated	45°	Supine	Upright Seated	45°	Supine
$DX_{rs,5Hz}$ ($cmH_2O.s.\ L^{-1}$)	-0.15 ± 0.13	0.04 ± 0.21*	-0.03 ± 0.20	0.52 ± 0.74	1.18 ± 1.51	2.05 ± 2.23	2.33 ± 2.96	4.45 ± 2.91	5.18 ± 2.65*
% of EFL breaths	0 ± 0	0 ± 0	0 ± 0	0 ± 0	20 ± 27	38 ± 3	35 ± 45	68 ± 4	73 ± 34*
Inspiratory Reactance									
$X_{insp,5Hz}$ ($cmH_2O.s.\ L^{-1}$)	-0.55 ± 0.21	-0.67 ± 0.43	-0.69 ± 0.43	-0.93 ± 0.89	-2.47 ± 1.66	-1.91 ± 0.91*	-2.60 ± 0.95	-3.38 ± 1.80	-2.69 ± 1.75
Inspiratory Resistance									
$R_{insp,5Hz}$ ($cmH_2O.s.\ L^{-1}$)	2.71 ± 0.55	3.45 ± 0.66	3.96 ± 0.99*	4.31 ± 1.34	6.20 ± 1.35	6.43 ± 1.52*	5.61 ± 1.86	6.42 ± 1.05	7.46 ± 1.11*
R5–19insp	-0.23 ± 0.18	0.04 ± 0.21	0.19 ± 0.31*	0.37 ± 0.49	1.29 ± 0.96	1.29 ± 0.56*	1.91 ± 0.89	2.20 ± 0.60	2.21 ± 0.81
Ti/Ttot	0.44 ± 0.04	0.45 ± 0.05	0.45 ± 0.03	0.43 ± 0.04	0.45 ± 0.03	0.45 ± 0.04	0.41 ± 0.05	0.41 ± 0.07	0.43 ± 0.07
VE (L/min)	14.26 ± 7.06	13.5 ± 5.19	14.62 ± 7.58	15.17 ± 5.67	17.72 ± 5.02	15.83 ± 4.75	20.88 ± 9.44	13.71 ± 3.32	13.3 ± 2.62
RR (bpm)	15.75 ± 1.99	17.9 ± 2.93*	17.5 ± 2.95*	20.61 ± 7.48	21.59 ± 6.52	21.7 ± 5.46	22.14 ± 6.90	20.84 ± 3.79	19.32 1.71

*Significantly different from seated position within group (ANOVA, p < 0.05)
Abbreviations: EFL=Expiratory Flow Limitation, VE=minute ventilation, RR=Respiratory Rate