Abstract P44 Figure 1 Adjusted odds ratios (95% CI) for oral steroid (A) and short-acting beta-agonist use (B) in the year preceding measurements of FEV₁ (<80% predicted, n = 140 vs >80% predicted, n = 302), FEV₁/FVC (<0.70, n = 131 vs >0.70, n = 311), FEF₂₅₋₇₅ (<60% predicted, n = 238 vs >60% predicted, n = 204), R5 (>150% predicted, n = 183 vs <150% predicted, n = 259) and R5-R20 (>0.10 kPa·L⁻¹·s, n = 185 vs <0.10 kPa·L⁻¹·s, n = 257). The 95% CIs which exclude unity are defined as being of statistical significance.

Methods Spirometry and IOS measurements from asthmatics were linked to a health informatics database for oral steroid and short-acting beta agonist (SABA) use 1 year prior to the measurements.

Results 442 patients had both spirometry and IOS, mean FEV₁ = 86% predicted, 94% on ICS, median dose 800 µg/day. IOS and spirometry measures were equally predictive of impaired asthma control for both oral steroid and SABA use. For oral steroid use, the adjusted odds ratio, OR (95% CI): FEV₁ <80%: 1.56(0.99–2.47) p = 0.056, FEV₁/FVC 25–75 <60%: 1.84(1.18–2.86) p = 0.007, R5 >150%: 1.91(1.25–2.95) p = 0.003 and R5-R20 >0.1 kPa·L⁻¹·s 1.73(1.12–2.66) p = 0.013. For SABA use, the adjusted OR (95% CI): FEV₁ <80%: 2.22(1.43–3.44) p < 0.001, FEV₁/FVC 25–75 <60%: 2.51(1.65–3.82) p < 0.001, R5 >150%: 1.76(1.18–2.63) p = 0.006 and R5-R20 >0.1 kPa·L⁻¹·s: 2.94(1.94–4.46) p.

Conclusion Spirometry or IOS measurements are equally useful as potential markers of asthma control in persistent asthmatic patients.

P45 SPUTUM AND NASAL MARKERS OF INFLAMMATION IN SEVERE ASTHMA - A PILOT STUDY

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Introduction and objectives The Manchester Severe Asthma Team has successfully been using sputum eosinophil monitoring

Abstract P45 Table 1 Characteristics of severe asthma patients enrolled on James Trust Study

	N	Minimum	Maximum	Mean	Std. Deviation
Patient age at sample	32	23	77	48.6	12.8
BMI	26	19.7	51	30.8	6.6
% predicted FEV1	26	43	131	79	23.1
FEV1/FVC	27	48	111	73	14.5
BDP equivalent	32	0	4800	1785	1245.2
Prednisolone equivalent	32	0	60	8.27	13.6
Total IgE	23	2.3	1600	287.6	450.8
Smoking pack years	26	0	60	7.8	14.3

for several years to help tailor steroid medication. Whilst very useful for patients able to produce a sputum sample, some patients cannot produce a sample. As a result we looked at developing an alternative method of monitoring using nasal lavage samples to study the intra-individual changes in inflammation in severe asthma.

Methods Patients requiring sputum monitoring as part of their clinical management were invited to take part in this pilot and to provide an additional nasal lavage sample obtained using an olive method. Participants were clear of infection at time of sampling. Sputum was either spontaneous or induced using the traditional 3%–4%–5% nebulised sodium chloride procedure. ECP (Eosinophil Cationic Protein) was measured in sputum and nasal supernatants using a commercial ELISA kit (Mesacup, MBL). Differential cell count (DCC) was attempted for both sputum and nasal sample types.

Results This abstract show the results obtained for the first 32 consecutive patients. Our patient population is described in Table 1, 69% female, 69% atopic (as defined by positive RAST of elevated total IgE), and 50% were non-smokers, 7.6 current smokers and 42.4% ex-smokers. No patient was immunosuppressed or on IM Triamcinolone.

ECP levels were as follows:

- Sputum: median 2650 (min:20.76–28603 ng/ml), 100% of samples had detectable levels.
- Nasal lavage: 0(0–7.6), 20%.

DCC were as follows:

- Sputum: 38% patients were eosinophil positive (as defined by >3%),
- Nasal lavage: no eosinophil was detected, 38.5% of samples had a DCC but interpretation was hindered by low cell yield.

Sputum DCC/ECP did not correlate significantly with nasal ECP (Pearson R=0.168, p = 0.374 and R=-0.048, p = 0.807 respectively). Nasal DCC data could not be computed as no patient was found to be eosinophil positive.

Sputum DTT and sputum ECP correlated significantly (Pearson R=0.607, p = 0.001) as reported in the literature.

Conclusions At this stage of our pilot, intermediate data analysis shows that nasal sampling does not appear to be a successful alternative to sputum monitoring in severe asthma.