NIV naïve initially, 13% went home established on NIV. Survival at 365 days post procedure was 64%. Subset analysis of outcomes in bulbar vs. non-bulbar MND, FVC < or > 50% and NIV for procedure only vs. discharge with NIV has not shown any statistically significant differences, although absolute numbers are small.

Conclusions High risk NMD patients can have PEGs inserted safely. Our complications and one year survival rates are better compared with current published evidence in lower risk groups. We believe this is due to intensive support and monitoring during the procedure and use of NIV. Although survival is largely related to disease progression, further analysis is required with larger numbers to fully assess the impact of PEG feeding on it.

P178 NOCTURNAL OXIMETRY MONITORING TO PREDICT HYPERCAPNIA IN OBESE PATIENTS

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Introduction Clinical commissioning standards have been developed to streamline clinical pathways. It is now common practice for obese patients with suspected sleep disordered breathing to undergo nocturnal oximetry monitoring prior to the clinic consultation. Although this test is useful for diagnosis and risk stratification of patients, there are limited data reporting the use of oximetry to predict hypercapnia. We hypothesised that overnight oxygen saturations could be used to predict hypercapnia.

Method 186 oximetry studies from patients with a body mass index (BMI) > 30 $kg.m^{-2}$ and an FEV_1/FVC >0.7 were analysed, including the percentage of total analysis time spent with an oxygen saturation (S_pO_2) below 90% (T < 90%), 80% (T < 80%) and 70% (T < 70%) as well as 4% and 3% oxygen desaturation index (ODI). Correlations and linear regression analyses were performed to determine the variables that predicted a daytime arterial partial pressure of carbon dioxide (P_aCO_2) > 6.0 kPa. Binary logistic regression and receiver-operator characteristic analyses assessed the utility of these parameters in predicting hypercapnia.

Results Compared to the eucapnic group the hypercapnic patients had a higher 4% ODI (42.6 ± 35.5 events/hour *vs.* 24.5 ± 19.5 events/hour, p = 0.003), lower mean SpO_2 (89.0 ± 7.4% *vs.* 94.1 ± 3.2% p = ns) and higher T < 90% (36.3 ± 32.1% *vs.* 13.5 ± 20.4%, p < 0.001).

Significant, albeit weak, correlations between $PaCO_2$ and 4% ODI, 3% ODI, T < 90%, T < 80%, T < 70% were observed (Table 1). Only T < 90% was predictive of hypercapnia. Using the total analysis time with an S_pO_2 < 90%, a cut off level of ≥7.2% had a sensitivity of 80% and a specificity of 60% in predicting a $PaCO_2$ >6 kPa, area under the curve was 0.76.

Conclusion The proportion of time spent with an S_pO_2<90% predicted hypercapnia in obese patients. This has the potential to risk stratify patients, optimising both the timing and type of treatment delivered, which in turn will enhance the delivery of care. Specifically, this would facilitate clinical decision making in directing patients towards investigation for receiving non-invasive ventilation rather than continuous positive airway pressure therapy if hypercapnia were predicted from the proportion of the time with an S_pO_2 < 90%.

P179 FORCED VITAL CAPACITY, SYSTEMIC INFLAMMATION AND CARDIOMETABOLIC MARKERS IN ADULTHOOD: A CROSS-SECTIONAL ANALYSIS

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Introduction Forced vital capacity (FVC) is a powerful predictor of mortality, more than airflow obstruction (Burney *et al*. Thorax 2011;66:49–54). FVC is associated with systemic inflammation as well as with cardiovascular disease and diabetes. Given that systemic inflammation is also associated with cardiovascular disease and diabetes, systemic inflammation could explain the observed association between FVC and cardiometabolic markers. Here, we examined the association between FVC, cardiometabolic markers and systemic inflammation in 3,731 individuals belonging to the Northern Finland Birth Cohort 1966.

Methods Using linear regression, we examined the association between i) cardiometabolic markers (systolic blood pressure, diastolic blood pressure, LDL cholesterol, triglycerides, fasting glucose, insulin and HOMA-IR) and inflammatory markers (C-reactive protein (CRP) and white blood cell count (WBC)), ii) FVC and inflammatory markers, and iii) FVC and cardiometabolic markers. We then tested whether the association between FVC and cardiometabolic markers could be explained by systemic inflammation, by adjusting the linear regression models of FVC on each cardiometabolic marker for the two inflammatory markers.

Results Increasing levels of inflammatory markers were associated with a decrease in FVC, -12mL per mg/L of CRP (95% confidence interval (CI): -17 to -7 mL) and -17 mL per 10^9 cells/L of WBC (95% CI: -28 to -7 mL), and with increasing levels of the cardiometabolic markers. FVC also decreased with

Abstract P179 Table 1. Association between FVC and cardiometabolic markers before and after adjustment for systemic inflammation

	FVC (mL)	
	Unadjusted analysis[a]	Analysis adjusted for systemic inflammation[b]
Cardiometabolic markers	beta (95 % CI)	beta (95 % CI)
Systolic blood pressure (SD=13.6mmHg)	-27 (-45;-8.1)**	-21 (-40;-2.9)*
Diastolic blood pressure (SD=11.4mmHg)	-60 (-78;-43)***	-57 (-75;-40)***
LDL Cholesterol (SD=0.88mmol/L)	-31 (-49;-13)**	-30 (-48;-13)**
Triglycerides (SD=0.73mmol/L)	-74 (-91;-56)***	-68 (-86;-50)***
Glucose (SD=0.58mmol/L)	-26 (-44;-8.8)**	-25 (-42;-7.2)**
Insulin (SD=4.3mU/L)	-74 (-91;-57)***	-68 (-85;-50)***
HOMA-IR	-61 (-75;-46)***	-55 (-70;-40)***

[a]Adjusted for height at 31 years and gender. [b] Adjusted for height, gender, C-reactive protein and white blood cell count, measured at 31 years. Except for HOMA-IR, associations are reported as regression coefficients (beta) per standard deviation (SD) change in the cardiometabolic marker, with 95% confidence interval (95 % CI). *P<0.05, **P <0.01, ***P <0.001.

increasing levels of cardiometabolic markers (Table, column 1) and adjusting these associations for the inflammatory markers did not substantially alter them (Table, column 2).

Conclusion The association between FVC and cardiometabolic markers is not explained by variation in inflammatory markers, as measured by CRP and WBC. However, due to the cross-sectional nature of the analysis, no inference can be made with regard to the directionality of the associations.

P180 NOCTURNAL OXIMETRY IN CYSTIC FIBROSIS (CF)

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Background It is common for CF children in the UK to receive sleep-disrupting 4 hourly observations day and night throughout their hospital stays for intravenous antibiotics or exacerbations judging from parental and patient feedback. As oxygen desaturations occur mainly during REM sleep rather than NREM sleep, these desaturations could be missed by the intermittently performed "routine observations". Our unit performs nocturnal oximetry studies on night 1 of admission for all CF patients admitted for intravenous antibiotics and discontinues them if oxygenation is normal and the patient improving.

Aims To determine whether day time physiology or lab work is predictive of nocturnal hypoxaemia.

Methods A retrospective comparison of nocturnal oximetry studies and daytime physiology plus HbA1c of all CF patients admitted for intravenous antibiotics between January and June 2013.

Results 54 CF patients were admitted, with 19/54 elective and 35/54 acutely. Table 1 summarises the clinical characteristics and nocturnal oximetry results of the patients. There was a weak positive correlation between Mean SpO2 and admission FEV1 ($p = 0.0012$, $r^2 = 0.21$) and admission FVC ($p = 0.0024$, $r^2 = 0.18$). Nocturnal oxygen was commenced in 4/54 children (7.4%) as their mean SpO2 was < 93%. All of them had an FVC < 60%, the only ones in the cohort. 3 of the children improved (FVC improved to 71%, 75% and 87% from 44%, 54% and 58% respectively), they were weaned off oxygen before discharge, with normal gas exchange on repeat nocturnal oximetry) and 1 died.

Abstract P180 Table 1. Nocturnal oximetry in Cystic Fibrosis (CF).

Age (years)	13[0.11–17.5] median[range]
Admission FEV1 (% predicted)	75[69–80] mean[95%CI]
Admission FVC (% predicted)	85[80–90] mean[95%CI]
Best FEV1 in past year (% predicted)	88[83–92] mean[95%CI]
Best FVC in past year (% predicted)	97[94–101] mean[95%CI]
Mean SpO2 (%)	97.1[91.4–98.9] median[range]
Desaturations ≥4%/ hour	1.5[0–10.05] median[range]
Percentage time sats<90%	0[0–10.9] median[range]
HbA1c (%)	5.7[4.2–7.1] median[range]

Conclusion Patients with an admission FVC of <60% warrant close monitoring for nocturnal hypoxaemia. In CF patients who are cardiovascularly stable, performing an oximetry study on first night of admission may be a less disruptive alternative to routine intermittent observations nightly for the whole duration of the admission.

P181 TRANSCUTANEOUS CO2 MONITORING IN HYPERCAPNOEIC RESPIRATORY FAILURE: A META-ANALYSIS OF PROSPECTIVE STUDIES

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Introduction The role of transcutaneous carbon dioxide monitoring ($P_{tc}CO_2$) for patients with respiratory failure has been studied in a variety of clinical settings. However, its accuracy compared to arterial partial pressure of carbon dioxide ($PaCO_2$) in patients undergoing non-invasive ventilation (NIV) for hypercapnoeic respiratory failure has not been validated. The degree to which $PtcCO_2$ approximates 'gold-standard' $PaCO_2$ in this context was evaluated in a meta-analysis including 16 prospective observational studies.

Methods 16 prospective studies evaluating $PtcCO_2$ as a correlate for $PaCO_2$ in patient cohorts undergoing NIV were included. In all cases, Bland-Altman analysis was used to compare agreement among measures. Mean bias between the two methods of

Abstract P181 Table 1.

Study	Type	N	Time	Mean bias/2SD
Berkenbosch et al (2001)	Paediatric trauma, ARDS	25	4 Hours	0.02 (3.27)
Chakravarthy et al (2010)	Ventilator weaning in cardiosurgery	32	4 Hours	-1.3 (7.80)
Cox et al(2006)	NIV for COPD exacerbations	22	4 hours	-0.15 (0.75)
Cuvelier et al (2005)	Long-term home NIV	12	40 minutes	-0.72 (2.98)
Gancel et al (2011)	Acute resp.failure in ED	29	2 hours	0.01 (0.81)
Hazenburg et al (2011)	Chronic resp.failure	15	8 hours	0.40 (0.87)
Janssens et al (1998)	NIV in ICU	26	4 hours	0.10 (0.69)
Janssens et al (2001)	NIV in ICU	28	8 hours	-2.8(3.8)
Johnson et al (2008)	Tracheostomised patients	41	10 hours	0.07 (0.55)
Kelly and Klim (2011)	Acute resp.failure in ED	46	Single	0.81 (1.87)
Nicolini and Ferrari (2011)	Acute resp failure requiring NIV	80	10 minutes	0.11 (0.73)
Paiva et al (2009)	Chronic resp.failure	65	6 hours	1.00 (5.20)
Parker and Gibson (2007)	Routine resp.practice	48	10 minutes	-0.04 (1.34)
Sivan et al (1992)	Paediatric resp.failure	134	Single	0.17 (1.92)
Storre et al (2007)	NIV titration	10	4 hours	0.61 (1.15)
Storre (2011)	Nocturnal NIV in chronic resp.failure	24	8 hours	0.11 (1.00)