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.

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