

Methods Comparison of the volume of new domiciliary NIV set-ups and the elective NIV set-up rate over three 12-month periods: Apr 2005-Mar 2006 (period 1), Apr 2011-Mar 2012 (period 2) and Apr 2012-Mar 2013 (period 3) in a dedicated 11-bedded ward-based NIV unit (established: Aug 2004) in a 1000-bedded central England teaching hospital trust, providing domiciliary NIV support to over 260 patients with over 392 under surveillance for respiratory failure.

Results The volume more than doubled from 19 new domiciliary NIV set-ups in period 1 to 39 new domiciliary NIV set-ups in period 2; to 64 set-ups in period 3. The elective domiciliary NIV set-up rate increased from 7/19 (36.8%) to 19/39 (48.7%) to 30/64 (46.9%) for periods 1, 2 and 3 respectively [Figure 1].

Discussion We have previously shown that the elective set-up rate for new HMV has gone up in our unit. In this survey we have shown that this increase in 'elective set-up rate' is associated with a consistent increase in volume of HMV set-ups. This is most likely to be due to an increased number of people at risk of respiratory failure coming under the unit's surveillance. HMV is well known to improve quality of life and reduce unscheduled care utilisation when started at the appropriate timepoint in chronic ventilatory failure through surveillance. Comparison of data between centres supervising domiciliary NIV/HMV, e.g 'elective set-up rates', is warranted in this rapidly evolving field.

REFERENCES

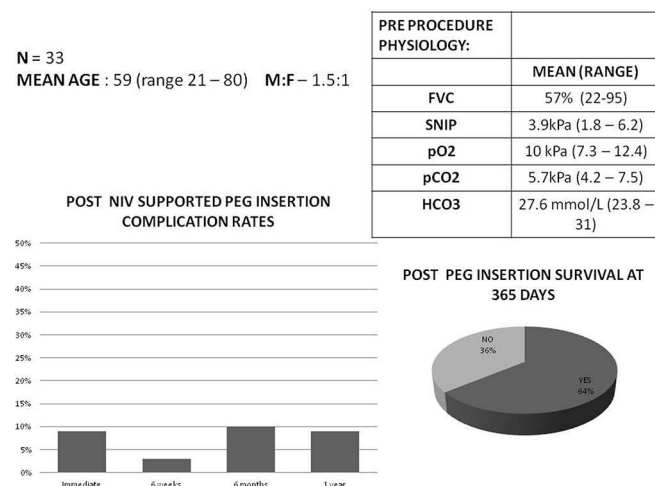
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P176 INDICATIONS AND DEMOGRAPHICS OF DOMICILIARY NIV SET-UPS IN AN ACUTE HOSPITAL

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Background Domiciliary NIV is being increasingly used to treat chronic ventilatory failure but there is little site-level data available describing the demographics of patients on domiciliary NIV under centres which have developed over the last decade. At our central England teaching hospital, domiciliary NIV is either set up following an acute admission with hypercapnic acidotic



Abstract P177 Figure 1.

respiratory failure through the dedicated 11-bedded ward-based unit or electively, through the surveillance of patients at risk of ventilatory failure. Currently we have 262 patients on domiciliary NIV. We aimed to analyse the primary diagnosis and demographics of patients started on domiciliary NIV in the last 18 months.

Method A retrospective analysis of all patients started on domiciliary NIV at a 1000-bedded central England teaching hospital from 01 Jan 2012 to 30 June 2013.

Results A total of 90 patients were analysed and there was a slight male predominance (55.5%). The mean age at initiation of domiciliary NIV was 56.3 years (SD 18.9, median 52.5 years). Primary diagnoses (reason for domiciliary NIV) were 1. neuro-muscular disorders (35.5%); 2. obesity-related disorders (33.3%); 3. COPD (13.3%); 4. thoracic cage disease other than obesity (15.6%) and 5. central pathology (2.3%). Of the COPD patients, 7/12 (58.33%) were GOLD class 4, 4/12 (33.33%) were GOLD class 3 and 1/12 (8.33%) were GOLD class 2. The mean domiciliary NIV set-up per month was 4.68 (SD 3.16). There was no clear relationship between number of set-ups per month and corresponding calendar month; 22.2% patients (20/90) had long term oxygen therapy prescribed with their NIV.

Discussion The role of domiciliary NIV is expanding with greater numbers of people living with chronic ventilatory failure, and this is set to increase with the rising problem of (a) obesity-related respiratory disorders and (b) improved survival of children with neuromuscular weakness. This study highlights the need for a domiciliary NIV registry for improved resource and workforce planning.

P177 ARE NIV SUPPORTED PEG INSERTIONS (NSPI) IN PATIENTS WITH NEUROMUSCULAR DEGENERATIVE DISORDERS (NMD) SAFE AND EFFECTIVE?

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Introduction Patients with NMD's suffer from feeding difficulties and respiratory failure which worsens prognosis. A survival advantage with PEG feeding has been suggested in case reports but there are concerns regarding safety and complications in this high risk group in or at risk of ventilatory failure. We have therefore reviewed the outcomes of NSPIs in our tertiary teaching hospital.

Methods 33 NSPIs were identified upto 2012. Disease background, baseline lung physiology, NIV use, peri-procedure details, complications and survival at 365 days were analysed. A subset analysis examining bulbar vs. non bulbar MND, baseline FVC and NIV use against survival at 365 days was also carried out.

Results 33 patients with NMD (MND 79%, DMD 9%, Myotonic Dystrophy 6%, others 6%) were included. Mean age was 59 (range 21–80). Mean pre-procedure FVC was 57% (22–95), SNIP was 3.9kPa (1.8–6.2kPa). Mean pre-procedure pO₂ was 10kPa (7.3–12.4), pCO₂ 5.7kPa (4.2–7.5) and HCO₃ 27.6mmol/L (23.8–31). 52% were previously on NIV. Mean pre-procedure NIV settings were IPAP 18cmH₂O, EPAP 3cmH₂O. Mean post procedure settings were 19 and 3cmH₂O respectively. 11% needed supplemental oxygen for a short period post procedure. Sedation was used in 95% and no medical reversals were needed. Complication rates were 9%, 3%, 10% and 9% (immediate, 6 weeks, 6 months, 1yr) respectively. Of those who were

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 FEV₁/FVC >0.7 were analysed, including the percentage of total analysis time spent with an oxygen saturation (S_pO₂) 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_aCO₂) > 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 SpO₂ (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 PaCO₂ 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_pO₂ < 90%, a cut off level of ≥7.2% had a sensitivity of 80% and a specificity of 60% in predicting a PaCO₂ >6 kPa, area under the curve was 0.76.

Conclusion The proportion of time spent with an S_pO₂ <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_pO₂ < 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 10⁹ 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

| Cardiometabolic markers | FVC (mL) | |
|----------------------------------------|----------------------------------|----------------------------------------------------------|
| | Unadjusted analysis ^a | Analysis adjusted for systemic inflammation ^b |
| | 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)*** |

^aAdjusted 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.