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)

measurement was calculated for each study, along with standard deviations for each data set. The mean weighted bias was subsequently calculated using the formula (Bias × N)/N

Results Mean weighted bias for the populations studied was 0.06 kPa (± 2SD 2.40 kPa).

Discussion Patients with hypercapnoeic respiratory failure undergoing non-invasive ventilation (NIV) require regular measurements of PCO_2 and pH status in order to optimise treatment. The current gold standard for measurement of PCO_2 is arterial blood gas sampling. In critical care environments this necessitates placement of an indwelling arterial catheter, a painful procedure which carries a risk of complications including thrombotic occlusion, distal ischaemia, infection and pseudoaneurysm formation. Whilst accurate, arterial PCO_2 measurement provides a single, static reading of an inherently dynamic process in the context of hypercapnoeic respiratory failure.

Transcutaenous PCO_2 monitoring may prove a suitable and reliable non-invasive method for analysing partial pressure of carbon dioxide, with good approximation to arterial values. Our results suggest that transcutaneous CO_2 monitoring may be used as a reliable surrogate to arterial CO_2 measurements in specific patient populations undergoing NIV. However, variance is wide and further studies are needed to establish whether $PtcCO_2$ is a sufficiently accurate marker on which to base treatment decisions.

P182 COPD: IS IT ALL IN VEIN?

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Introduction & Objectives In patients with COPD, an arterial blood gas (ABG) is considered the gold standard method of directly measuring serum pH, pO_2, pO_2 and calculating bicarbonate (HCO_3) levels. These values allow the assessment of a patient's acid base status and adequacy of ventilation and oxygenation. The aim of this study was to identify if a venous blood gas can accurately reflect an arterial blood gas in determining the ventilatory function of patients presenting with acute exacerbations of COPD.

Methods This prospective observational study was conducted in a Scottish urban ED. All consecutive patients presenting with acute exacerbations of COPD were eligible. An ABG was taken from each patient, as deemed necessary by the treating physician, along with a venous gas at the time of venepuncture. Pearson's correlation coefficient and Bland Altman analysis methods were used to identify correlation and agreement between the arterial and venous data sets.

Results 68 paired samples were obtained over a two month period. Correlation was strong (r = 0.953) between arterial and venous pH. Bland Altman analysis showed an average difference (bias) of 0.017, with 95% limits of agreement (LOA's) of -0.052 to 0.087. Arterial and venous values for HCO_3 were also strongly correlated (r = 0.914). The agreement was -0.834mmol/l with 95% LOA's of -4.82 to 3.15 mmol/l. Despite arterial and venous pCO_2 strongly correlating (r = 0.973), the agreement bias was 4.66mmHg with 95% LOA's of -4.94 to 14.26 mmHg. Arterial hypercarbia, defined as $pCO_2 > 45$ mmHg, was present in 31 patients (46%). All cases of arterial hypercarbia were detected on venous blood gas sampling using a pCO_2 screening cut-off of 45mmHg. This was found to be 100% sensitive (95% CI 89–100%) and 86% specific (95% CI 71–95%).

Conclusion There is a strong correlation between arterial and venous pH, pCO_2 and HCO_3. Agreement between pH and HCO_3 is acceptable enough to substitute a venous blood gas value for an arterial blood gas value. However, venous pCO_2 does not agree with arterial pCO_2, therefore cannot be substituted. A venous pCO_2 screening cut-off of 45 mmHg has 100% sensitivity in detecting arterial hypercarbia. Had a venous blood gas been performed initially, 47% of ABG's could be avoided in these patients.

P183 AN OFF-LINE END-TIDAL BREATH SAMPLING METHOD IN ANAESTHETISED PATIENTS WITH ANALYSIS BY SELECTED ION FLOW TUBE MASS SPECTROMETRY

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Introduction Monitoring of breath volatiles could provide non-invasive rapid assessment of metabolic stress, inflammation and intravenous anaesthetic drug concentrations in anaesthetised patients. This study was designed to validate an off-line single breath end-tidal sampling method with analysis by selected ion flow tube mass spectrometry (SIFT-MS). Exhaled acetone was used as a model compound due to its abundance in breath and ease of measurement.

Methods End-expiratory breath samples from 22 healthy, non-diabetic, elective and semi-elective surgical patients were collected into Tedlar® bags via a T-piece adjacent to the endotracheal tube. The effects of different breathing systems, the breathing circuit filter, and consequences of altering the inspiratory gas flow rate and adjustable pressure limiting (APL) valve on exhaled acetone concentrations were explored in subgroups of these patients.

Results Median exhaled acetone concentration was 738 ppb (range 257–6594 ppb) for samples collected on the patient side of the circuit filter with the APL valve open (usual position) (n = 22). Median intra-subject coefficient of variation for exhaled acetone concentration using this method was 8.3% (interquartile range 6.9–14.5%). Higher inspiratory but not exhaled acetone concentrations were seen when using the ADU Carestation compared to the Aysis Carestation anaesthesia machines (median inspiratory concentration 276 ppb v 131 ppb, p = 0.0005; median exhaled concentration 630 ppb v 513 ppb, p = 0.95). Altering the inspiratory gas flow rate did not significantly affect exhaled acetone concentration; however APL valve closure resulted in a reduction in exhaled acetone concentration. Higher concentrations of acetone were measured in breath samples collected from the patient side of the circuit filter, since collection of samples after the filter resulted in dilution by deadspace air. Breath acetone concentration was related to plasma acetone (r_s = 0.80, p < 0.0001) and plasma beta-hydroxybutyrate concentrations (r_s = 0.49, p = 0.029).

Conclusions This non-invasive method of end-tidal breath collection in anaesthetised patients is reproducible for the analysis of acetone and is suitable for repeated sampling. With appropriate validation, the same method could be applied to the collection of other volatiles in the breath of intubated and ventilated patients, making it possible to investigate the concentrations of other potential biomarkers in this patient group.