during awake CPAP titration to quantify the effect of chest inflation on the load of the respiratory system.

Patients and methods Obese patients (body-mass-index, BMI >30) with confirmed obstructive sleep apnoea (OSA) were studied and NRD ($sEMG_{para}$) and the surface EMG of the external oblique ($sEMG_{abd}$) were recorded and normalised to baseline activity (awake, supine). The apnoea-hypopnoea index (AHI) and 95th percentile of CPAP were determined in sleep studies. The patients were then studied whilst awake and breathing on CPAP (4–20 cmH_2O, increments of 2 cmH_2O/3 mins), with the modified Borg score (mBorg) recorded.

Results 15 patients (age 48 ± 10 years, 12 male, BMI 38.9 ± 5.8) suffering with moderate-severe OSA (AHI 32.2 ± 21.1/h, 95th percentile nocturnal CPAP 14.1 ± 3.8 cmH_2O) were studied. Awake, $sEMG_{para}$ declined by 15.1 ± 1.5% from baseline when CPAP was applied, with the nadir at a CPAP of 10.6 ± 3.4 cmH_2O (p = 0.026). Further increase in CPAP levels led to a rise in $sEMG_{para}$ and breathlessness (mBorg at lowest $sEMG_{para}$ 0.9 ± 0.8 points, at CPAP of 20 cmH_2O 2.7 ± 2.7 points, p = 0.02).

Conclusion The respiratory system is maximally offloaded with subtherapeutic CPAP levels in OSA. Levels of NRD observed at effective CPAP levels are associated with breathlessness which can impact on CPAP compliance.

S49 VENTILATORY IRREGULARITY QUANTIFIED BY APPROXIMATE ENTROPY IDENTIFIES DISORDERED BREATHING IN PATIENTS WITH UNEXPLAINED DYSPNOEA

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10.1136/thoraxjnl-2015-207770.55

Introduction Patients with idiopathic persistent exertional dyspnoea are often labelled as having a breathing pattern disorder (BPD). There are no agreed objective diagnostic measures for BPD, which complicates its characterisation and response to therapy. Approximate entropy (ApEn) is a measure of unpredictability, based on chaos theorem, which quantifies the degree of irregularity in time-series data.

Objectives To measure ApEn of ventilatory variables during a cardiopulmonary exercise test (CPET) in patients referred with unexplained dyspnoea. We hypothesised that ApEn of tidal volume and breathing frequency would be greater (i.e. more irregular) in patients with BPD than healthy controls.

Methods We studied 20 adults (14 female) with unexplained dyspnoea referred for CPET and diagnosed with BPD (by a senior respiratory physiotherapist blinded to ApEn data) and 15 age-gender- and BMI-matched healthy controls. Underlying cardiorespiratory disease was excluded using various investigations (e.g. imaging and echocardiography) prior to referral, in addition to tests performed on the day of CPET; namely pulmonary function and blood gas analysis. ApEn of various ventilatory parameters including tidal volume, breathing frequency and minute ventilation was calculated at rest and during a cycle-ergometer CPET.

Results BPD patients had greater dyspnoea (modified BORG) at rest (1.4 ± 1.2 vs 0.2 ± 0.6; P < 0.01) and lower peak oxygen uptake (VO_2) (P < 0.01; Table 1). Peak exercise respiratory exchange ratio was similar between groups (1.14 ± 0.17 vs 1.13 ± 0.08, P = 0.84) as were nadir values for ventilatory equivalent for CO_2 (28.5 ± 5.2 vs 25.5 ± 3.6, P = 0.07) and end-exercise arterial PCO_2 (4.21 ± 0.68 vs 4.1 ± 0.67, P = 0.68). ApEn was significantly greater in the BPD cohort for the duration of the test (Table 1); however differences were not apparent at rest. There was no relationship between ApEn and baseline symptom scores.

Conclusion Measurement of ventilatory ApEn in patients referred with unexplained dyspnoea quantified irregularity of breathing pattern and was significantly greater (more irregular) in BPD than controls. These differences were not apparent from resting phase analysis. Quantifying increased dys-regulation in exercise hyperpnoea using ApEn can be applied to ventilatory variables collected during standard CPET, and thus could aid in diagnosis and evaluating treatment response in BPD. Further work should explore how ventilatiory ApEn relates to perception of symptoms.

Abstract S49 Table 1 Participant characteristics and exercise responses

	BPD (N = 20)	Healthy Controls (N = 15)
Age (years)	49 (14)	50 (18)
BMI (kg/m^2)	26.0 (5.0)	24.5 (3.7)
FEV_1 (% predicted)	107 (18)	95 (18)*
FEV_1/FVC	0.78 (0.06)	0.75 (0.12)
VO_2/kg Peak (ml/min/kg)	20.7 (7.1)	37.9 (14.9)**
VO_2 Peak (% Predicted)	79.8 (17.5)	124.8 (27.3)**
ApEn Tidal Volume	1.31 (0.23)	1.04 (0.28)**
ApEn Breathing Frequency	1.42 (0.22)	1.24 (0.24)*
ApEn Minute Ventilation	1.01 (0.29)	0.64 (0.22)**

S50 UNDERSTANDING HEROIN OVERDOSE: A STUDY OF THE ACUTE RESPIRATORY DEPRESSANT EFFECTS OF INJECTED PHARMACEUTICAL HEROIN

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10.1136/thoraxjnl-2015-207770.56

Introduction and objectives Opioids are respiratory depressants and heroin/opioid overdose is a major contributor to the excess mortality of heroin addicts. The individual and situational variability of respiratory depression caused by intravenous heroin is poorly understood. The aim of this study was to use advanced physiological monitoring to follow the time course and severity of acute opioid-induced respiratory depression.

Methods 10 patients (9/10 with chronic airflow obstruction) undergoing supervised injectable opioid treatment for heroin addiction received their usual prescribed dose of injectable opioid (diamorphine or methadone) (IOT), and their usual prescribed dose of oral opioid (methadone or sustained release oral morphine) after 30 min. The main outcome measures were pulse oximetry (SpO_2%), end-tidal CO_2% ($ETCO_2$%) and neural respiratory drive (NRD) (quantified using parasternal intercostal muscle electromyography). Significant respiratory depression was defined as absence of inspiratory airflow >10s, SpO_2% <90% for >10s and $ETCO_2$% per breath >6.5%.

Results $ETCO_2$% indicated significant respiratory depression following IOT in 8/10 patients, with levels increasing from baseline 4.7 (4.5 to 5.4)% to 5.4 (5.1 to 5.7)% at 30 min, p = 0.01. The median (range) peak $ETCO_2$% per breath was 6.9% (5.2 to 7.8).