Nocturnal asthma monitoring by chest wall electromyography

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ABSTRACT

Rationale Patients with suboptimal asthma control often have nocturnal symptoms which wake them, causing sleep fragmentation.

Objectives It was hypothesised that symptomatic patients were more accurately identified by measuring respiratory effort using chest wall electromyography than by pulmonary function testing.

Methods Nocturnal electrical activity of the parasternal intercostal muscles (EMG_{para}) in the second intercostal space was measured in subjects with controlled (diurnal peak expiratory flow (PEF) variability <20%, n=12) and uncontrolled (diurnal PEF variability >20%, n=12) asthma, and it was compared with that in normal subjects (n=12).

Results Subjects with controlled and uncontrolled asthma did not differ significantly in age (mean (SD) 42 (17) vs 46 (17) years, p=0.64), body mass index (BMI; 26.6 (2.9) vs 27.5 (3.5) kg/m^2, p=0.48) or gender distribution (males:females, 6:6 vs 7:5, p=0.68); the normal subject group was younger (27 (11) years, p=0.011) and slimmer (BMI 21.1 (2.9) kg/m^2, p<0.001). An elevated respiratory disturbance index (RDI) was associated with poor asthma control (RDI in normals 0.5 (0.9), in controlled asthma 4.0 (1.9), p<0.001, and in poorly controlled asthma 7.4 (4.3) h^{-1}; p<0.021). Similarly, EMG_{para}%max (normals 4.9 (3.2)% max evening, 4.9 (3.5)%max morning) was higher in controlled asthma (7.2 (2.3)%max evening, 8.1 (4.0)% max morning, p=0.049) and higher still in uncontrolled asthma (16.8 (14.2)%max in the evening, 18.4 (11.6)% max in the morning, p<0.008).

Conclusions Nocturnal respiratory effort is increased in those with asthma and neural respiratory drive is more variable in patients with poorly controlled asthma. Changes in the EMG_{para} inversely reflect changes in pulmonary function tests. Measuring the EMG_{para} offers a method to monitor asthma breath-by-breath while subjects are asleep, which could be adapted for home use.

Key messages

What is the key question?

- We hypothesised that patients with symptomatic asthma were more accurately identified by measuring respiratory effort using chest wall electromyography than by pulmonary function testing.

What is the bottom line?

- Nocturnal respiratory effort is increased in those with asthma, and neural respiratory drive, as measured by the surface electromyogram of the parasternal intercostals, is more variable in patients with poorly controlled asthma, inversely reflecting changes in pulmonary function tests.

Why read on?

- This article explores the relationship between nocturnal asthma, neural respiratory drive and pulmonary function tests, and highlights a non-volitional way of monitoring respiratory effort.

INTRODUCTION

While asthma can, in most cases, be controlled by adjusting medication in response to symptom enquiry and review of peak flow measurements, there remain a minority of patients with persistent nocturnal symptoms, although the proportion is now not likely to reach the figure of 74% described by Turner-Warwick.[1] The coefficients of variation for the nocturnal symptoms are associated, probably causally, with sleep-disordered breathing,[2 3] and with increased asthma mortality.[4] A greater proportion of those with asthma die at night compared with the general population.[5]

Although peak expiratory flow (PEF) can be easily monitored at home or by the bedside, these recordings, which are made at convenient points of the diurnal cycle rather than at the peak and nadir of bronchoconstriction, underestimate the magnitude of diurnal variability.[6] To date a technique for continuously monitoring patients with asthma throughout the night has not been available.

The parasternal intercostal muscles are important extradiaphragmatic inspiratory muscles, whose activity increases with the severity of respiratory disease.[7 8] Physiological abnormalities typical of asthma include increased airway resistance, altered chest wall and respiratory muscle geometry as a consequence of hyperinflation, increased neural respiratory drive and, therefore, increased respiratory muscle activity.[9 10] Worsening asthma would be expected to increase neural respiratory drive and reflect the sum of individual pathophysiological changes (airways obstruction, hyperinflation and increased positive end-expiratory pressure).[10]

Neural respiratory drive to the diaphragm has been quantified by measuring the diaphragm electromyogram (EMG_{di}) using a multipair oesophageal electrode.[11 12] The EMG_{di} is increased in chronic obstructive pulmonary disease (COPD) and asthma when compared with normal subjects.[9] EMG_{di}, measured continuously during sleep on a breath-by-breath basis, is a potentially useful method of monitoring respiratory disease severity.[13–15] Indeed

we have previously shown that EMG can in a specialised setting be used through the night to assess the impact of ventilatory support on sleep-disordered breathing.[16]

The invasive nature of EMG_{di} means that it is unlikely to be widely adopted, so we considered the possibility that chest wall electromyography (aiming at capturing parasternal activity) could be a useful monitoring tool in asthma, supported by recent work specific to childhood asthma by Maarsingh *et al* showing an inverse relationship between the EMG_{di} and parasternal intercostal EMG (EMG_{para}) and forced expiratory volume in 1 s (FEV_1) during histamine challenge.[17] The ability to accurately identify deterioration in patients not able to perform lung function tests, such as when asleep, could potentially allow treatment to be intensified, improve clinical care and reduce length of hospital stay, or, if applied at home, avoid admission.

We therefore hypothesised that neural respiratory drive measured by chest wall surface electromyography may allow the severity of nocturnal asthma to be assessed, and sought to evaluate this in adult patients with asthma with good and poor asthma control, and healthy volunteers.

PATIENTS AND METHODS

We measured nocturnal electrical activity of the parasternal intercostal muscles (EMG_{para}) bilaterally in the second intercostal space, as described elsewhere,[7 8 17] using surface electrodes (Tyco Healthcare, Neustadt ad Donau, Germany) in those with controlled (diurnal PEF variability <20%, n=12) and uncontrolled (diurnal PEF variability >20%, n=12) asthma, and compared it with that of normal subjects without airways obstruction (n=12). Subjects were non-smokers and aged >18 years. The two asthma groups were recruited from outpatient clinics and via newspaper advertisements. The asthma groups were not matched, but we recruited patients to have a similar distribution in age, height, weight and gender. Patients were asked not to take any long-acting β-agonists on the day preceding the sleep study and were restarted following the last measurement on the morning after the sleep study. The normal subjects were recruited from a population of young healthy students. The study was approved by King's College Hospital Local Research Ethics Committee and each participant gave written informed consent.

Protocol

1. Patients completed an Asthma Control Questionnaire (ACQ).[18]
2. EMG_{para} was recorded over a 10 min period at rest, seated, wearing a noseclip in the evening (between 22:00 and 23:00 h) and in the morning (between 06:00 and 07:00 h).
3. Pulmonary function tests and earlobe blood gases were also measured in the evening and morning.
4. Following the baseline measurements in the evening (at 23:00 h), the EMG_{para} was continuously recorded throughout the night, during full polysomnography. Data were stored and analysed offline.

Analysis of EMG_{para}

The raw data of the EMG_{para} signal were converted to root-mean-square (RMS). The mean of the peak RMS of the EMG_{para} per breath was calculated, while awake for the final 5 min of each 10 min of resting breathing at 22:00–23:00 h and 06:00–07:00 h, and while asleep, analysing minutes 8–10/18–20/28–30 of each 30 min division of sleep time, reporting the mean for each 30 min epoch. The mean of the RMS of the EMG_{para} was transformed into percentage maximum EMG_{para} activity, dividing it by the maximum RMS EMG_{para} obtained in the evening, during one of four maximum respiratory manoeuvres. The maximum breathing manoeuvres were inspiration to total lung capacity (TLC), maximum sniff, maximum inspiratory pressure (PI_{max}) and maximal voluntary ventilation (MVV), all of which are known to achieve maximal or close to maximal EMG activity of the diaphragm.[19]

Statistical analysis

EMG data were normally distributed, baseline subject characteristics and physiological variables were reported as mean (SD), and comparisons between subject groups for overnight data were made with a one-way analysis of variance (ANOVA) and post hoc analysis using the Dunn correction for multiple comparisons. Where data were not normally distributed, they are presented as median (25th–75th centiles) and tested using a Kruskal–Wallis rank sum test. Statistical analysis of demographic data was made using paired and independent group t tests, as appropriate.[20] The primary outcome measures were $\%maxEMG_{para}$ overnight while asleep, and at 22:00–23:00 h and 06:00–07:00 h while awake.

Associations between mean RMS EMG_{para} and standard lung function variables,[21] and between each of these variables and the frequency of nocturnal arousals from sleep, were measured and compared. The mean EMG_{para} thoughout the night was assessed regarding absolute levels; deviation from baseline as a marker for variability of neural drive was calculated (RMS of successive deviation; RMS_{SD})[22] and compared between the groups. The area under the curve (AUC) was determined for EMG and pulmonary function tests to define asthma control, with the respiratory disturbance index (RDI) as dependent variable. Following this analysis, a stepwise linear regression analysis was performed entering PEF, FEV_1 and EMG_{para} data as independent parameters to establish independent predictors for AHI. SPSS 16.0 for Mac was used for analysis of the data, and MS Excel for Mac to create figure 2. Parameters in tables 1 and 3–5 are shown as mean (SD), data in table 2 and the respiratory disturbance index (RDI) in table 3 are presented as median (25th–75th centiles) because

Table 1 Demographics, questionnaire data and blood gas values

	Normal subjects	Controlled asthma	Uncontrolled asthma
Age (years)	26.6 (10.8)*	42.3 (16.6)	45.6 (17.1)
BMI (kg/m^2)	21.1 (2.9)†	26.6 (2.9)	27.5 (3.5)
Neck circumference (cm)	34.4 (3.3)‡	37.8 (3.2)	39.2 (4.2)
Gender (M:F)	7:5	6:6	7:5
Waist/hip ratio	0.83 (0.06)	0.89 (0.11)	0.98 (0.09)§
Epworth Sleepiness Scale (points)	2.1 (0.9)*	6.0 (4.2)	8.6 (4.9)
SABAs (n/12)	0	9	10
LABAs (n/12)	0	3	11
Inhaled corticosteroid (n/12)	0	6	12
Pao_2 (kPa)	11.8 (0.4)	12.0 (1.5)	10.6 (1.8)§
$Paco_2$ (kPa)	5.0 (0.2)	4.9 (0.4)	5.2 (0.7)
pH	7.40 (0.01)	7.43 (0.02)	7.41 (0.02)
HCO_3^- (mmol)	24.0 (0.4)	23.7 (1.1)	24.3 (2.7)

Comparisons are based on results from ANOVA.
*Difference between normal group and controlled asthma ($p<0.01$).
†Difference between normal group and controlled asthma ($p<0.001$).
‡Difference between normal group and controlled asthma ($p<0.05$).
§Difference between uncontrolled versus controlled asthma and uncontrolled asthma versus normal group ($p<0.05$).
BMI, body mass index; F, females; LABAs, long-acting β-agonists; M, males; SABAs, short-acting β-agonists.

Table 2 Asthma Control Questionnaire data

	Normal	Controlled asthma	Uncontrolled asthma
All questions	0.0 (0–0)*	1.0 (0–1)*	2.0 (2–3.5)*
Nocturnal waking	0.0 (0–0)*	0.0 (0–1)*	2.0 (1–2)*
Morning symptoms	0.0 (0–0)*	1.0 (0–1)*	2.0 (2–3)*
Activity limitation	0.0 (0–0)*	0.5 (0–1)*	1.5 (1–2)*
Short of breath	0.0 (0–0)*	1.0 (1–1)*	2.0 (2–3)*
Wheeze	0.0 (0–0)*	1.0 (0.25–1)*	2.0 (1.25–2.75)*
FEV_1 %pred	0.0 (0–0)*	1.0 (1–2)*	4.0 (4–5)*
β2-agonist alone	0.0 (0–0)*	1.0 (0.25–1)*	3.5 (3–4)*

Total and single item questionnaire data, presented as median (25th–75th centiles). There was a significant difference between the groups.
*$p<0.001$ for each single question and the total score.
FEV_1, forced expiratory volume over 1 s.

they were not normally distributed; table 6 reports the SE. A p value <0.05 was chosen as level of significance.

RESULTS

Out of 40 subjects who were initially screened, three subjects with controlled and one with uncontrolled asthma did not agree to participate in the study. The remaining 36 subjects were a group with controlled asthma (n=12), uncontrolled asthma (n=12) and normal subjects (n=12). Those with controlled and uncontrolled asthma did not differ significantly in age, BMI or gender distribution, but the control group was younger and lighter. In addition to PEF variability, asthma control, as measured by the ACQ, differed between the groups. Patients with asthma had more daytime sleepiness, as measured with the Epworth Sleepiness Scale, and those with uncontrolled asthma had slightly lower blood oxygen tensions (tables 1 and 2).

Sleep disturbance differed between the groups, with normal subjects having normal sleep, those with controlled asthma being close to the conventional threshold for sleep-disordered breathing (five respiratory events/h), and those with uncontrolled asthma having reduced rapid eye movement (REM) sleep time and increased RDI in REM and non-REM sleep (table 3). The highest RMS EMG_{para} values were generated during maximal sniff manoeuvres (table 4).

Neural respiratory drive while awake, as measured by the EMG of the parasternal intercostals, was elevated in those with uncontrolled asthma at the beginning of the night, and when waking up in the morning; those with controlled asthma had mildly elevated levels of EMG_{para} only in the morning. Lung function results in the evening and in the morning reflected the groups' characteristics, with decreased FEV_1 and PEF indicating increased airway obstruction (table 5).

The coefficient of variation for the EMG_{para} signal throughout the night was comparable between the normal subjects and the

Table 3 Polysomnography data

	Normal subjects	Controlled asthma	Uncontrolled asthma
TST (min)	392.8 (103.3)	370.7 (109.6)	305.9 (86.1)
Sleep efficiency (%)	80.4 (10.8)	81.6 (15.0)	77.9 (14.8)
REM sleep (min)	80.2 (20.3)	82.7 (38.1)	55.4 (19.6)*
RDI total	0.0 (0–0.8)†	4.3 (2.4–5.5)†	7.4 (4.4–9.7)†
RDI during REM sleep	0.0 (0–0.6)†	2.1 (0.3–4.3)†	6.5 (3.5–9.2)†

There was no significant difference in overall sleep time, but REM sleep duration was reduced in uncontrolled asthma and RDI was elevated in those with asthma.
*Difference between normal group versus uncontrolled asthma ($p=0.005$) and controlled versus uncontrolled asthma ($p=0.038$).
†Significant difference ($p<0.001$).
RDI, respiratory disturbance index, non-normally distributed and presented as median (IQR); REM, rapid eye movement; TST, total sleep time.

Table 4 Parasternal EMG

	Normal subjects	Controlled asthma	Uncontrolled asthma
$EMG_{para,\ TLC}$	90.0 (28.3)*	56.0 (22.2)	48.0 (32.5)
$EMG_{para,\ Sniff}$	90.9 (43.1)	74.6 (30.2)	54.0 (32.3)
$EMG_{para,\ PImax}$	60.5 (22.4)	54.3 (41.9)	39.7 (33.3)
$EMG_{para,\ MVV}$	61.8 (16.7)	46.7 (32.9)	36.8 (24.8)

Maximum inspiratory EMG of the parasternal intercostals during four different maximal respiratory manoeuvres. All EMG data are in μV. The highest root-mean-square mean EMG_{para} was achieved using sniff manoeuvres.
*Difference between normal group versus controlled asthma ($p<0.05$).
EMG_{para}, electromyogram of the parasternal intercostals; MVV, maximum voluntary ventilation; PI_{max}, maximum inspiratory pressure; TLC, total lung capacity.

subjects with controlled asthma (32.7% (10.9%) vs 35.3% (9.1%)), and was significantly higher in the subjects with uncontrolled asthma (58.2% (16.4%), $p<0.001$; figures 1 and 2, table 5). The variability of the pulmonary function test results was lower in each group than the variability in EMG_{para} recordings, with a range of the coefficient of variation for normal subjects between 5% and 24%, for the subjects with controlled asthma 8–35%, and for the subjects with uncontrolled asthma 12–34%. The measure of EMG_{para} for successive deviation from baseline as an additional marker for variability thoughout the night was EMG_{para} RMS_{SD} 2.83 (2.16) for normal subjects, 3.53 (1.51) for those with controlled asthma and 5.38 (3.37) for those with uncontrolled asthma ($p=0.048$ for controlled vs uncontrolled asthma).

The group data of the mean RMS %maxEMG_{para} indicated that normal subjects had stable EMG activity throughout the night (figure 2). In contrast, in subjects with controlled and uncontrolled asthma EMG_{para} activity fell in the early hours of the morning and rose prior to waking (figures 2 and 3). This pattern was most pronounced in those with uncontrolled asthma.

Comparison of techniques

To predict nocturnal asthmatic control we compared the AUC for receiver operating characteristics (ROC) curves predicting sleep fragmentation and disturbance. We entered pulmonary function tests, EMG_{para} in the evening and morning, and variability of the EMG signal overnight (RMS_{SD}) into the equation, with sleep-disordered breathing (RDI>5/h) as the dependent variable. The AUC for the EMG signals was similar to that for lung function parameters with no significant differences (table 6).

A stepwise linear regression analysis using RDI as dependent variable with EMG_{para}, FEV_1 and PEF entered as independent

Table 5 Parasternal EMG and pulmonary function test results

	Normal subjects	Controlled asthma	Uncontrolled asthma
EMG_{para} (%max) evening, awake	4.9 (3.2)	7.2 (2.3)	16.8 (14.2)*
EMG_{para} (%max) morning, awake	4.9 (3.5)	8.1 (4.0)†	18.4 (11.6)†
FEV_1 (%pred) evening	96.1 (10.4)	87.4 (13.9)	63.8 (13.3)‡
FEV_1 (%pred) morning	95.9 (11.3)	85.3 (14.5)	62.1 (13.3)‡
PEF (l/min) evening	492 (107.4)	404 (137.7)	360 (114.7)
PEF (l/min) morning	490 (107.6)	390 (134.8)	347 (119.0)
FEV_1/VC (%) evening	88.8 (4.8)	69.8 (10.2)§	62.3 (19.0)
FEV_1/VC (%) morning	89.0 (4.5)	67.3 (9.9)§	62.5 (18.3)

Changes in the EMG activity and pulmonary function tests. Evening (22:00–23:00 h) and morning (06:00–07:00 h) parameters represent the mean of the root-mean square %maxEMG_{para} activity.
*Difference between controlled versus uncontrolled asthma ($p<0.05$).
†Difference between normal group and controlled asthma ($p<0.05$) and controlled versus uncontrolled asthma ($p<0.01$).
‡Difference between controlled versus uncontrolled asthma ($p<0.001$).
§Difference between normal group vs controlled asthma ($p<0.001$).
FEV_1, forced expiratory volume over 1 s; PEF, peak expiratory flow; VC, vital capacity.

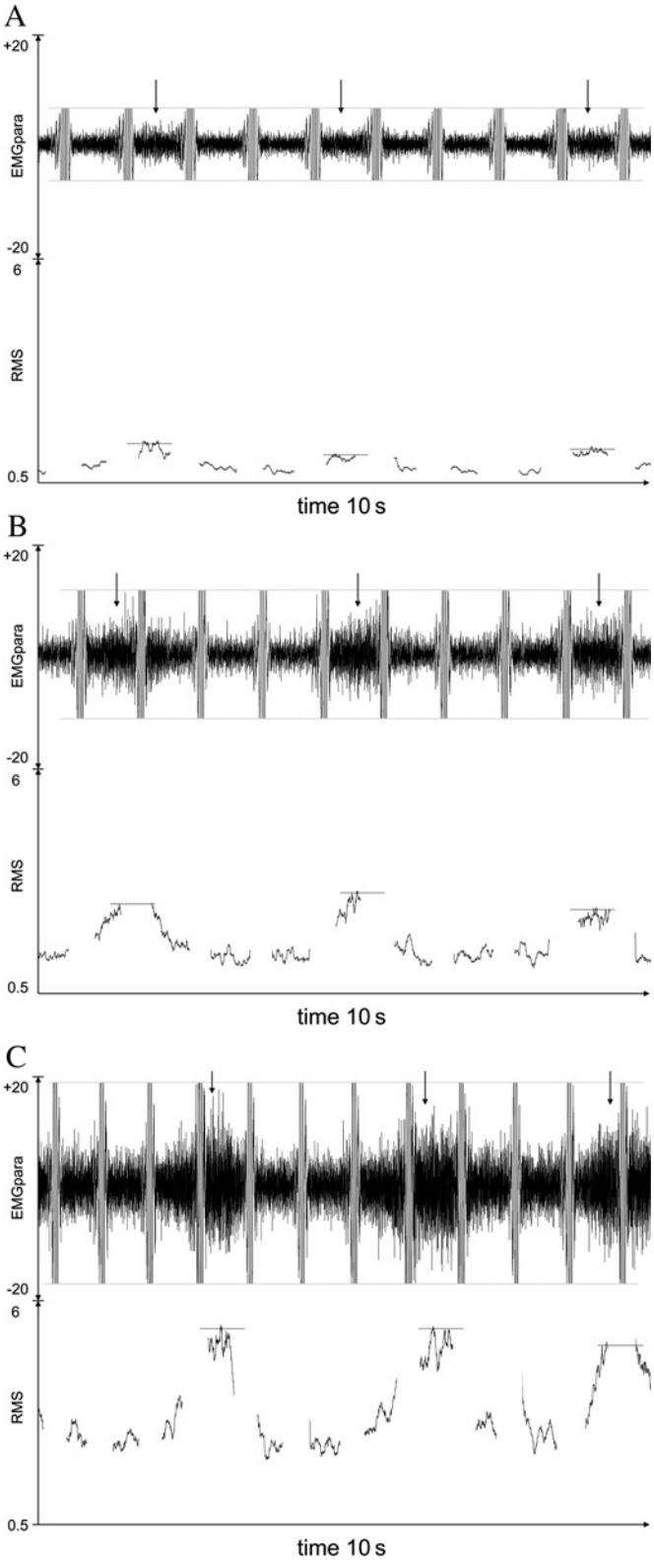

Figure 1 Ten second period of parasternal electromyogram (EMG) recordings asleep in a normal subject (A), one with controlled asthma (B) and one with uncontrolled asthma (C). The ECG artefact from the QRS-complex can be easily seen in the raw data (upper trace; EMG_{para} in μV) and has been truncated (horizontal dashed lines); the artefact was eliminated from the lower trace (root-mean-square (RMS) in μV) analysed with a time constant of 100 ms and a moving window). Inspiratory muscle activity is indicated by the arrows and maximal RMS values are marked by the dotted horizontal bars. The same scales on both axes were used for all three groups.

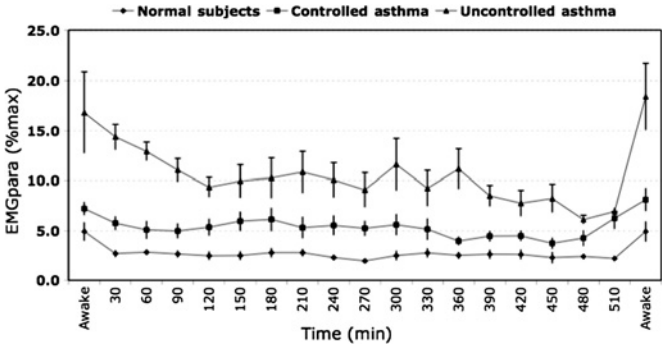

Figure 2 Data (mean±SE of the mean, SEM) of the parasternal electromyogram (EMG), awake and asleep. The recordings start at ~22:00–23:00 h in the evening and finish at ~06:00–07:00 h in the morning; epochs on the *x*-axis indicate 30 min each. Normal subjects and those with controlled asthma have a similar SE, indicating the variability of the EMG; subjects with uncontrolled asthma have the highest variability of the signal. While normal subjects stay on the same level of drive for most of the night and have a small drop in EMG activity in the morning, subjects with asthma have lower levels of activity than during the daytime until the early morning, the uncontrolled group more than the controlled group, with EMG activity rising to initial levels at the end of the night.

variables revealed an R^2 of 0.311 (R=0.558; SE of the estimate=3.247), and EMG_{para} variability overnight (EMG_{para} RMS_{SD}; β coefficient −0.023; 95% CI −0.045 to −0.019; p=0.005) and ΔFEV_1 (β coefficient −0.402; 95% CI −2.711 to −0.310; p=0.019) were identified as independent predictors for AHI.

DISCUSSION

Chest wall electromyography allows non-invasive and continuous recording of inspiratory muscle activity throughout the night. Neural respiratory drive, as measured by the EMG activity of the parasternal intercostals, is elevated in those with both controlled and uncontrolled asthma. Changes in EMG activity from evening to early morning correlate with asthma control and nocturnal symptoms, and reflect pulmonary function tests performed at the same time. It is of interest that subjects with asthma had a decrease in their average neural respiratory drive and higher levels of variability in the early morning hours (figures 1 and 3), a time that is known to be associated with increased symptoms.[1]

The high variability of the EMG_{para} signal during the night in patients with uncontrolled asthma suggests that the signal could be a sensitive marker of changes in disease severity. During sleep, the measurement of inspiratory muscle activity by recording EMG_{para} enables monitoring of sleep quality and asthma control without awakening patients.

Nocturnal asthmatic symptoms may be triggered by problems that can be more severe at night, including gastro-oesophageal reflux, postnasal drip syndrome and sleep apnoea.[23–26] Sleep studies reliably record ventilatory effort and breathing while asleep, using flow measurements, and recording abdominal and thoracic movements, thereby providing information on nocturnal asthma control. However, sleep studies, including electro-encephalography to measure arousal from sleep, require experienced staff and are time consuming and expensive. Measuring inspiratory muscle activity from surface electrodes has the advantage of being non-invasive and easily recordable. In addition, the set-up used for this study may be helpful for

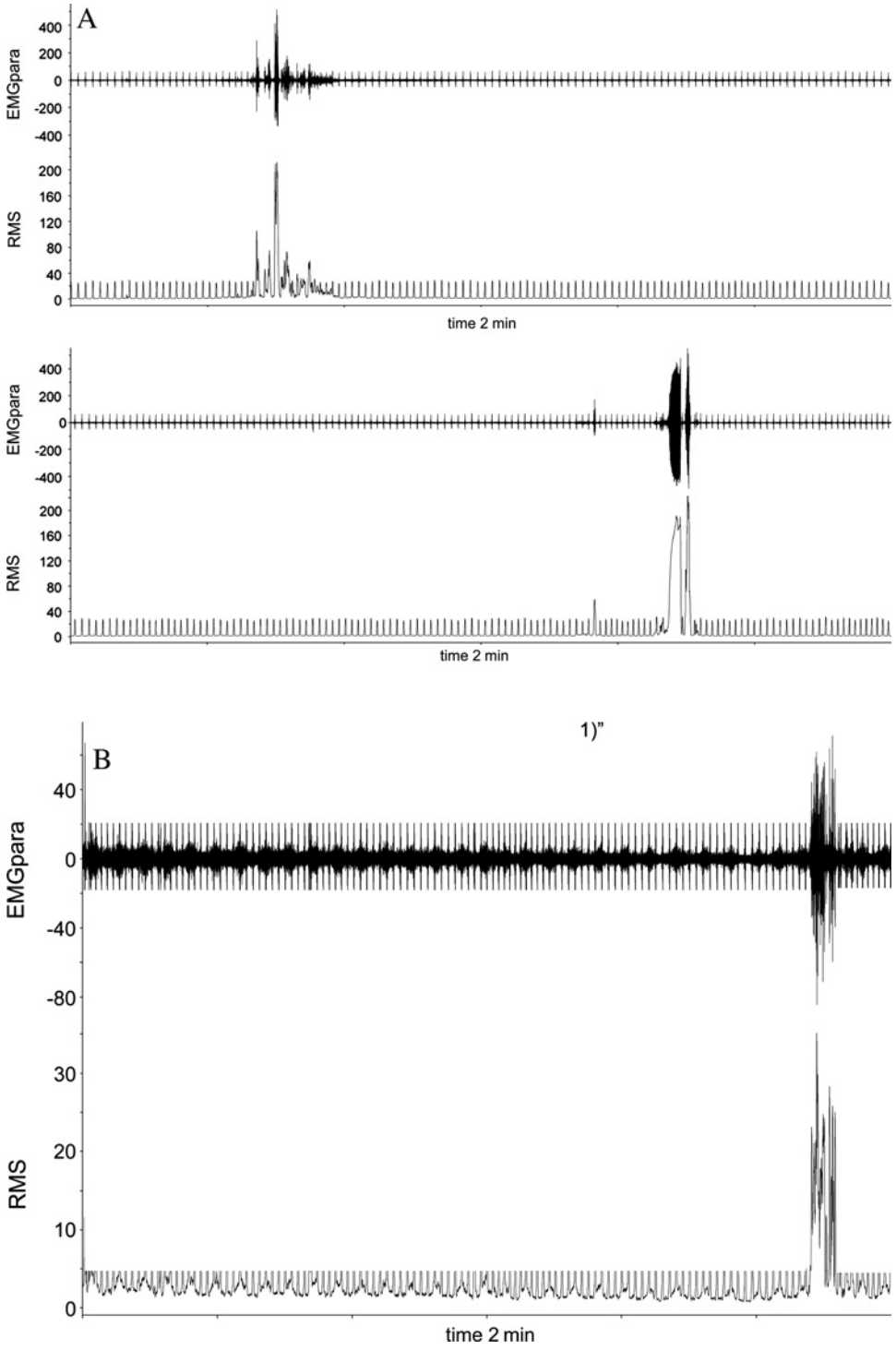

Figure 3 Representative recordings from the parasternal electomyogram (EMG; upper channel) and root-mean-square (RMS; lower channel) over two periods of 2 min (A) taken in the early morning from a subject with uncontrolled asthma while having fragmented sleep. Arousals can be observed interrupting the regular pattern of activity in each section. (B) An epoch of 2 min ending with an arousal. During the breaths preceding inspiratory arousal in the EMG there is a slow and continuous decline of EMG magnitude (B). The QRS-complex of the ECG artefact can clearly be seen and has been truncated in (B).

monitoring inpatients, for example those on intensive care, those being treated with non-invasive ventilation and selected patients in the emergency room.

As a clinical tool, measuring inspiratory muscle activity is a useful parameter to adjust treatment and detect deterioration of ventilation rapidly. The introduction of neurally adjusted ventilator assist (NAVA)[27] has recently demonstrated that assessing neural activity and adjusting treatment to levels of neural respiratory drive may indeed be useful in the clinical setting.

Limitations

In this study, the control group was younger than the subjects with asthma. This could have contributed to the lower EMG values in these subjects. However, such a difference in age is known to have little effect on neural drive as measured by the EMG_{di}.[9] Similarly the variation in weight across the groups is unlikely to have significant impact because EMG_{para} in obese subjects has been reported previously, and is lower in morbidly obese subjects than in those with asthma.[28] Most of the respiratory events at night were non-obstructive, and the matching of age, gender and BMI between the controlled and uncontrolled group supports the hypothesis that differences in the RDI, EMG_{para} and symptom scores are secondary to asthma control rather than sleep apnoea.

An important limitation of this method is the reference of EMG at rest to maximal breathing manoeuvres. While we know

Table 6 Area under the curve (AUC) for EMG and lung function data

Variable	AUC	SE
EMG_{para} evening	0.783	0.084
EMG_{para} morning	0.706	0.089
FEV_1 evening	0.700	0.177
FEV_1 morning	0.721	0.167
PEF evening	0.684	0.095
PEF morning	0.672	0.097

AUC for receiver operating characteristics (ROC) curves. There was no significant difference between EMG and lung function parameters.
EMG, electromyogram; FEV_1, forced expiratory volume over 1 s; PEF, peak expiratory flow.

for the diaphragm that maximal or close to maximal EMG activity is achieved using respiratory manoeuvres as described (TLC, PI_{max}, sniff and MVV),[19] this has not been validated for the parasternal intercostals. The signal intensity during manoeuvres could reflect submaximal effort, or surface recordings could pick up EMG activity from muscles other than the parasternal intercostals. Relating the signal to change from baseline over the night, as well as variability, may overcome these criticisms. It is the qualitative information of whether respiratory effort has changed that is important for the clinician. NAVA,[27] a method recording the diaphragm EMG transoesophageally to adjust ventilator settings, is providing similar information to the clinician. The greater the increase and variability of the EMG signal the higher is the ventilatory load; it is this information that makes the measurement valuable in various clinical settings, not least for monitoring patients during sleep.

Conclusion

Absolute levels of respiratory drive are increased in patients with controlled and uncontrolled asthma overnight. The diurnal change in neural activity of the parasternal intercostals in asthma reflects changes in pulmonary function tests. Measurement of parasternal intercostal EMG could provide a continuous method of monitoring asthma severity during sleep.

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Competing interests None.

Ethics approval This study was conducted with the approval of the King's College Hospital Local Research Ethics Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

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