In summary, these findings suggest that acute exposure to biomass smoke in women with small airways disease or restrictive lung disease is associated with blood deoxygenation, suggesting that in these individuals continued exposures may increase the risk of disease exacerbation.

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Simulated driving performance coupled with driver behaviour can predict the risk of sleepiness-related car accidents

Obstructive sleep apnoea syndrome (OSAS) is associated with the risk of sleepiness-related car accidents and worse driving simulator performances. Nevertheless, driving simulator studies failed to predict the real-world accident risk. Two-thirds of patients with OSAS will never experience a car crash, so that restricting their driving licence because of an OSAS diagnosis is not feasible.[1]

Our previous studies validated a 30 min monotonous driving scenario (STISIM 300 Driving Simulator, Systems Technology, Hawthorne, California, USA) versus subjective and objective measures of sleepiness in healthy volunteers undergoing a sleep deprivation challenge and in patients with OSAS. Our test disclosed that primary vehicle control parameters (crashes, lane position variability) were the strongest predictors of sleepiness and, potentially, of crash risk.[2 3] We reviewed the driving simulator and sleepiness (maintenance of wakefulness test (MWT) and Epworth sleepiness scale (ESS)) data of 43 men (mean age 53±9 years) with severe OSAS (mean apnoea-hypopnoea index of 55±16/h) together with their responses to a questionnaire on driving history. Clinical features, self-reported behaviour and driving simulation data are reported in table 1, together with statistical comparisons (Mann–Whitney or χ^2 tests). Twenty patients (47%) had crashed in the previous 5 years and frequently considered sleepiness the main cause of their accident. They were rated sleepier on the ESS (p=0.038), and trended to crash sooner in the driving simulation (p=0.05), without any other difference. Therefore, the identification of patients at risk based only on laboratory measures is not feasible.

Twenty-eight (65%) patients continued to drive while sleepy ('risky behaviour'), without differing significantly from patients that stopped driving ('safe behaviour'). Within the 'risky behaviour' subgroup, the 14 (50%) patients with a crash history were sleepier according to the ESS (p=0.019) without reaching statistical significance on the MWT (p=0.062), crashed more frequently (p=0.036) and sooner (p=0.007) at the driving simulation, and showed worse tracking performance (p=0.017) than patients without self-reported crash history. Conversely, there were no differences between subjects with (40%) and without (60%) a crash history in the subgroup of 15 patients that used to stop driving while sleepy.

Previous driving simulator studies were controversial, showing either the presence[3 4] or the absence[5] of association between driving simulator performance and accident risk. These conflicting results could reflect the confounding effect of the unexplored

Table 1 Clinical features, self reported behaviour and driving simulation data of patients with and without crash history and in the subgroups with safe and risky behaviour

	All patients			Safe behaviour			Risky behaviour		
Clinical features	**No crash**	**Crash**	**p Value**	**No crash**	**Crash**	**p Value**	**No crash**	**Crash**	**p Value**
Age	53.9±7.4	52.6±9.9	NS	54.3±5.9	46.3±11.6	NS	53.6±8.5	55.3±8.1	NS
BMI	31.9±5.0	31.9±4.5	NS	34.7±6.0	33.8±5.7	NS	30.2±3.5	31.1±3.9	NS
AHI	51.8±15.1	57.8±17.5	NS	52.2±17.2	57.9±17.1	NS	51.7±14.4	57.7±18.3	NS
ESS score	9.2±4.0	12.0±4.2	0.038	11.5±4.2	12.3±3.8	NS	7.7±3.3	11.8±4.5	0.019
Mean sleep latency on MWT (min)	23.4±14.3	16.1±12.7	NS	17.1±13.3	14.6±13.2	NS	27.4±13.9	16.7±13.0	[0.062]
Smoking (%)	17.4	36.8	NS	22.2	40.0	NS	14.3	35.7	NS
Habitual coffee drinking (%)	69.6	63.2	NS	77.8	100.0	NS	64.3	50.0	NS
Habitual alcohol drinking (%)	47.8	42.1	NS	55.6	40.0	NS	42.9	42.9	NS
Self-reported behaviour									
Driving >20000 km/year (%)	60.9	50.0	NS	44.4	50.0	NS	71.4	50.0	NS
Driving at night (%)	47.8	75.0	NS	33.3	66.7	NS	57.1	64.3	NS
Sleepy while driving at least monthly (%)	43.5	35.0	NS	66.7	50.0	NS	28.6	28.6	NS
Stopping driving while sleepy (%)	39.1	30.0	NS	100.0	100.0	NS	0.0	0	
Sleep attacks while driving at least monthly (%)	26.1	35.0	NS	33.3	50.0	NS	21.4	28.6	NS
Near-miss accidents in the last 5 years (%)	17.4	35.0	NS	0.0	16.7	NS	28.6	42.9	NS
Sleepiness-related accident (%)	0.0	70.0	<0.0001	0.0	66.7	0.004	0.0	71.4	0.0001
Mean driving simulator parameters									
Crashes (n)	0.5±1.0	2.7±5.9	NS	0.9±1.5	6.3±9.8	NS	0.2±0.3	1.2±2.4	0.036
Time to first crash (min)	27.1±5.1	22.3±8.6	0.050	14.2±7.1	22.3±10.8	NS	28.9±1.8	22.2±8.0	0.007
Lane position variability (m)	0.4±0.1	0.6±0.3	NS	0.5±0.1	0.7±0.5	NS	0.4±0.1	0.6±0.2	0.017

Continuous data are presented as mean±SD.
AHI, apnoea–hypopnoea index; BMI, body mass index; ESS, Epworth sleepiness scale score; MWT, maintenance of wakefulness test; NS, not significant.

individual behaviours of sleepy drivers. Our data, albeit from a limited number of patients with OSAS, support the reliability of a driving simulator approach for the identification of patients with OSAS at risk: poor performers have high risk if they keep on driving when sleepy. Accordingly, poorer simulated driving performance was associated with crash history only in our subjects with 'risky' behaviour. Nevertheless, the use of driving simulators is still recommended as a research tool given the absence of a standardisation that is the prerequisite for use in clinical practice.

Finally, crash risk is a multifactorial entity. Even if it is highly influenced by sleepiness, individual behaviours have a prominent effect in letting sleepiness determine a car accident. We emphasise that educational programmes, potentially involving driving simulators in different settings, remain the key instrument for risk management of sleepiness-related car accidents.

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Effect of acute hypoxia on QTc interval in respiratory patients undergoing fitness to fly tests

INTRODUCTION

Current UK guidelines recommend administration of in-flight supplemental oxygen to patients with chronic respiratory disease who have sea level arterial oxygen saturations <92% or partial pressure of oxygen (Pa_{O_2}) <6.6 kPa (50 mm Hg) during a hypoxic challenge fitness to fly test.[1] Hypoxia has been shown to prolong cardiac repolarisation, assessed by the QT interval corrected for heart rate (QT_c), and this may underlie the occurrence of potentially life-threatening cardiac arrhythmias[2–4]; however, few data exist about the cardiac response to hypoxia in patients with respiratory disease.

To establish whether hypoxia prolongs the QT_c, potentially increasing the risk of significant arrhythmias in patients with respiratory disease, we analysed data from respiratory patients referred to our lung function department for fitness to fly testing.

METHODS

Between 1 April 2008 and 27 February 2009, 101 patients (median age 57 years, range 20–87 years, 57.4% female) underwent hypoxic challenge (breathing 15% oxygen from a Douglas bag). Pulse oximetry was recorded continuously and an ECG recorded at baseline and after 15 min hypoxic exposure. In 65 patients (64.4%), capillary blood gases were analysed at the same time points. Further details are available online.

RESULTS

Disease aetiology was interstitial lung disease (39.6%), chronic obstructive pulmonary disease (COPD) (11.9%), bronchiectasis (11.9%), sarcoidosis (7.9%), cystic fibrosis (6.9%), systemic sclerosis (5.9%), asthma (5.0%), extrinsic allergic alveolitis (3.0%) and other chronic lung conditions (7.9%). Fifteen subjects (14.9%) had known cardiac disease.

Following hypoxic exposure, mean±SEM arterialised capillary P_{O_2} decreased from 10.56±0.14 kPa to 6.82±0.09 kPa ($p<0.001$) and mean arterial oxygen saturation (Sa_{O_2}) from 95.8±0.15% to 87.2±0.45% ($p<0.001$). Arterial carbon dioxide partial pressure, bicarbonate and transcutaneous carbon dioxide partial pressure also decreased ($p<0.05$, table 1).

Twenty patients (19.8%) became symptomatic during the test (combinations of dyspnoea, palpitations, nausea and dizziness). Eighty patients (79.2%) met the BTS criteria for use of supplemental oxygen in-flight.

Hypoxic challenge resulted in a significant increase in heart rate (from 83.2±1.48 bpm to 86.9±1.50 bpm; $p<0.001$) and decrease in PR interval (161.2±1.64 ms to 158.0±2.07 ms; $p=0.02$). In keeping, the QT interval decreased (357.8±4.08 ms to 348.8±3.49 ms; $p<0.001$). However, ECG frontal axis and QT_c did not change (415.2±2.57 ms to 417.0±2.39 ms; $p=0.50$).

There was no correlation between changes in QT_c and Pa_{O_2}/Sa_{O_2}. No patient suffered arrhythmias or ischaemic ECG changes. The presence of cardiac disease was not associated with differences in baseline measures or hypoxia response, including variation in QT_c. ECG responses did not differ between those who had capillary blood gases performed (n=65) and those who did not (n=36; $p>0.5$ in all cases)

DISCUSSION

Exposure to acute hypoxia (15% fractional inspired oxygen) is not associated with significant changes in cardiac QT_c in patients with chronic respiratory disease, in contrast to the QT_c prolongation seen in healthy subjects at altitude.[2 4 5] The absence of response might be due to hypoxic preconditioning[6 7] or drug effects upon autonomic efferent response (eg, salmeterol, ipratropium) or through other means (eg, renin-angiotensin system antagonists[8]). Given the association between prolonged QT_c and sudden death in COPD,[9] these data are reassuring to patients with chronic lung disease who wish to fly. However, further studies are needed to confirm these findings, as well as the effects of prolonged hypoxia and exercise.

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