Respiratory changes associated with rapid eye movements in normo- and hypercapnia during sleep

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Schäfer, Thorsten, and Marianne E. Schläfke. Respiratory changes associated with rapid eye movements in normo- and hypercapnia during sleep. J. Appl. Physiol. 85(6): 2213–2219, 1998.—Rapid eye movements during rapid-eye-movement (REM) sleep are associated with rapid, shallow breathing. We wanted to know whether this effect persisted during increased respiratory drive by CO2. In eight healthy subjects, we recorded electroencephalographic, electrooculographic, and electromyographic signals, ventilation, and end-tidal Pco2 during the night. Inspiratory Pco2 was changed to increase end-tidal Pco2 by 3 and 6 Torr. During normocapnia, rapid eye movements were associated with a decrease in total breath time by −0.71 ± 0.19 (SE) s (P < 0.05) because of shortened expiratory time (−0.52 ± 0.08 s, P < 0.001) and with a reduced tidal volume (−89 ± 27 ml, P < 0.05) because of decreased rib cage contribution (−75 ± 18 ml, P < 0.05). Abdominal (−11 ± 16 ml, P = 0.52) and minute ventilation (−0.09 ± 0.21 ml/min, P = 0.66) did not change. In hypercapnia, however, rapid eye movements were associated with a further shortening of total breath time. Abdominal breathing was also inhibited (−79 ± 23 ml, P < 0.05), leading to a stronger inhibition of tidal volume and minute ventilation (−1.84 ± 0.54 l/min, P < 0.05). We conclude that REM-associated respiratory changes are even more pronounced during hypercapnia because of additional inhibition of abdominal breathing. This may contribute to the reduction of the hypercapnic ventilatory response during REM sleep.

Irregular breathing is a common feature of rapid-eye-movement (REM) sleep. Since the original description of REM sleep by Aserinsky and Kleitman (3) in 1953, several studies have investigated the correlation of phasic eye movements and changes in respiratory patterns (1, 2, 4, 6, 9, 15, 16). Quantitative analyses by Millman et al. (15) and Gould et al. (9) showed that increasing eye-movement activity was associated with a reduction in tidal volume (VT) and a shortening of total breath time (TT), whereas inspiratory time (TI) was unchanged. This association of phasic events and changes in respiration causing the irregularity remained unchanged in the course of the night, although the frequency of events increased from early to late REM sleep (16).

We wanted to know whether these inhibitory effects on VT and the offsetting increase in respiratory rate were attenuated by hypercapnia during stimulated breathing, or whether they persisted and could contribute to an explanation for the diminished hypercapnic ventilatory response during REM sleep compared with wakefulness as described by Phillipson et al. (21) in the dog and Douglas et al. (7) in humans. We have, therefore, tested the effect of different levels of hypercapnia on the ventilatory pattern in the presence vs. absence of rapid eye movements during REM sleep in healthy subjects.

METHODS

Subjects

The study was performed in eight healthy men, who ranged in age from 24 to 32 yr. They were nonobese without a history of cardiac, pulmonary, neurological, or sleep-related breathing disorders. No subject was taking psychotropic drugs or other regular medications. Each participating subject gave informed and written consent. The study was approved by the ethics committee of the Faculty of Medicine at the Ruhr-University Bochum.

Protocol

Subjects reported to the sleep laboratory at 9 PM. Before formally entering the study, each subject underwent lung function testing and a general examination. Then they were prepared for polysomnography. Electrodes were attached to the scalp by using the international 10/20 system, to the skin of the nose, and to the submental regions according to Rechtschaffen and Kales (22). Thus two electroencephalographic (EEG) signals (C3–A2, C4–A1), a bipolar electrooculographic (EOG) signal, and a bipolar electromyographic (EMG) signal were continuously recorded on a polygraph (Schwarzer-Picker ED14 Digital). The EEGs and the EOG were filtered with time constants of 0.3 s (high-pass) and a low-pass filter of 30 Hz.

Ventilation was measured by pneumotachography (PT; ScreenMate, J. aeger, Würzburg, Germany) by using a comfortably fitting nose mask (nasal continuous positive airway pressure mask; Respironics) and was measured noninvasively by means of respiratory inductive plethysmography (RIP; Resptrace, Ambulatory Monitoring, Airdale, NY). Inductive bands were secured with adhesive tape around the chest and abdomen. To obtain the calibration factors for the inductive plethysmograph, the subjects performed isovolume maneuvers (14) in the supine position, during which gains of the rib cage (RC) and abdominal (Abd) channels were adjusted, until the resulting sum of the two signals (Sum) was zero. Afterwards, the three RIP signals (RC and Abd motion and Sum) were recorded, together with the PT signal, by using a mouthpiece and nose clip during 3 min of the subjects' breathing with different VT and compartmental volume to validate the RIP calibration. The correlation of RIP and PT was calculated by least squares linear regression. The regression coefficients were highly significant [r = 0.96 ± 0.02 (SD), P < 0.001] in all eight subjects.

The RIP and the PT signal flow and volume were recorded simultaneously on a chart recorder together with the EEG data and end-tidal PCO2 (PETCO2) and PO2 by means of an infrared CO2 and paramagnetic O2 analyzer (Normocap 200 Oxy, Datex, Helsinki, Finland). The PT was then attached to the outlet of a constant stream of gas flow (60 l/min) supplied by an air pump. The PT was calibrated by injection of 1 liter of air into the circuit at the constant background flow. The
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subject's nose mask was then attached to the circuit so that he could breathe from and into the background flow. This setup enabled easy changes of the inspired-gas composition without the need of valves. All data were fed into an analog-digital converter (CED 1401 plus, Cambridge Electronic Design, Cambridge, UK) and stored on hard disk. During the entire night's sleep, the measurements were monitored on the paper polygraph and the personal computer screen.

To get different steps of inspiratory and thus alveolar and arterial $P_{CO_2}$ during REM sleep, $CO_2$ was added to the inspiratory line so that the $P_{ETCO_2}$ was increased by 3 and 6 Torr, respectively, every 6–7 min. Thus one cycle with baseline $P_{ETCO_2}$ increased by 3 and 6 Torr, lasted 20 min.

Data Processing

At first the paper records were scored, according to the rules of Rechtschaffen and Kales (22), by two independent observers to identify clear REM sleep periods. Ventilation during these REM periods was analyzed breath by breath by using a computer-based analysis system developed in our laboratory on the basis of the Spike2 laboratory software package (CED). For each breath, the PT and the three RIP signals, RC, Abd, and Sum, as well as gas concentrations of $O_2$ and $CO_2$ in the inspired and expired air, were displayed consecutively on the screen, together with markers indicating the detected TI and expiratory times (TE) and amplitudes to check the computerized analysis. The program provided tabular listings of the following variables: 1) respiratory timing: described by $T_T$, $T_t$, $T_E$, and respiratory duty cycle ($T_t/T_T$); and 2) $V_T$: $V_T$ calculated by digital integration of the airflow signal obtained from the PT ($V_{PT}$), rib cage ($V_{RC}$), and abdominal ($V_{Abd}$) compartmental volumes from the RIP signal, as well as $P_{ETCO_2}$. The simultaneous EOG segments were analyzed by means of a fast Fourier transformation. The resulting total power density was a highly sensitive parameter to identify rapid eye movements. In parallel, a visual analysis of eye movements was performed by application of scoring criteria adapted from Neilly et al. (16) for bipolar recordings. By comparison with the visually obtained data, a threshold in total power density for detection of rapid eye movements was defined for each subject. Each set of data was carefully checked. A total of 75 ± 27 s (range 0–191 s) was excluded from the analysis because of artifacts, EEG arousals (increased mean EEG frequency > 16 Hz lasting 3–30 s and increases in chin EMG), and/or movement artifacts in respiratory signals.

Analysis of $P_{ETCO_2}$

Because of intermittent small breaths, end-tidal plateaus were not always achieved for each breath. Only breaths with a true end-tidal plateau were included in the calculation of the step changes in $P_{ETCO_2}$. Overall, 8.4 ± 0.5 (SD) % of breaths were excluded from the analysis of the $P_{ETCO_2}$.

Validation of $V_T$ Measurements

Stability of the PT signal. After calibration of the PT in the evening by means of a 1,000-ml calibration syringe, application of the same volume after ~7 h in the morning resulted in a reading of 995 ± 17 ml (range 981–1,021 ml). The differences from the initial calibration values were not significant ($P > 0.47$, paired t-test).

Detection of mouth breathing. The subject's mouth was not taped shut for safety reasons and to minimize disturbances. Mouth opening was detected by means of the PT by a drop or rise of the ratio of separately measured inspiratory to expiratory volumes, in conjunction with a decrease of the ratio of $V_{PT}$ to the $V_T$ estimates of the RIP ($V_{TRIP}$), provided that there were no movement-related changes in the RIP signal. Overall, a 10-min episode of mouth breathing in one subject was excluded from the analysis.

Validation of RIP data. Although the RIP method has been shown to give a good estimate of $V_T$ after appropriate calibration (5), there is considerable doubt over its accuracy during a whole night's sleep after only one initial calibration procedure (27). In our study, we profited by the fact that $V_T$ were simultaneously measured by means of the PT and the RIP. In addition to the initial calibration procedure, the $V_{TRIP}$ were correlated with the $V_{PT}$ for each REM sleep episode. The mean of the correlation coefficients between $V_{PT}$ and $V_{TRIP}$ was 0.97 ± 0.02 (SD). $V_T$, respiratory timing, and ventilation were calculated by using the PT signal. Component ventilation ($V_{RC}$ and $V_{Abd}$) was analyzed by using the RIP signal. For breaths to be accepted, the difference between $V_{PT}$ and $V_{TRIP}$ had to be <10%. This was the case in 3,625 of 4,455 breaths (81.4%).

Timing Between Rapid Eye Movements and Breaths

As the density and the timing of eye movements have an effect on the magnitude of changes in ventilation (9, 15), we further restricted the analysis to those breaths that occurred during a burst of eye movements, with the preceding and following breaths also associated with eye-movement activity designated as REM+. These breaths were compared with breaths called REM−, which occurred within a REM sleep period but were not associated with any eye movement. There was at least one breath interspersed between REM− breaths and preceding or following eye-movement activity. A total number of 1,994 breaths (44.8%) fulfilled these criteria and was included in the analysis.

Statistical Methods

From the tables containing the breath-by-breath information, we calculated mean values of breaths in the absence of rapid eye movements (REM−) and the means of those coinciding with rapid eye movements (REM+) for each of the three $CO_2$ steps in the eight subjects. The data were entered into an ANOVA by $CO_2$ step (0, 1, or 2) and by REM activity (0 or 1) with a repeated-measurement design by using SPSS V.6. If significant differences were shown to exist between the group means, comparisons were made by using paired t-tests. A level of significance was corrected by using the Bonferroni method. A P value < 0.05 was considered significant. Data are expressed as means ± SE unless otherwise stated.

RESULTS

REM sleep was recorded in all eight subjects. Eighteen REM sleep periods (mean duration 19.6 ± 2.9 min, total duration 353 min) were included in the analysis. From a total of 1,994 breaths that met the inclusion criteria, 1,397 (70.1%) occurred in the absence of rapid eye movements (REM−) and 597 (29.9%) in the presence of rapid eye movements (REM+). With respect to $CO_2$ steps, 494 breaths occurred during normocapnia, 828 during the first step of hypercapnia, and 672 during the second. The baseline $P_{ETCO_2}$ of 40.9 ± 1.0 Torr was increased to 44.1 ± 0.8 Torr in the first step of hypercapnia and to 46.8 ± 0.7 Torr in the second step by adding $CO_2$ to the inspired air. Figure 1 shows an
original recording including a step change in the inspiratory CO₂ partial pressure.

Differences between mean values obtained from breaths in the absence and presence of rapid eye movements are shown in Fig. 2. During normocapnia, breaths coinciding with rapid eye movements had a smaller VT (405 ± 29 ml), compared with breaths in the absence of rapid eye movements (495 ± 26 ml, P < 0.05), due to an inhibition of rib cage movements (120 ± 19 vs. 195 ± 23 ml, P < 0.05). The TT was shorter (3.6 ± 0.2 vs. 4.3 ± 0.1 s, P < 0.05) because of a shorter TE, i.e., respiratory rate increased from 14.0 to 16.8 breaths/min during rapid eye movements. These effects, however, were offsetting. Minute ventilation [6.86 ± 0.48 l/min, not significant (NS)] remained unchanged during rapid eye movements.

Increasing hypercapnia during REM sleep led to an overall increase of VT and rib cage and abdominal breathing movements and a shortening of TT, Ti, and TE, resulting in an increased minute ventilation (Fig. 2). Under these hypercapnic conditions, breaths in the presence of rapid eye movements, compared with those without rapid eye movements, showed differences in the same directions as observed during normocapnia but were even more pronounced (see Fig. 3 for details of proportional changes during rapid eye movements compared with in the absence of rapid eye movements). In addition, abdominal breathing movements were now reduced during rapid eye movements by 24 ± 15 ml (NS) in the first CO₂ step and by 79 ± 23 ml (P < 0.05) in the second. These differences also affected ventilation, as minute ventilation was also smaller during rapid eye movements (−0.61 ± 0.44 l/min, NS, first step of hypercapnia; and −1.84 ± 0.54 l/min, P < 0.05, second step) in hypercapnia (Figs. 2 and 3).

Hypercapnia had a significant modulating influence on REM-associated changes of respiratory patterns. The ANOVA revealed a significant influence of the end-expiratory CO₂ level on differences in abdominal contribution and minute ventilation, which were more and more reduced in the presence of rapid eye movements, compared with in the absence of rapid eye movements, with higher levels of hypercapnia (P < 0.05, Fig. 3). Changes in all other measures, Tr, Ti, and TE, duty cycle, and rib cage contribution, were not significantly different among the three CO₂ steps.

Subjects with only minor differences in mean respiratory measures between breaths, with and without rapid eye movements, so-called low responders (16), were hard to find. At least during increased respiratory drive, relative differences in respiratory timing or VT exceeded reductions of 10% from baseline values. Figure 4 demonstrates that there was a strong correlation between the reduction in VT and the increase in respiratory frequency, with a tendency to stabilize minute ventilation by offsetting effects. Within-subject differences and the tendency to stronger effects during hypercapnia (see Fig. 3) can be seen.

**DISCUSSION**

The present study demonstrates that phasic REM activity coincides with typical changes of respiratory measures even under the conditions of increased respiratory drive by hypercapnia. Our results extend previous findings of interactions during normocapnia (2, 4, 15, 16) and hypercapnia (25), presenting quantitative data on REM-associated changes in respiratory timing, VT, compartmental volume, and ventilation in relation to increased chemical drive of respiration. One observation of interest is that respiratory changes during normocapnia are offsetting with respect to ventilation, whereas, with increasing hypercapnia, abdominal breathing movements are more and more inhibited during phasic REM events. This leads to decrements in ventilation, which might be responsible for the observation of attenuated hypercapnic ventilatory responses, as described to occur during phasic REM sleep in dogs (25), or eye-movement-dense REM sleep in humans.
The results with respect to normocapnia are similar to those reported previously (2, 9, 15). We found eye-movement-related shortening of \( T_T \) and \( T_E \) but only small effects on \( T_I \). Results on changes in \( V_T \) and compartmental volume during normocapnia (open circles, PET\( \text{CO}_2 \) = 40.9 Torr), mild hypercapnia (shaded circles, PET\( \text{CO}_2 \) = 44.1 Torr), and stronger hypercapnia (solid circles, PET\( \text{CO}_2 \) = 46.8 Torr). Values are means ± SE of 8 subjects. Differences between breaths with and without eye-movement activity: \( *P < 0.05, **P < 0.001 \) (ANOVA with repeated-measurement design); \( +P < 0.05, ++P < 0.01 \) (post hoc \( t \)-tests with Bonferroni correction). n.s., Not significant.

**Normocapnia**

Fig. 2. Summary of differences between breaths in absence (REM \(-\)) and presence (REM \(+\)) of rapid eye movements of tidal volume (\( V_T \)), total breath time (\( T_T \)), minute ventilation (\( V_E \)), \( V_{T,rc} \), \( V_{Tabd} \), and inspiratory time (\( T_I \)) during normocapnia (open circles, PET\( \text{CO}_2 \) = 40.9 Torr), mild hypercapnia (shaded circles, PET\( \text{CO}_2 \) = 44.1 Torr), and stronger hypercapnia (solid circles, PET\( \text{CO}_2 \) = 46.8 Torr). Values are means ± SE of 8 subjects. Differences between breaths with and without eye-movement activity: \( *P < 0.05, **P < 0.001 \) (ANOVA with repeated-measurement design); \( +P < 0.05, ++P < 0.01 \) (post hoc \( t \)-tests with Bonferroni correction). n.s., Not significant.

This finding confirms earlier studies performed in infants (10), in which falls in \( V_T \) and \( T_T \) were associated with eye-movement activity. Although in that study, instantaneous ventilation did not change, other studies indicated that ventilation was reduced (9, 15). Gould et al. (9) found an 8.7% reduction in minute ventilation in intervals with eye movements compared with those without eye movements. These differences might be due to the mode of analysis used on the background of the large variation of ventilation during REM sleep. Whereas Gould et al. (9) used 20-s epochs with and without eye movements, our results were obtained from pooled data of single breaths with and
without eye movements distributed over the REM sleep periods.

Hypercapnia

In several studies, phasic irregularities in REM sleep breathing were observed during experimentally increased levels of afferent input to medullary respiratory neurons by hypercapnia. Irregular breathing persisted in the cat (19) and the dog (21, 25) despite progressive hypercapnia. We now can show that, during hypercapnia in men, REM activity affected respiratory timing and rib cage compartmental breathing in a similar manner compared with normocapnia. Relative changes in $T_T$ and $V_{Trc}$ of breaths with and without eye movements were not significantly different for the three CO$_2$ levels. Abdominal movements, however, which remained unchanged during normocapnia, showed increasing inhibition with increased levels of respiratory drive. In contrast to normocapnic conditions, ventilation significantly decreased during eye-movement activity in hypercapnia.

**High and Low Responders**

On the basis of the findings of large between-subject differences in respiratory changes during REM sleep, Neilly et al. (16) defined high and low responders to phasic REM events. With respect to these individual characteristics, we observed differential changes in decrements in $V_T$ and compartmental volume and increments in respiratory frequency in association with the level of the PCO$_2$. Some individuals had only minor changes, e.g., during normocapnia, but stronger effects during hypercapnia.

**Mechanisms**

Our results could be explained by rapidly increased upper or lower airway resistance coinciding with rapid eye movements during REM sleep. Our data, however, give no information about the behavior of the airway resistance. By means of genioglossus and alae nasi EMGs, Wiegand et al. (28) showed reduced upper airway dilator muscle activation during phasic REM events. Supraglottic pharyngeal resistance, however, did not change significantly during rapid eye movements in normocapnia. On the other hand, there is experimental evidence for centrally mediated effects on respiration during phasic REM activity. Generally, human intercostal EMG activity declines during REM sleep, compared with NREM sleep and wakefulness, whereas diaphragmatic EMG increases (26). Several other studies (15, 16) on the association of phasic REM events with changes in ventilation under normocapnic conditions showed additional inhibition of rib cage motion, whereas very little of the variation in abdominal motion was associated with phasic eye movements. These differences in effects on compartmental ventilation have been related to differences in the response to lesions of brain stem regions that mediate motor atonia during REM sleep. Hendricks et al. (12) showed that elimination of an area in the dorsal pontine tegmentum abolished REM sleep atonia and disinhibited diaphragmatic EMG during inspiratory bursts by increasing the average rate of rise of diaphragmatic activity. Stimulation of this area inhibited phrenic nerve activity only slightly but inhibited intercostal nerves more (13). Thus intrinsic neuronal REM sleep mechanisms seem to trigger both the phasic REM events and the effects on respiration, as suggested by the strong association of pontogeniculaooccipital waves with changes in the activity of medullary respiratory neurons (17).

**Fig. 3.** Relative % changes of respiratory measures during REM activity compared with breaths in absence of rapid eye movements. There was a 12–17% decrease in $T_T$ with rapid eye movements. $V_T$ decreased by 18–26%, $V_{Trc}$ decreased by 34–39%. During normocapnia, $V_{Tabd}$ and $V_E$ remained unchanged. Hypercapnia increased REM-associated proportional changes of $V_{Tabd}$ and $V_E$ (*P < 0.05, ANOVA). There was no significant effect of CO$_2$ level on proportional changes in $T_T$, $V_T$, and $V_{Trc}$.

**Fig. 4.** Decreases in $V_T$ and $T_T$ of breaths in presence compared with in absence of rapid eye movements tend to offset each other. Relative changes of $V_T$ and $T_T$; lines connect data of individual subjects; arrows at end of lines indicate "hypercapnic direction"; dotted line, linear regression with corresponding correlation coefficient $r$. 

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- **Fig. 3** shows the relative % changes of respiratory measures during REM activity compared with breaths in absence of rapid eye movements. Differences in $T_T$, $V_T$, $V_{Trc}$, $V_{Tabd}$, and $V_E$ are presented.

- **Fig. 4** illustrates the decreases in $V_T$ and $T_T$ of breaths in presence compared with in absence of rapid eye movements, showing the tendency to offset each other.

- The data in **Fig. 3** reveal a 12–17% decrease in $T_T$ with rapid eye movements. $V_T$ reduces by 18–26%, $V_{Trc}$ by 34–39%. During normocapnia, $V_{Tabd}$ and $V_E$ remain unchanged. Hypercapnia increases REM-associated proportional changes of $V_{Tabd}$ and $V_E$ (*P < 0.05, ANOVA). There is no significant effect of CO$_2$ level on proportional changes in $T_T$, $V_T$, and $V_{Trc}$.

- The changes in ventricular volume and compartmental volume are noted, illustrating the decrease in $V_T$ and $T_T$ during REM activity.

- The **Fig. 4** demonstrates decreases in $V_T$ and $T_T$ of breaths in presence compared with in absence of rapid eye movements, with a correlation coefficient $r = 0.66$, $P < 0.001$.
eral effects, however, do not explain the CO₂-dependent differential effect of eye-movement activity on abdominal breathing and ventilation. Recent studies by Smith et al. (23) on the neural-mechanical coupling of breathing in REM sleep in chronically instrumented dogs may offer an explanation of mechanisms involved. They analyzed the diaphragmatic EMG during airway occlusion in phasic REM sleep, compared with NREM sleep, and found a reduced and more variable diaphragmatic EMG activity during occluded breaths in phasic REM sleep, attributed to a reduced respiratory neural output. Brief interruptions, so-called fractionations (18) of the diaphragm EMG ramp, accounted for at least some of the blunted response to airway occlusion and the related asphyxic blood-gas changes. These fractionations were present in 41% of occluded breaths. In addition, Hendricks and Kline (11) showed considerable regional heterogeneity of fractionations in the costal diaphragm of cats. Thus many local interruptions may occur in different regions of the diaphragm at the same time within one breath, which would impair the recruitment of the diaphragm during increased respiratory drive. Several other mechanisms might also be involved, such as direct interactions of phasic REM events at the level of respiratory neurons, leading to premature switch off of inspiration (23) or interference of phasic events with the central chemosensitive mechanism, either directly or at the level of integration of chemosensory inputs to the neural respiratory network. Even short-term changes in local cerebral blood flow in the central chemosensitive area could account for reductions in ventilation (20). Conclusions about the roles of these mechanisms in the genesis of REM-related changes in diaphragmatic activity are, however, beyond the scope of this paper.

Implications

The CO₂-dependent differential effect on abdominal breathing might be responsible for the findings of attenuated hypercapnic ventilatory responses during REM sleep (7), despite similar normocapnic ventilation during REM and non-REM sleep (26). The blunted ventilatory response to hypercapnia during eye-movement-dense REM sleep in humans could also account for the prolongation of obstructive apneas observed during REM sleep (24), as during the buildup of asphyxia the ventilatory effort is reduced compared with during NREM sleep. This in turn leads to reduced sensory input from the chest wall, which plays an important role in the occurrence of arousal from sleep and termination of apneas (8).

In conclusion, the typical changes in respiration associated with rapid eye movements, e.g., rapid, shallow breathing with predominant effects on rib cage excursions and Te, are by no means attenuated during increased respiratory drive during hypercapnia but are even more pronounced. In addition, abdominal excursions are inhibited, which leads to a decrease in minute ventilation during rapid eye movements with increasing levels of PETCO₂.

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