Laryngeal Abductor Activity during Sleep

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In normal adult cats, upper airway resistance increases during both nonrapid eye movement (NREM) and rapid eye movement (REM) sleep. There is an increase in the mean resistance level during NREM even though the decreases in resistance which occur during inspiration are larger than in wakefulness. In REM, expiratory resistance is high, and the decreases in resistance during inspiration are small. These observations suggest that the diameter of the upper airways decreases during sleep, and that the dilation of the upper airways during inspiration is reduced in REM sleep. The present study shows that reduced activity in the laryngeal abductors, the posterior cricoarytenoid muscles, can account for the increase in upper airway resistance during sleep.

Materials and Methods

Electrodes were implanted in 6 adult cats for recording sleep parameters. Skull-holts for head-restraint, electrodes and their connector were cemented to the skull. Breathing was monitored through a chronic tracheal fistula using a pneumotachograph. The larynx was exposed ventrally and two small stainless steel electrodes were inserted bilaterally into the posterior cricoarytenoid (PCA) muscles using a transglottic approach. The electrode wires were led under the skin to the head-cap. The animals were sleep-deprived for 12-14 hours prior to each recording session. Each animal was recorded for a minimum of 2 days during each of which at least 4 sleep-wakefulness cycles were observed. The cats were atraumatically restrained for recordings. Sleep was defined according to conventional polygraphic criteria. PCA activity was integrated and expressed as activity per unit time for both inspiration and expiration.

Results

The PCA muscles were most active during inspiration (Fig 1, Table 1). Integrated expiratory PCA activity during wakefulness averaged 60% of the inspiratory activity. The expiratory or tonic PCA activity decreased in sleep. In NREM it was 30% of the wakefulness inspiratory activity, and, in REM, expiratory activity averaged 18% of wakefulness inspiratory levels.

Phasic or inspiratory PCA activity also decreased progressively from W to NREM and NREM to REM although the amount of the reduction was less than for the tonic activity. The phasic inspiratory bursts in NREM were about 88% of those in wakefulness. In REM, inspiratory activity was 73% of wakefulness levels.

Discussion

The changes in tonic and phasic PCA activity can be related to upper airway resistance during sleep and wakefulness. In wakefulness, expiratory upper airway resistance is almost as low as the inspiratory resistance. The substantial tonic PCA activity and the relatively small difference between tonic and phasic activity in wakefulness are consistent with a glottis which is nearly as dilated in expiration as in inspiration. In NREM sleep, expiratory resistance increases but inspiratory resistance is low. The decrease in tonic PCA activity can explain the increase in expiratory resistance, and the large drops in resistance during inspiration may derive from the large difference in tonic and phasic PCA activity which characterizes this state of sleep. In REM sleep, expiratory resistance is highest, and tonic PCA activity is least; the resistance drops during inspiration are small, and phasic PCA activity is minimal.

Table 1—Means and Standard Deviations of PCA Activity from 9 Muscle Groups in 6 Cats

<table>
<thead>
<tr>
<th></th>
<th>Awake</th>
<th>NREM</th>
<th>REM</th>
</tr>
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<tbody>
<tr>
<td>Inspiratory</td>
<td>.505 ± .194</td>
<td>.444 ± .190</td>
<td>.367 ± .214</td>
</tr>
<tr>
<td>N = 170</td>
<td>N = 168</td>
<td>N = 188</td>
<td></td>
</tr>
<tr>
<td>Expiratory</td>
<td>.304 ± .140</td>
<td>.151 ± .076</td>
<td>.089 ± .102</td>
</tr>
<tr>
<td>N = 170</td>
<td>N = 168</td>
<td>N = 188</td>
<td></td>
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PCA activity is expressed in arbitrary units as activity per unit time. The differences between wakefulness, NREM and REM are all significant (P < .05)

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The present results indicate a narrowing of the glottic aperture and a progressive weakening of inspiratory abduction of the vocal folds during sleep. This reduction in respiratory drive to the abductors of the larynx may be a generalized phenomenon for the muscles of the upper airways. Reductions in genioglossal activity during sleep have been reported and obstructive sleep apnea is recognized as a major sleep disorder.

References

Abnormal Ventilatory Response to CO2 during Quiet Sleep in Aborted SIDS

Daniel C. Shannon, M.D., and Dorothy Kelly, M.D.

We have measured expired tidal volume (Vt), frequency (f), minute ventilation (Ve) and the ventilatory response to inhaled CO2 (ΔVe/ΔPaCO2) using the nasal pneumotachograph technique in 11 infants with aborted SIDS (AS), (gestational age > 37 wks) who required 2 or more resuscitations for apneic, cyanotic, atonic spells and in 12 age-matched controls during quiet sleep (no eye movements, regular respirations, high voltage slow EEG and startles). Ve (205.3 ± 42.5 ml/kg/min BTPS) and Vt (7.19 ± 2.41 ml/kg BTPS) in AS infants were comparable to control infants. PaCO2 in AS (38.9 ± 3.5 mm Hg) was higher than in control (35.1 ± 1.9 mm Hg) infants (P = < .01). ΔVe/ΔPaCO2 in AS (22.2 ± 8.9) was significantly lower than in control (63.1 ± 19.1) infants (P = < .001), due mainly to a lower ΔVt/ΔPaCO2 in AS (0.74 ± 0.48) compared to control (1.98 ± 0.68) infants (P = < .001). PaCO2 at the intercept (Ve = 300 ml/kg/min) was shifted down to the right in AS (44.3 ± 7.7) compared to control (36.5 ± 2.8) infants (P = < .01). Thus, some infants with AS have sleeping alveolar hypoventilation and impaired chemoreceptor regulation of Vt. This could explain the propensity for sleep apnea and the need for resuscitation in infants with AS.

Disordered Breathing and Oxygen Desaturation during Sleep in Patients with Chronic Obstructive Pulmonary Disease


Patients with chronic obstructive pulmonary disease (COPD) often experience episodic oxygen desaturation during sleep,1-4 although why this occurs is not known. Sleep-induced episodic oxygen desaturation is also seen in patients suffering from the sleep apnea syndrome.5 Such patients may experience hundreds of episodes of apnea during a single night’s sleep, many of which may lead to desaturation. Sleep-disordered breathing, including periodic breathing and apnea, is also seen in some normal subjects, but to a limited extent.6 With these facts in mind, we recently demonstrated a relationship between sleep-disordered breathing and oxygen desaturation in patients with COPD who were monitored during daytime naps.7 In the present study, we have extended our observations to include prolonged nocturnal sleep.

Methods

Seven patients with COPD were selected from the general medical wards and outpatient clinics of the Gainesville Veterans Administration Hospital on the basis of having abnormal pulmonary function tests and a clinical history and physical examination indicative of COPD. All patients denied having difficulty sleeping or excessive daytime somnolence. With one exception, all were monitored during sleep in a sleep laboratory using standard polygraphic techniques. Chest wall movement was sensed by impedance pneumography, nasal and oral airflow by thermistor probes in the nose and mouth. Oxygen saturation was measured by ear oximetry. These variables were recorded simultaneously on a multichannel recorder. Electroencephalographic (EEG) and electro-oculographic (EOG) tracings were obtained simultaneously, but were recorded separately. Analysis of EEG and EOG tracings was performed by experienced sleep laboratory technicians.

For the purpose of our study, oxygen desaturation was defined as a reduction in oxygen saturation of greater than 4% from baseline; apnea, as cessation of airflow at the nose and mouth lasting at least 10 seconds; and periodic breathing, as alternating hyperpnea and hypopnea with or without apnea.

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