Sleep Apnea as a Feature of Bulbar Stroke

J.J.M. Askenasy, MD, PhD, and I. Goldhammer, MD

Medullary disorders can be associated with a sleep apnea syndrome. The present patient developed a sleep apnea syndrome with approximately 25 episodes of apnea or hypopnea during each hour of sleep following a lateral medullary infarction. The presence of predisposing factors and involvement of respiratory centers in the area of the medullary lesion may determine the appearance of sleep apnea with brainstem infarction. Investigation of breathing during sleep may be helpful in such cases. (Stroke 1988;19:637–639)

The respiratory neurons are located in the reticular formation of the medulla oblongata. They are closely related to motor activity centers of the upper respiratory tract involving the tongue and pharynx. The larynx ventilation undergoes changes during rapid eye movement (REM) and non-REM sleep as a consequence of changes in the reticular system of the medulla oblongata.1 The anatomic proximity of the centers controlling breathing, sleep, and upper respiratory tract motility suggests a close physiologic interrelationship. Sleep in general and REM sleep in particular depress upper respiratory tract motor function, favoring and accentuating respiratory disturbances.

Sleep apnea has been reported in medullary infarction, high cervical cordotomy, syringobulbia, bulbar poliomyelitis, olivopontocebellar degeneration, near-drowning, neoplasms, hindbrain abnormalities, multiple sclerosis, and trauma (Table 1).2-9 Of the medulla oblongata disorders, vascular disturbance is the most frequent cause of respiratory sleep distress.3,5,16

Case Report

A 49-year-old man with no history of hypertension or obesity suffered a left Wallenberg’s syndrome. The clinical signs consisted of hoarse voice, intractable hiccups, left Horner’s syndrome, mild skew deviation, left soft palate palsy, left vocal cord paralysis, ataxia (left more than right), right dissociated hemianalgesia with no left face hemianalgesia, right Babinski’s sign, and Romberg’s sign. Following the stroke the patient had severe difficulty in swallowing. Routine laboratory examinations were normal. Computed tomography (CT scan) with and without contrast done at the time of admission showed no abnormality. Fine cuts through the posterior fossa could not be obtained because of temporary technical limitations.

During the next 10 days, as his swallowing disturbance improved, very noisy snoring appeared, with abrupt pauses accounting for apneic spells. An ear-nose-throat examination revealed a long uvula and a septum deviation occluding his left nostril. Based on anamnestic data collected from his wife, there was no history of loud snoring or respiratory arrest before the brainstem stroke. Sleep was monitored for two nights following an adaptation night, using a routine polygraphy technique. The routine polygraphy consisted of electroencephalography, electro-oculography, electromyography, breathing monitoring, and closed-circuit television. Breathing monitoring consisted of nasal-oral airflow, rib-abdominal cages, and ear oxymetry. The mean data for two nights showed 150 episodes of apnea and hypopnea per night’s sleep. Hypopnea was defined as apneic episodes lasting 5–10 seconds. Apnea was defined as apneic episodes lasting >10 seconds. The total number of apneic and hypopneic episodes per hour of sleep representing the apneahypopnea index was 25.0. The diagnosis of a sleep apnea syndrome is made when there are >30 breath arrests lasting >10 seconds during a night’s sleep. Sleep apnea has been categorized as central, obstructive, or mixed. Central apnea is present when the respiratory center fails to contract the diaphragm. Obstructive apnea is characterized by the blockage of air flow in the upper airway despite the presence of diaphragm contraction. Of the total 150 episodes of apnea and hypopnea, 99 were obstructive or mixed and 51 were of the central type. Fifty apneic episodes were associated with <85% O2 saturation and 10 with 80% O2 saturation, that is, with a PaO2 of <50 mm Hg. At this level of hypoxia, pulmonary vasoconstriction can occur, which may predispose to pulmonary and systemic hypertension and lead to worsening hypoxia. No cardiac changes were found.

The longer apneic episodes occurred during REM sleep, while the patient was sleeping on his back and rarely on his right side, but never while sleeping on his left side. The apneic episodes that did not affect O2 desaturation lightened the level of his sleep. The lightening was marked by very frequent switches from sleep stage 2 and REM stage to sleep stage 1 when compared with a normal routine polysonogram. Morning tiredness and a need to nap after lunch

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TABLE 1. Sleep Apnea in Medulla Oblongata Disorders; Review of Literature 1951–1986

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Disorders</th>
<th>References</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Bulbar stroke</td>
<td>2, 3, 4, 5</td>
</tr>
<tr>
<td></td>
<td>High cervical cordotomy</td>
<td>6, 7, 8, 9</td>
</tr>
<tr>
<td>2</td>
<td>Syringobulbia</td>
<td>10, 11, 12</td>
</tr>
<tr>
<td>3</td>
<td>Bulbar poliomyelitis</td>
<td>13, 14</td>
</tr>
<tr>
<td>4</td>
<td>Olivopontocerebellar degeneration</td>
<td>12, 15</td>
</tr>
<tr>
<td>5</td>
<td>Near-drowning (bulbar ischemia)</td>
<td>16</td>
</tr>
<tr>
<td>6</td>
<td>Bulbar neoplasms</td>
<td>17</td>
</tr>
<tr>
<td>7</td>
<td>Hindbrain abnormalities</td>
<td>18</td>
</tr>
<tr>
<td>8</td>
<td>Multiple sclerosis</td>
<td>19</td>
</tr>
<tr>
<td>9</td>
<td>Medullary trauma</td>
<td>2</td>
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</tbody>
</table>

Decreasing order of frequency according to number of published observations.

TABLE 1. Sleep Apnea in Medulla Oblongata Disorders; Review of Literature 1951–1986

In mammals, three aggregations of rhythmic respiratory neurons in the medulla oblongata were described: the dorsal and ventral respiratory nuclei in the lower medulla and the nucleus parabrachialis medialis in the upper medulla (Figure 1). To generate a normal respiratory pattern the three centers require vestibulocerebellar regulation of the tonus of supraglottic, genioglossal, pharyngolaryngeal, diaphragmatic, and intercostal muscles. Disorders of the vestibulocerebellar system, such as olivopontocerebellar degeneration, may cause sleep apnea due to impaired function of the respiratory muscles.12,15

The proximity of the hypnogenic and somatic upper respiratory airway centers may explain the association of lateral medullary infarction with sleep apnea. The type and severity of the apnea-hypopnea index may depend on the number of injured neurons and axons and on the compensatory function of the contralateral undamaged respiratory centers. This compensatory function may vary between individuals and may play an important role in sleep apnea. Unilateral lesions may provoke sleep apnea in individuals whose compensatory capacity is weak or in whom congenital abnormal control of respiration exists.20 Bilateral damage of all three respiratory nuclei may result in failure of automatic respiration, termed Ondine’s curse.29

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In conclusion, it is recommended that the possibility of the association of medullary disorders with sleep apnea in patients with predisposing factors and a certain topography of the lesion be taken into consideration. In patients with snoring and respiratory arrests following bulbar stroke, supervising or monitoring breathing during sleep is indicated.

References


**Key Words**: sleep apnea • brainstem • cerebrovascular disorders
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