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REVIEW ARTICLE

Sleep apnea and cervical spine pathology

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Abstract
Purpose Sleep apnea is a multi-factorial disease with a variety of identified causes. With its close proximity to the upper airway, the cervical spine and its associated pathologies can produce sleep apnea symptoms in select populations. The aim of this article was to summarize the literature discussing how cervical spine pathologies may cause sleep apnea.
Methods A search of the PubMed database for English-language literature concerning the cervical spine and its relationship with sleep apnea was conducted. Seventeen published papers were selected and reviewed.
Results Single-lesion pathologies of the cervical spine causing sleep apnea include osteochondromas, osteophytes, and other rare pathologies. Multifocal lesions include rheumatoid arthritis of the cervical spine and endogenous cervical fusions. Furthermore, occipital–cervical misalignment pre- and post-cervical fusion surgery may predispose patients to sleep apnea.
Conclusions Pathologies of the cervical spine present significant additional etiologies for producing obstructive sleep apnea in select patient populations. Knowledge of these entities and their pathophysiologic mechanisms is informative for the clinician in diagnosing and managing sleep apnea in certain populations.

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Introduction

Sleep apnea is a chronic condition characterized by repeated anatomic obstruction or partial collapse of the upper airway during sleep. With an estimated prevalence of 9–28 % in adults, sleep apnea is often characterized by daytime impairment, poor neurocognitive performance, and excessive daytime sleepiness [1]. Important sequelae of sleep apnea include decreased cognitive function and excessive morbidity and mortality secondary to cardiovascular disorders such as hypertension, coronary artery disease, and stroke [2].

Sleep apnea may be composed of obstructive apneas, central apneas, or their combination. Obstructive apnea is caused by narrowing of the upper airway, whereas central apnea is caused by dysregulation of the respiratory center. The two categories are differentiated by respiratory pattern: obstructive sleep apnea (OSA) is characterized by increased respiratory effort without airflow, whereas central sleep apnea (CSA) is marked by the absence of both respiratory effort and airflow. OSA is the most common variant of sleep apnea, comprising 84 % of cases. CSA comprises 0.4 % of sleep apnea cases. A mixed combination of OSA and CSA comprises 15 % of cases [3]. The apnea–hypopnea index (AHI), a measure of the number of respiratory disturbances per hour of sleep, is often used as a proxy for the severity of sleep apnea. AHI values are typically categorized as: 5–15/h, mild; 15–30/h, moderate; and >30/h, severe.

In OSA, patients experience multiple episodes of airway collapse during sleep due to direct compromise of the

integrity of the upper airway. The upper airway is normally maintained by the bony and cartilaginous structures surrounding the naso- and oropharynx. Multiple factors such as obesity, craniofacial abnormalities, and upper airway tissue abnormalities may predispose patients to apneic events [4]. Craniofacial abnormalities include abnormal maxillary or short mandibular size, a wide craniofacial base, adenoid hypertrophy, or extended head posture [4]. Furthermore, anatomic studies of the pharynx and upper airway have demonstrated a greater tendency for a functionally passive and anatomically narrow pharynx to collapse more easily in patients with OSA [5].

These structures of the neck have all been demonstrated to play a role in upper airway patency. However, there has been limited research examining the role of another crucial structure involved in upper airway patency, the cervical spine. Located posterior to the pharynx, the cervical spine is composed of seven vertebrae that provide full range of motion to the neck through articulations with the occiput and between the vertebrae. Due to the cervical column's intimate association with the upper airway, an understanding of the relationship between the cervical spine and sleep apnea is important for the clinician and researcher. This article reviews the literature examining the relationship between cervical spine pathology and sleep apnea.

Methods

A complete search of the PubMed database for English-language literature concerning the cervical spine and its relationship with sleep apnea was conducted. The search terms used were cervical spine sleep and cervical column sleep for all articles published between the years 1980 and 2012. The bibliography of each article was reviewed for additional relevant articles. Each article was carefully analyzed and included in our study if discussion of cervical spine pathology causing sleep apnea was detailed.

Results

Our initial search resulted in 177 articles. Of these, 17 discussed an association between the cervical spine and sleep apnea. Topics explored in the literature included case reports of cervical spine pathology causing sleep apnea, studies of cervical column anomalies in sleep apnea, and instances of iatrogenic cervical spine fusion leading to OSA.

Abnormal morphology

In the literature, only two studies have attempted to investigate morphology of the cervical spine in patients with sleep apnea [4, 6]. In patients with an AHI score greater than 5, lateral radiographs with standardized head posture were taken of 91 [4] and 74 [6] consecutive patients. Abnormal morphology in these studies was categorized as non-iatrogenic endogenous fusions or posterior arch deficiencies. The prevalence of morphological deviations in patients with sleep apnea was 46 and 43 % in the two studies, with endogenous fusions being the vastly predominant abnormality [4, 6]. The 46 % prevalence of cervical fusion in patients with OSA described by Sonnesen [4] was significantly higher than the 14 % prevalence of cervical fusion in a control group of healthy students and staff from the researchers' academic department. In addition, a qualitative difference was observed in these groups, as fusions of the cervical vertebrae in controls always occurred between C2 and C3, while those in the OSA group were located diffusely, between C2 and C3, C3 and C4, or C4 and C5 [4]. The authors found neither gender nor age to correlate with morphology. However, they did not account for other important demonstrable factors, such as patient weight and upper airway tissue abnormalities.

The difference in prevalence and pattern of cervical abnormalities in OSA patients has not been well explained. Some authors have attempted to account for the difference by examining craniofacial features. Previous studies have demonstrated an association among the incidence of cervical fusions and a larger cranial base angle (defined as the angle between the nasion, sella turcica, and basion) and extended head posture in patients [7]. An explanation for the association may be found in early embryogenesis. As the notochord determines the development of cervical vertebrae and the basilar part of the occipital bone, it also induces the formation of the para-axial mesoderm forming the vertebral arches and the remaining aspect of the occipital bone [4]. Other authors argue that patients with OSA have a more extended head posture as a physiological adaptation to maintain airway patency [8]. However, Svanholt et al. [6] revealed that OSA patients with endogenous cervical fusions demonstrated no significant difference in cranial base angle or posture of the head from that of controls. These patients did possess statistically larger anterior face heights (128.26 vs. 122.90 mm in controls) and greater mandibular lengths (121.39 vs. 116.99 in controls), but no association with cranial base angle or head posture [6]. As craniofacial morphology and head posture are considered predisposing factors for OSA, it is unclear whether the role of craniofacial profile helps explain the association between cervical morphology abnormality and sleep apnea [9, 10].

Rheumatoid arthritis of the cervical spine

Rheumatoid arthritis (RA) is a systemic inflammatory condition that afflicts multiple organ systems, particularly

synovial joints. As the occiput-C1 and C1–C2 articulations are purely synovial, these vertebrae are the primary targets for rheumatoid involvement [11]. In addition, because the C1–C2 facets are oriented in the axial plane, there is no bony articulation to prevent subluxation [12]. Thus, the three most common deformities seen in rheumatoid cervical spines are atlantoaxial subluxation, superior migration of the odontoid, or subaxial subluxation [11].

Sleep apnea associated with RA was first described by Davies and Iber [13]. Since that time, multiple case reports have described RA patients presenting with combinations of OSA and CSA. In separate studies, the prevalence of sleep apnea in RA patients was reported to be 53 and 79 % in those with occipito-cervical lesions [14, 15]. A cross-sectional study of OSA in RA patients with occipito-cervical lesions was performed by Shoda et al. [15]. They cataloged the radiographic features of 29 RA patients by measuring the atlanto-dental interval, cervical angles (O/C1, C1/2, and C2/6), and cervical lengths (O–C2 and O–C6 distance). On imaging, small atlanto-dental intervals (4.9 vs. 9.5 mm in controls) and cervical lengths (O–C2: 24.8 vs. 32.9 mm in controls; O–C6: 87.0 vs. 104.6 mm in controls) were shown to be significantly associated with the presence of sleep apnea. The horizontal atlantoaxial subluxation typically seen in RA may explain the vertical translocation and shortening of these radiographic parameters [15].

Individual case reports documenting central apneas in RA patients hypothesize that compression of the medulla by the odontoid process may be the causative factor [16]. However, a more recent study found less than 5 % of apneas to be central in RA patients with medullary compression and sleep apnea [17]. Even in RA patients with medullary compression, obstructive apnea remains the predominant mechanism of sleep disordered breathing.

Indications for operative intervention of the cervical spine in RA patients are traditionally dependent on the presence of neurologic deficits [11]. There are no current guidelines for surgical treatment of sleep apnea in RA patients with cervical involvement. Akata et al. [17] performed a prospective study of occipito-cervical fusion in eight patients with RA of the cervical spine and sleep apnea. Six of the eight patients demonstrated postoperative improvement in their sleep apnea and an increase in the O/C2 angle exceeding at least 5° on radiographic imaging. As the O/C2 angle is an indicator of alignment of the craniovertebral junction, negative and small O/C2 angles indicate flexed alignment causing narrowing and increased collapsibility of the upper airway. Symptomatic improvement with radiographic corollary demonstrates that alignment of the craniovertebral junction may improve sleep apnea in this population [17].

Cervical osteophytes

Osteophytes are bony projections that form along joint margins, commonly noted to be a sign of degeneration in the spine. Degeneration first starts in the nucleus pulposus due to loss of water content, which leads to a decrease in intervertebral disk height [18]. The annulus fibrosus loses its flexibility, and to compensate for the maximum load-carrying capacity, apophyseal joints start to grow new bone [18]. Risk factors commonly associated with osteophyte formation include obesity, diabetes mellitus, and diffuse idiopathic skeletal hyperostosis (DISH). DISH is a degenerative or metabolic disorder causing ossification of the anterior longitudinal ligament of the cervical vertebrae, and is known to produce osteophytes [18, 19]. Other clinical symptoms of cervical osteophytes include dysphagia and dysphonia.

Osteophytes may commonly present with neurological impairment due to the close relationship of the spinal canal with its neural contents. In contrast, the size of the retropharyngeal space is quite variable [20]. Small osteophytic changes rarely cause compression of the pharynx, esophagus, or upper airway despite the close relationship of the vertebral column to pharyngeal structures [20]. Eyigor et al. [18] compiled all 10 published cases of OSA secondary to cervical osteophytes. The size of osteophytes in these cases was large enough to cause narrowing of the retropharyngeal space and exert pressure on the posterior pharynx, producing symptoms. All patients exhibited OSA without any report of CSA. AHI values ranged from 9.4 to 42 [18]. A few of the patients also had the diagnosis of DISH [19–21]. Treatment modalities included constant positive airway pressure (CPAP), medical treatment, and surgery based on preferences and needs of the patients. All patients who underwent treatment demonstrated symptomatic improvement and/or improvement in their AHI values.

Osteochondromas

Spinal osteochondromas are rare benign tumors of the spine thought to arise from lateral displacement of a portion of the epiphyseal growth cartilage [22]. Most spinal osteochondromas occur in the posterior cervical spine, causing possible myelopathy or radiculopathy. Only two reports describe the unique situation of osteochondroma involving encroachment by the anterior cervical spine on the retropharyngeal space to cause OSA [23, 24]. Although osteochondromas are often part of the multiple hereditary exostoses syndrome, the two aforementioned cases were spontaneous lesions. Wang and Chou [23] described the case of a sporadic osteochondroma arising from the anterior arch of the first cervical vertebra, causing sleep apnea

in a 16-year-old female. Yoshida et al. [24] described the appearance of an osteochondroma of the atlas causing OSA in a 61-year-old female. Tumors in both patients produced symptoms by exerting pressure on the upper airway in the pharynx. Surgical resection was performed on both tumors with follow-up demonstrating improvement of the patients' OSA symptoms.

Other pathologies

There are other single reports in the literature of unique cervical column pathologies causing sleep apnea.

Kawaguchi et al. [25] described a case of os odontoideum presenting with OSA. Os odontoideum describes the separation of the odontoid process from the body of the axis. This renders the transverse atlantal ligament ineffective at restraining atlantoaxial motion. The patient presented with posterior subluxation of C1 in neutral and extended positions, which produced narrowing of the airway, causing the patient's symptoms. Fusion of the cervical vertebrae corrected the narrowing and left the patient symptom-free for 3 years of follow-up [25].

A case presented by Heike et al. [26] reported mixed OSA and CSA in a patient with 22q11.2 deletion syndrome. Apneas were explained by craniofacial features, such as a small mandible in conjunction with an elevated tongue. Central apneic events, though, were attributed to spinal cord compression related to significant occiput-C1 instability and C1–C2 instability. The patient underwent subsequent surgeries, first a mandibular distraction that corrected a large number of obstructive apneas, followed by occiput-C3 posterior fusion and segmental fixation, leading to a significant decrease in central apneic events [26].

Surgical cervical spine fusions

Cervical spine fusion surgery is often indicated as treatment for injuries of the cervical spine or spinal cord, and can be approached anteriorly, posteriorly, or both. Guilleminault et al. [27] published the only study to investigate an association between anterior cervical fusion surgery and postoperative sleep apnea. Twelve retrospective cases and four prospective cases of anterior cervical spinal fusion for cervical discopathy were included. Retrospective patients were identified postoperatively to have developed sleep apnea symptoms following anterior cervical fusion surgery. Prospective patients were objectively monitored for symptoms of sleep apnea before and after surgery. Patients in the prospective group did not have sleep apnea, as defined by an AHI score greater than 5, prior to surgery. After surgery, these four patients reported OSA symptoms via interview and questionnaire 6–8 months after surgery. In the prospective group, pre-surgery AHI ranged from 2 to 2.6, and post-surgery AHI ranged from 11 to 36. Cephalometric X-rays demonstrated that placement of cervical fusion plates along the anterior border of the cervical vertebrae reduced the size of the upper airway. However, after administration of nasal CPAP, all patients demonstrated reduction in the frequency of apneic events on repeat sleep study [27].

Discussion

Biomechanical forces that may lead to collapse of the upper airway and subsequent sleep apnea have been divided into intraluminal and extraluminal forces. Extraluminal forces include the cervical column as well as the varying muscle and adipose tissue that support the pharyngeal wall. Although some of the pharyngeal musculature is attached to the vertebral column, the cervical spine provides mostly passive support [28]. Biomechanical studies have accounted for other bony structures such as the mandibular retrognathism and a low-lying hyoid bone as influencing neck position and consequent upper airway compromise during sleep. The relatively minor role of the cervical spine in maintaining upper airway patency may help explain the dearth of biomechanical and clinical studies investigating cervical spine pathology and sleep apnea.

Cervical spine lesions may be categorized into singular lesions or multifocal lesions. Solitary projections from the cervical spine causing sleep apnea include osteochondromas, osteophytes, and other rare pathologies. Isolated cervical spine pathologies listed in the literature are included in Table 1. As reported in a handful of case reports identified in the literature, these lesions have caused OSA by exerting pressure on the upper airway in the pharyngeal space. Often these patients additionally present with other symptoms of retropharyngeal compression, including dysphagia. Although these projections are identified as solitary in many case reports, they often can present as a component of a syndrome. Osteophytes may be a component of DISH, and osteochondromas are often a component of multiple hereditary exostoses [18, 22]. In resecting osteochondromas, complete removal of the cartilaginous cap is important to prevent tumor recurrence [24]. The reported clinical improvement after surgical correction suggests that the cervical lesion was the cause for sleep apnea, not other risk factors such as weight or soft tissue abnormality.

Pathology of the cervical spine in patients with RA commonly affects the occiput-C1 and C1–C2 synovial articulations. Involvement of these joints may cause subluxation of the vertebrae and vertical translation, leading to reduced neck width and a decreased angle of the craniovertebral junction. Reduced neck width and decreased

Table 1 Isolated cervical spine pathologies

Author	Pathology	Severity (AHI score)	Level	Treatment
Hughes et al. [21]	Osteophyte	40	C2, C7	CPAP
Fuerderer et al. [20]	Osteophyte	NA	C2–C3, C7–T1	Surgery
Naik et al. [33]	Osteophyte	42	C2, C6	CPAP, surgery
Yoshida et al. [24]	Osteochondroma	47	C1	Surgery
Heike et al. [26]	Cervical instability	60	Occiput-C2	Surgery
Wang et al. [23]	Osteochondroma	NA	C1	Surgery
Ando et al. [19]	Osteophyte	9	C4–C6	NA
Kawaguchi et al. [25]	Os odontoideum	22.9	C1	Surgery
Eyigor et al. [18]	Osteophyte	62	C2, C6–C7	CPAP

AHI apnea–hypopnea index, *CPAP* constant positive airway pressure, *NA* not available

craniovertebral angles, such as the O/C2 angle, may crowd the retropharyngeal space and increase the propensity for a horizontal bending force on the upper airway, especially during neck flexion, leading to apneic events [15]. This mechanism may help account for the larger prevalence of sleep apnea in RA patients when compared to the general population [14]. However, since RA is a systemic disease with multiple sites of involvement, it is important to account for other sites outside the cervical spine, such as the temporomandibular joint. An association between temporomandibular joint disorders and OSA has been documented in the literature [29]. Thus, studies promoting a causal association between the rheumatoid cervical spine and sleep apnea must account and control for temporomandibular joint destruction and pathologies near the upper airway seen in RA.

A high prevalence of endogenous cervical fusions has been demonstrated in OSA patients [4]. Detection of an abnormality with such a high prevalence may indicate cervical fusion to be a phenotypical variant of OSA; however, the mechanism of apnea in these cases is unclear [4]. Posited explanations include poor head posture and associated craniofacial morphology, but there is little evidence to support such claims [9, 10]. Other anatomical features, such as the O/C2 angle or cervical column width, may be confounders or intermediary mechanisms for this phenomenon, as was demonstrated in our review of rheumatoid arthritic lesions of the cervical spine. Unfortunately, the reviewed studies did not record these features. Exploring such imaging and biomechanical studies of this population subset may imply diagnostic and management nuances for this possibly phenotypic variant.

Anterior cervical fusion surgery, a treatment modality for a variety of spinal pathologies, has also been associated with sleep apnea [27]. Another well-known complication of anterior cervical fusion surgery is dysphagia. The incidence of dysphagia after cervical fusion has been reported to be 50 % early after surgery, but only 5 % of patients continue to endorse moderate to severe dysphagia 6 months after surgery [30]. Postulated mechanisms include injury to the superior laryngeal nerve, direct ischemic injury to the esophagus related to retraction, and narrowing due to anterior plating. Soft tissue swelling, which had previously been reported as a possible explanation, has recently shown to have no significant difference in incidence among patients who developed dysphagia and those who did not [31]. Given the co-existence of dysphagia in many of the other cervical spine etiologies of sleep apnea described above, these mechanisms for post-surgical dysphagia may also account for sleep apnea.

In the anterior approach, fusion plate placement along the anterior border of the cervical vertebrae anteriorly displaces the posterior pharyngeal wall, leading to narrowing of the lumen of the upper airway [27]. The sole paper describing this phenomenon was based on a small cohort of patients. No other study has further investigated this association; as such, the evidence is poor. Furthermore, resolution of the dysphagia 6 months after surgery suggests that sleep apnea may also be a transient complication that naturally resolves among this patient population. A larger study of a patient population undergoing anterior cervical fusions with preoperative and postoperative sleep studies along with radiographic imaging is needed to confirm this finding. As all patients in this study demonstrated improvement when placed on nasal CPAP, effective treatment exists for patients who underwent anterior cervical fusion surgery and experienced sleep apnea symptoms. Therefore, surgeons performing anterior cervical fusion surgeries should consider monitoring their patients for postoperative symptoms of OSA, which are amenable to CPAP therapy.

There have been no investigations into an association between posterior cervical fusion surgery and sleep apnea. However, a study by Miyata et al. [32] demonstrated an association between negative changes in the O/C2 angle after posterior cervical fusion surgery in RA patients with symptoms of dyspnea, dysphagia, or both. In a separate study reviewed above, improvement in the O/C2 angle after occipito-cervical fusion surgery in RA patients was associated with symptomatic improvement in patients with

concomitant sleep apnea. Given a shared mechanism of upper airway stenosis, a negative or decreased O/C2 angle may also help predict sleep apnea in RA patients undergoing posterior cervical spine surgery. Thus, radiographic imaging of a decreased O/C2 angle after posterior cervical fusion surgery may be a helpful predictor of sleep apnea as well [17].

Conclusions

Sleep apnea is a multi-factorial disease with many identified risk factors and mechanisms. The cervical spine is one structure that may contribute to this disease. Specific cervical column pathologies, such as osteochondromas, osteophytes, and rheumatoid arthritic lesions that cause sleep apnea, have been identified in case reports. In prospective studies, an increased prevalence of endogenous cervical spine fusions in OSA patients has been reported. In addition, occipital–cervical alignment may be a factor in sleep apnea. It is important for the clinician to be aware of the role of the cervical spine in producing symptoms of OSA in certain patients. Further research is needed to elucidate the association between cervical spine anomalies and sleep apnea.

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