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ORIGINAL RESEARCH

Swallowing Function During Sleep in Patients with Head and Neck Cancer: A Polysomnographic Comparison with Matched Controls

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Purpose: We hypothesized that patients with head and neck cancer (HNC) have impaired pharynglaryngeal function during sleep compared with individuals without HNC, making them more susceptible to aspiration. We assumed polysomnography (PSG) would reveal decreased swallowing frequency, prolonged swallowing duration, reduced submental electromyographic (EMG) amplitude during swallowing, and abnormal timing of swallowing relative to respiratory phases.

Patients and Methods: Patients with HNC who underwent radiotherapy to the pharyngolaryngeal region were enrolled. Age-, sex-, and body mass index-matched individuals without HNC served as controls. Swallowing frequency, duration, submental EMG amplitude during swallowing, and respiratory phases before and after swallowing were assessed using PSG and compared between groups. Data normality was assessed using the Shapiro–Wilk test, appropriate parametric or nonparametric analyses were applied with statistical significance set at $p < 0.05$.

Results: Fifteen male patients with HNC and matched controls were analyzed. Swallowing frequency during sleep was significantly higher in the HNC group overall ($p = 0.016$), during rapid eye movement (REM) sleep ($p = 0.033$), and during non-REM sleep ($p = 0.015$). Swallowing duration during wakefulness was longer in the HNC group ($p = 0.015$), while no significant difference was observed during sleep ($p = 0.73$). Submental EMG amplitude during swallowing significantly decreased during sleep in the HNC group ($p = 0.025$). Respiratory pauses before and after swallowing were more frequent in the HNC group ($p = 0.002$ and $p = 0.013$, respectively).

Conclusion: Swallowing function during sleep may be impaired in patients with HNC. Because the pharyngolaryngeal muscles are common to both swallowing and airway patency, dysfunction in this region may contribute to OSA-related breathing disturbances. Early identification and management of OSA, combined with targeted swallowing rehabilitation, may help reduce the risk of aspiration, improve airway safety, and potentially lower the risk of life-threatening complications, including sudden death.

Keywords: head and neck cancer, swallowing function, obstructive sleep apnea, polysomnography, dysphagia

Introduction

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by transient hypoxia and sleep fragmentation due to upper airway obstruction. In adults, major risk factors include aging, male sex, obesity, anatomical narrowing of the upper airway, and craniofacial abnormalities. OSA is associated with various comorbidities, including hypertension, stroke, and heart failure.[1,2] Anatomical features contributing to OSA include soft tissue deposition around the upper airway, craniofacial morphology, tongue volume, and tonsillar hypertrophy.

Similarly, head and neck cancer (HNC) can cause anatomical abnormalities in the upper airway, thereby serving as a potential risk factor for OSA.[3,4] HNC refers to malignant tumors of the pharynx, larynx, or oral cavity. According to previous reports, the prevalence of OSA among patients with HNC is approximately 76% for oral and pharyngeal cancers and 72% for nasopharyngeal cancer. At our institution, we also observed that 78% of patients with HNC presented with comorbid OSA. Before treatment, tumor presence was considered a contributing factor to upper airway narrowing and

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consequent OSA.[5–7] Additionally, we encountered a case of severe OSA that developed due to laryngeal edema following chemoradiotherapy (CRT) for hypopharyngeal cancer, which was successfully managed with continuous positive airway pressure (CPAP) therapy.[8] These observations suggest that reductions in tumor size, body mass index (BMI), or neck circumference do not fully account for persistent OSA after treatment, and that impaired pharyngolaryngeal function may represent an important, yet underrecognized, contributor to ongoing sleep-disordered breathing.

Dysphagia is another major clinical concern in patients with HNC. Although radiotherapy (RT) preserves anatomical structures better than surgical resection, irradiation of the cervical region adversely affects mucosal integrity, salivary gland secretion, and neuromuscular coordination. As a result, dysphagia may occur both during the acute treatment phase and as a late complication, often impairing nutritional intake and quality of life. Recent global studies have demonstrated that a high proportion of patients already exhibit swallowing impairments before beginning HNC treatment, and that pretreatment swallowing function can predict the degree of post-treatment deterioration. For example, baseline swallowing measures have been shown to predict early recovery following transoral robotic surgery and other treatments.[9] These findings highlight the clinical relevance of understanding swallowing physiology throughout the disease course.

Despite the growing attention to dysphagia in HNC, nocturnal swallowing function remains poorly understood, not only in HNC but in clinical populations more broadly. Most previous swallowing studies rely on videofluoroscopy, endoscopy, or salivagram assessments performed during wakefulness. However, swallowing behavior varies markedly during sleep: protective reflexes are diminished, airway closure is less robust, and spontaneous swallowing frequency decreases substantially. These changes may increase susceptibility to silent aspiration, which is a major risk factor for aspiration pneumonia and has been implicated in sleep-related respiratory instability and sudden exacerbations of chronic disease in older adults.[10] Nevertheless, the mechanisms and characteristics of swallowing during sleep, especially in patients with altered upper airway anatomy after HNC treatment, remain largely unexplored. This represents an important knowledge gap with significant clinical implications.

Based on these considerations, our central hypothesis is that nocturnal swallowing function is significantly impaired in patients with HNC compared with individuals without HNC. Given the high risk of dysphagia and potential vulnerability to aspiration in patients with HNC, a more detailed understanding of nocturnal swallowing is needed. We hypothesized that patients with HNC have impaired pharynglaryngeal function during sleep compared with individuals without HNC, making them more susceptible to aspiration. Specifically, we assumed that polysomnography (PSG) would reveal decreased swallowing frequency, prolonged swallowing duration, reduced submental electromyographic (EMG) amplitude during swallowing, and abnormal timing of swallowing relative to respiratory phases—features that may increase aspiration risk. Therefore, the present study was designed to systematically evaluate swallowing function during sleep in patients with HNC using PSG. By characterizing nocturnal swallowing patterns in this population, we aimed to clarify potential mechanisms of aspiration during sleep and explore their implications for aspiration pneumonia prevention and overall clinical management in patients with HNC.

Methods

Patients

Patients diagnosed with HNC and treated with RT to the pharyngolaryngeal region at our institution were included in this study, provided they consented to undergo PSG prior to treatment. A control group of non-HNC individuals was established, matched to the patients with HNC by sex, age (±1 year), and BMI (±1 kg/m^2). To increase statistical power, we applied a one-to-two matching strategy, with two controls assigned to each HNC patient. Swallowing frequency, swallowing duration, amplitude of electromyographic (EMG) activity during swallowing, and respiratory phases before and after swallowing were compared between the groups. For each patient with HNC, two matched controls were selected for between-group comparison. The sample size was determined by the number of consecutive eligible HNC patients who underwent PSG during the study period; therefore, no a priori power calculation was performed. As HNC is more prevalent in men in our clinical population, the study sample reflects this epidemiological imbalance. This limitation and the need for inclusion of more female participants in future studies are acknowledged in the Discussion. Although the control group included individuals with moderate to severe OSA, matching was performed

based on age, sex, and BMI to minimize confounding by demographic and anthropometric factors. Because this study aimed to compare nocturnal swallowing physiology rather than to evaluate OSA severity itself, we selected controls who underwent PSG for clinical indications within the same diagnostic framework. The comparable apnea-hypopnea index (AHI) distribution between groups allowed us to isolate swallowing-related differences attributable to HNC rather than variations in OSA severity.

Polysomnography

Before treatment, all patients with HNC underwent overnight PSG using the Alice PDX system (Philips Respironics, Murrysville, PA, USA). PSG was conducted the day before the initiation of RT at the Department of Otolaryngology–Head and Neck Surgery, Juntendo University Hospital (Tokyo, Japan). Sleep data were scored according to the standard criteria of the American Academy of Sleep Medicine (AASM).[11] The diagnosis of OSA was made based on the International Classification of Sleep Disorders, Third Edition (ICSD-3).[12]

The apnea-hypopnea index (AHI) was defined as the total number of apneas and hypopneas per hour of sleep. OSA was diagnosed when the AHI exceeded five events per hour and ≥ 50% of the events were obstructive in nature. The following parameters were assessed: AHI, lowest oxygen saturation (SpO_2), time spent with oxygen saturation below 90%, oxygen desaturation index (ODI), swallowing frequency, swallowing duration, amplitude of EMG activity during swallowing, and respiratory phase immediately before and after swallowing.

Identification of Swallowing Events

Swallowing waveforms were confirmed during calibration in the awake state, and swallowing events during sleep were identified using these reference waveform characteristics. Consistent with a previously published method, swallowing events were defined as a simultaneous increase in the submental EMG activity and cessation of nasal and oral airflow on PSG.[13] Two experienced sleep specialists independently evaluated the swallowing events (Figure 1a). Airflow was measured using both a thermocouple and a pressure sensor; however, pressure signals were primarily used for analysis due to their higher sensitivity compared to thermal signals. Swallowing duration was defined as the time interval during which EMG activity remained elevated. Since EMG amplitude may vary between individuals due to factors such as electrode placement and subcutaneous fat, amplitudes during sleep were normalized to those recorded during wakefulness and expressed as relative ratios. For each swallowing event, the peak EMG amplitude was identified and used as an indicator of swallowing strength.

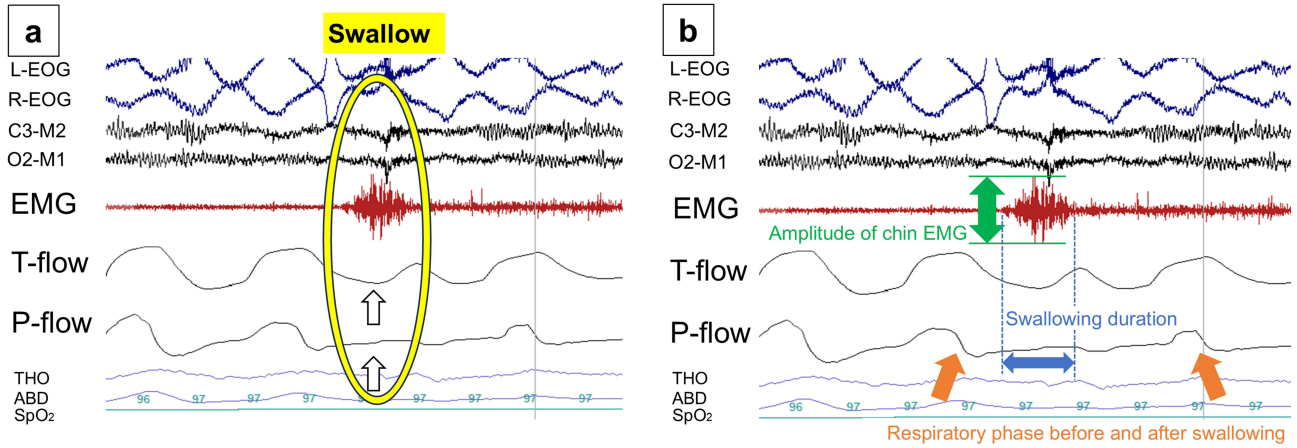

Figure 1 Submental EMG characteristics of swallowing events during PSG. (**a**) Representative submental EMG waveform of a swallowing event during PSG. The circle indicates a swallowing event, defined as a simultaneous increase in submental EMG activity and cessation of airflow detected by the thermistor (T-flow) and nasal pressure sensor (P-flow) on PSG (open arrows). (**b**) Duration and amplitude of a single swallow captured by submental EMG. Horizontal double arrow indicates the swallowing duration (blue); vertical double arrow indicates the amplitude of submental chin EMG (green); single arrows indicate the respiratory phase before and after swallowing (Orange).
Abbreviations: ABD, abdominal respiratory effort; C3-M2, EOG derivation between the left central electrode (C3) and the right mastoid (M2); EMG, electromyography; L-EOG, left electrooculogram; O2-M1, EOG derivation between the right occipital electrode (O2) and the left mastoid (M1); P-flow, pressure airflow; PSG, polysomnography; R-EOG, right electrooculogram; SO2, saturation of peripheral oxygen; T-flow, thermistor airflow; THO, thoracic respiratory effort.

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The respiratory phase before and after each swallow was categorized into one of three classifications: inspiration, expiration, or respiratory pause, and was analyzed accordingly (Figures 1b and 2). This method does not allow direct visualization of bolus flow or pharyngeal pressure patterns; therefore, gold-standard modalities such as videofluoroscopic swallowing study (VFSS) or high-resolution manometry (HRM) were not used. Consequently, our approach may underestimate subtle swallowing abnormalities. These methodological limitations are addressed in the Discussion.

Patient Selection

We included patients diagnosed with head and neck squamous cell carcinoma (HNSCC) – including nasopharyngeal, hypopharyngeal, laryngeal, and tongue cancers – at Juntendo University Hospital between April 1, 2017, and December 31, 2020. All patients received RT to the pharyngolaryngeal region and agreed to undergo PSG prior to treatment. Exclusion criteria were: (i) age < 20 years; (ii) previous treatment for sleep-disordered breathing before PSG, such as oral appliance (OA) use, CPAP therapy, or upper airway surgery; (iii) history of surgery for HNC and/or planned adjuvant therapy; (iv) history of severe lung disease or neuromuscular disease; (v) untreated cancers other than HNC; and (vi) presence of distant metastases.

This study was conducted in accordance with the Declaration of Helsinki (1964) and its later amendments. Ethical approval was obtained from the Ethics Committee of the Juntendo University Faculty of Medicine (E24-0351). As a retrospective study, an opt-out method was used to obtain informed consent. Participants were provided with information about the study and given the opportunity to decline participation. If no refusal was received, the data were considered available for use. Detailed oncologic characteristics such as tumor stage, radiation dose, and treatment duration were not included in the present analysis. Because our study focused on pre-treatment swallowing physiology, treatment-related dose–response relationships were beyond the scope of the study.

Statistical Analysis

All statistical analyses were conducted using SPSS Statistics version 29.0 (IBM Corp., Armonk, NY, USA). Normality of continuous variables was assessed using the Shapiro–Wilk test. For variables following a normal distribution, paired t-tests were used; for non-normally distributed variables, the Wilcoxon signed-rank test was applied. Categorical variables were analyzed using Fisher's exact test as a substitute for the chi-square test due to small expected cell counts. Statistical significance was set at $p < 0.05$. Unless otherwise specified, data are presented as median (interquartile range). Pre-treatment data alone were analyzed to avoid confounding effects from RT-related mucosal changes, edema, or treatment-induced dysphagia.

Results

Fifteen patients with HNC underwent PSG. Their clinical characteristics and sleep architecture parameters, including total sleep time, sleep efficiency, and proportions of non-rapid eye movenebt (N) and rapid eye movement (REM) sleep, are

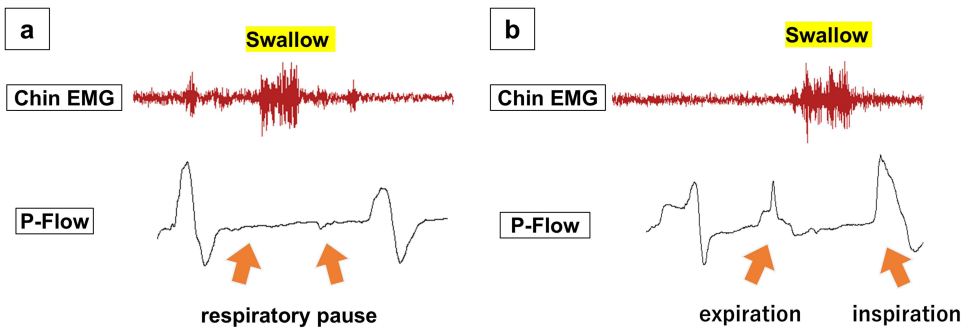

Figure 2 Example of submental EMG and pressure airflow during swallowing in PSG. Two of the six observed respiratory–swallowing patterns are illustrated. (**a**) Representative example showing a swallowing event after a respiratory pause, with respiratory pause observed both before and after the swallow. (**b**) Representative example showing a swallowing event after a expiration and a inspiration after swallowing. The arrows indicate each respiratory event.
Abbreviations: EMG, electromyography; P-flow, pressure airflow.

summarized in Table 1. Sleep architecture measures did not vay significantly between the groups, indicating that differences in swallowing activity were unlikely to be explained by variations in overall sleep structure. However, stage N3 sleep was significantly reduced in the HNC group compared with controls. All participants were male. The median age at the time of examination was 65.5 years (62.3–70.5) in the HNC group and 65 years (61.5–70.5) in the control group. The pre-treatment AHI was 31.1 events/h (15.6–46.2) in the HNC group and 31.2 events/h (15.4–41.7) in the control group.

Swallowing frequency during sleep was significantly higher in the HNC group, at 1.07 events/h (0.42–2.2), compared to 0.17 events/h (0–0.44) in the control group ($p = 0.016$). Regarding sleep stages, swallowing frequency during stage REM was 0 events/h (0–0.97) in the HNC group, compared to 0 events/h (0–0) in the control group, showing a significant difference ($p = 0.033$). During non-REM (NREM) sleep, swallowing frequency was also significantly higher in the HNC group, at 0.96 events/h (0.27–2.7), compared to 0.22 events/h (0–0.47) in the control group ($p = 0.015$) (Figure 3).

Swallow duration during EEG-defined wakefulness was significantly longer in the HNC group (2.5 s [2.0–4.0]) than in the control group (1.9 s [1.4–2.6]) ($p = 0.015$). In contrast, during sleep, no significant difference was observed between the HNC group (2.0 s [1.5–3.1]) and the control group (1.9 s [1.4–2.8]) ($p = 0.73$) (Figure 4).

Submental EMG amplitude during swallowing was significantly reduced during sleep compared to wakefulness in the HNC group (0.74-fold [0.56–1.1], $p = 0.025$), whereas no significant change was observed in the control group (1.0-fold [1.0–1.1], $p = 0.43$) (Table 2).

Regarding the respiratory phase before and after swallowing, the proportion of respiratory pauses preceding swallowing was significantly higher in the HNC group (32.3% [24.3–45.8]) than in the control group (0.0% [0–23.6]) ($p = 0.002$).

Table 1 Patient Charactertics

	HNC Patients (n=15)	Control (n=30)	*P* value
Age (years)	65.5 [62.3–70.5]	65 [61.5–70.5]	0.88
BMI (kg/m^2)	23.1 [21.0–24.3]	23.1 [21.2–24.5]	0.89
Sleep data			
TST (min)	409.5 [133.5–244.5]	377.3 [317.6–410.8]	0.21
Sleep efficiency (%)	79.5 [60–82.6]	74 [66.2–79.9]	0.40
Stage R (%)	14.4 [4.4–19.4]	14.9 [6.9–16.9]	0.92
Stage N1 (%)	41.7 [30.4–43.4]	28.8 [17.9–39.4]	0.13
Stage N2 (%)	46.5 [38.3–54.5]	48.9 [39–55.4]	0.69
Stage N3 (%)	0 [0–2.3]	4.4 [0–9.0]	0.009
Supine time/TST (%)	48.7 [20–96.4]	57.3 [39.3–89.9]	0.44
Arousal index	29.9 [21.6–38.5]	36 [22.3–47.5]	0.28
AHI (events/h)	31.1 [15.6–46.2]	31.2 [15.4–41.7]	0.80
AI (events/h)	7.7 [2.3–19.1]	7.1 [2.0–24.3]	0.84
HI (events/h)	11.8 [6.5–23.7]	13.9 [8.0–21.9]	0.72
Supine AHI (events/h)	37.5 [24.9–48.8]	37.5 [21.8–48.6]	0.92
3%ODI (events/h)	24 [8.9–41.3]	20.6 [5.8–36.3]	0.47
Lowest SpO_2 (%)	86 [83–90]	87 [81–90]	0.89
OSA (AHI>5) (n)	12	29	0.289
Tumor location			
Hypopharynx (n)	9	–	–
Larynx (n)	2	–	–
Nasopharynx (n)	2	–	–
Tongue (n)	1	–	–
Maxillary sinus (n)	1	–	–

Note: Values are expressed as median [interquartile range].
Abbreviations: HNC, head and neck cancer; BMI, body mass index; TST, total sleep time; R, rapid eye movement; N, non-rapid eye movement; AHI, apnea hypopnea index; AI, apnea index; HI, hypopnea index; ODI, oxygen desaturation index; SpO_2, saturation of peripheral oxygen; OSA, obstructive sleep apnea.

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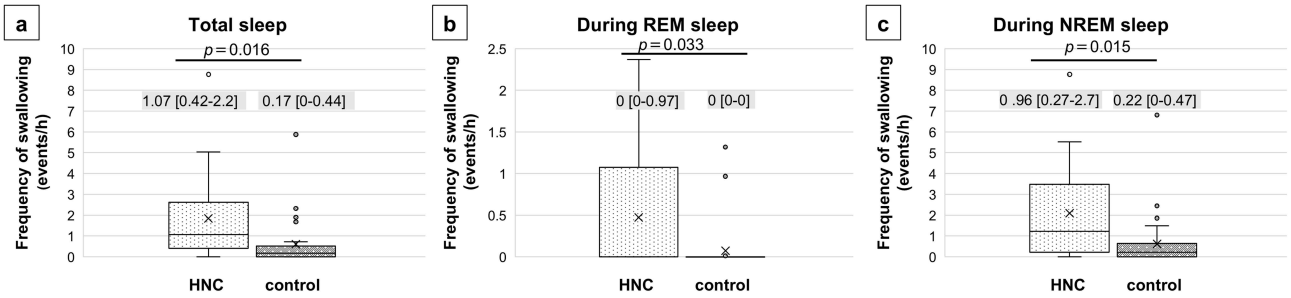

Figure 3 Swallowing frequency during sleep in the HNC and control groups. (**a**) Swallowing frequency during total sleep was significantly higher in the HNC group than in the control group (p = 0.016). (**b**) Swallowing frequency during REM sleep was significantly higher in the HNC group compared to the control group (p = 0.033). (**c**) Swallowing frequency during NREM sleep was significantly higher in the HNC group than in the control group (p = 0.015).
Abbreviations: HNC, head and neck cancer; NREM, non rapid eye movement; REM, rapid eye movement.

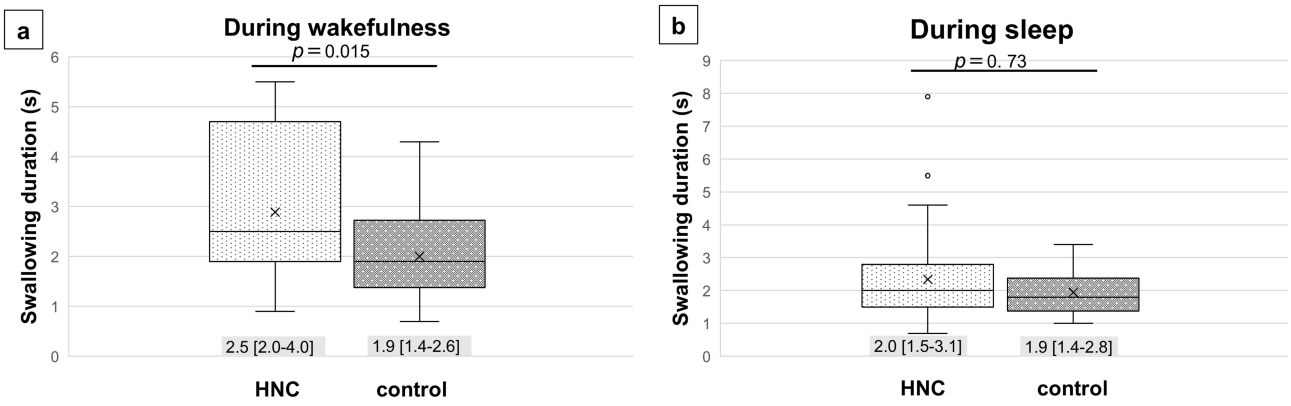

Figure 4 Swallowing duration was during wakefulness and sleep in the HNC and control groups. (**a**) Swallowing duration during EEG-defined wakefulness was significantly longer in the HNC group than in the control group (p = 0.015). (**b**) During sleep, there was no significant difference in swallow duration between the HNC group and the control group (p = 0.73).
Abbreviation: HNC, head and neck cancer.

Similarly, the proportion of respiratory pauses following swallowing was significantly greater in the HNC group (38.1% [9.2–50]) compared to the control group (0.0% [0–15.7]) (p = 0.013). In contrast, no significant differences were observed between the groups in the distribution of expiratory and inspiratory phases before and after swallowing (Table 2).

Table 2 Swalling-Relared EMG Amplitude and Respiratory Phase Patterns

	Variable	HNC patients	Control	*P* value
EMG amplitude	Awake	1	1	–
	Sleep	0.93±0.58	1.1±0.51	–
	Within-group comparison (awake vs sleep), *P* value	0.025	0.43	-
Respiratory phase patterns (%)	Swallowing after **respiratory pause**	32.3 [24.3–45.8]	0 [0–23.6]	0.002
	Swallowing after **expiration**	13.1 [4.2–39.3]	10 [0–75]	0.687
	Swallowing after **inspiration**	33.3[27.0–52.5]	48.4[32.1–55]	0.924
	Respiratory pause after swallowing	38.1 [9.2–50]	0 [0–15.7]	0.013
	Expiration after swallowing	8.8 [0–28.6]	29.2 [0–70]	0.224
	Inspiration after swallowing	48.4 [32.1–55]	50 [0–87.1]	0.924

Note: Values are expressed as median (interquartile range).
Abbreviations: EMG, electromyography; HNC, head and neck cancer.

Discussion

In the present study, we compared the number of swallows, swallowing duration, and respiratory phases before and after swallowing during sleep between patients with HNC and healthy controls. We also assessed submental EMG amplitude during swallowing under both wakefulness and sleep conditions. These analyses were performed to address our study aim of clarifying nocturnal swallowing physiology in HNC and identifying potential mechanisms related to airway protection during sleep. Our findings showed that swallowing frequency during sleep was significantly higher in the HNC group than in the control group. Although swallowing duration during wakefulness was longer in the HNC group, no significant difference was observed during sleep. Respiratory pauses before and after swallowing occurred more frequently in the HNC group. A significant decrease in submental EMG amplitude during sleep compared to wakefulness was noted in the HNC group, while no such change was observed in controls.

Previous studies have evaluated swallowing during nocturnal sleep using methods such as esophageal pressure monitoring or laryngeal elevation assessment during PSG.[14] However, no standardized approach has been established. Esophageal pressure monitoring involves transnasal catheter insertion, which may be uncomfortable for patients. In contrast, our study utilized standard PSG, enabling a minimally invasive assessment of swallowing in patients undergoing evaluation for sleep-disordered breathing.

While current clinical practice guidelines highlight the importance of dysphagia rehabilitation as a treatment-related complication in HNC, there is no specific mention of swallowing function during sleep.[15] Lear et al reported a mean swallowing frequency of 5.3 ± 1.7 times per hour in healthy adults, with increased activity during sleep onset and awakening, and prolonged intervals of over 20 min without swallowing events.[16] Similarly, Lichter et al observed an average swallowing frequency of 5.8 times per hour, with most events occurring during REM sleep (33.3%), NREM stage 1 (20.2%), and NREM stage 2 (31.7%).[17] In the present study, overall sleep architecture did not differ significantly between the groups, suggesting that variations in swallowing activity were not attributable to global differences in sleep structure. However, Stage N3 sleep was significantly reduced in the HNC group compared with controls. Although alterations in deep sleep could theoretically influence sensorimotor processes involved in swallowing, the limited degree of this reduction makes it unlikely to fully account for the distinct swallowing activity patterns observed. These findings indicate that factors more directly related to HNC pathology or its physiological impact may play a more prominent role than sleep architecture in shaping swallowing behavior during sleep.

Sato et al reported a median swallowing frequency of 2.1 times per hour in young adults and 0.6 times per hour in healthy elderly individuals, suggesting that swallowing frequency decreases with age.[18] In patients with OSA, this reduction is also observed, although there is no clear consensus on its magnitude.[14,19] Yagi et al reported that swallowing frequency during sleep increases with greater OSA severity. This has been attributed to arousal responses triggered by respiratory events, which may provoke swallowing. It has also been suggested that changes in oropharyngeal moisture and airflow associated with respiratory events act as stimuli for swallowing.[19] In our study, the overall swallowing frequency was lower than previously reported values, possibly because identification relied solely on submental EMG activity. Nevertheless, when comparing groups, patients with HNC exhibited significantly more swallowing events than controls. This may be explained by not only OSA-related impairment in salivary clearance but also tumor-related airway compression during sleep, which may necessitate increased swallowing to manage secretions. Tumor-related airway compression interfering with salivary clearance during sleep, necessitating increased swallowing to manage secretions. These results directly support our study aim by indicating that HNC alters nocturnal swallowing dynamics and may rely on compensatory increases in swallowing frequency to maintain airway patency.

Swallowing duration is also a clinically relevant parameter. While several studies have evaluated this during wakefulness using EMG or videofluoroscopic swallowing studies,[20–22] few have examined swallowing duration during sleep. One study found that patients with HNC who received swallowing rehabilitation showed no change in duration before and after CRT, whereas those without rehabilitation experienced prolongation.[23]

In our findings, swallowing duration during wakefulness was significantly longer in the HNC group, possibly due to anatomical compression and impaired salivary clearance. However, during sleep, no significant difference was observed. This may reflect a general prolongation of swallowing duration in both groups due to the reduced arousal state. Additionally, the

inclusion of elderly individuals and some patients with OSA in the control group may have contributed to reduced strength in swallowing-related muscles. In patients with OSA, swallowing events may be triggered by respiratory events, leading to abnormal patterns and a possible increase in swallowing duration. Taken together, the differences observed between wakefulness and sleep further highlight the need to assess both states when evaluating swallowing function in HNC.

Swallowing strength is another critical aspect. A 2019 study using HRM reported that pharyngeal pressure during swallowing is lower during sleep than wakefulness.[24] In our study, submental EMG amplitude was used as a surrogate marker of swallowing strength. While EMG amplitude does not directly reflect pharyngeal pressure, it represents muscle contraction intensity and serves as a supplementary indicator. In the present study, high-precision assessments such as HRM or VFSS were not performed due to concerns regarding invasiveness, particularly during sleep. Based on this approach, our findings showed reduced EMG amplitude during sleep in patients with HNC. Although no significant difference was observed between wakefulness and sleep in controls, the decrease in EMG amplitude was more pronounced in the HNC group. These results suggest weaker swallowing during sleep in patients with HNC. This diminished nocturnal swallowing strength may contribute to incomplete salivary clearance and increased vulnerability to aspiration.

Coordination between swallowing and respiration is also vital. Sato et al reported that patients with OSA often resume respiration with inspiration after swallowing. If both pre- and post-swallowing respiratory phases involve inspiration, the risk of aspiration of oral secretions increases, suggesting a less protective swallowing pattern. However, the use of CPAP has been shown to reduce the proportion of inspiratory swallows in patients with OSA.[25] In the present study, we hypothesized that tumor-related airway compression would increase the frequency of inspiratory swallowing in patients with HNC due to impaired function. Contrary to expectations, the HNC group showed a significantly higher proportion of respiratory pauses before and after swallowing. This may be due to the presence of moderate-to-severe OSA with AHI values of approximately 30 events/h in both groups, leading to a high prevalence of uncoordinated swallows and no significant difference in inspiratory swallows between groups. The frequent respiratory pauses observed in the HNC group may reflect a compensatory breath-holding mechanism during swallowing in response to tumor-induced obstruction. This compensatory pattern may represent a protective adaptation to minimize aspiration risk, consistent with the clinical relevance of our findings. These physiological changes during sleep carry important clinical implications. Nocturnal swallowing is essential for maintaining oral and pharyngeal hygiene, clearing bacteria, and neutralizing nocturnal reflux – vital functions for survival.[26] Our findings indicate that patients with HNC demonstrate higher swallowing frequency and reduced swallowing pressure during sleep compared to controls. This suggests that nocturnal swallowing in HNC may be incomplete, and an increased swallowing frequency could be a compensatory mechanism to maintain airway clearance and reduce aspiration risk. Such insights may guide future rehabilitation or screening strategies, including early nocturnal swallowing assessment or evaluation for coexisting OSA in HNC patients.

This study included patients with HNC with a mean AHI of approximately 30 events/h. Although some control participants had moderate to severe OSA, the mean AHI was comparable between the HNC and control groups. Therefore, the potential impact of sleep-disordered breathing on nocturnal swallowing is expected to be similar in both groups. As a result, differences observed in the swallowing activity are more likely attributable to the presence of HNC rather than variations in OSA severity. HNC is frequently associated with lifestyle risk factors such as smoking and alcohol use, which are also linked to OSA. Cardiovascular complications and an increased risk of sudden death have been reported in untreated OSA. Likewise, cancer patients, including those with HNC, are known to have an elevated risk of sudden death.[2,27] One of the pathophysiological features of OSA is decreased responsiveness of upper airway dilator muscles such as the genioglossus.[28] Aspiration may also involve reduced strength in swallowing muscles like the genioglossus and geniohyoid, suggesting an overlap in the muscular systems involved in both OSA and swallowing. Treatments for OSA, such as CPAP and OAs, may not only improve OSA but also help maintain swallowing function and reduce aspiration risk by supporting these muscle groups. Therefore, the evaluation of coexisting OSA using PSG should be considered in patients with HNC. If present, treatment options such as CPAP or OA should be explored to reduce the risk of aspiration pneumonia and sudden death. However, it may be difficult to implement daily-use interventions like CPAP or OA or perform sleep-related surgeries in patients undergoing HNC treatment. Thus, daytime rehabilitation strategies, including swallowing and myofunctional therapy, may help preserve pharyngeal muscle strength, prevent aspiration, and potentially aid in the management of OSA (Figure 5). Given the limited sample size

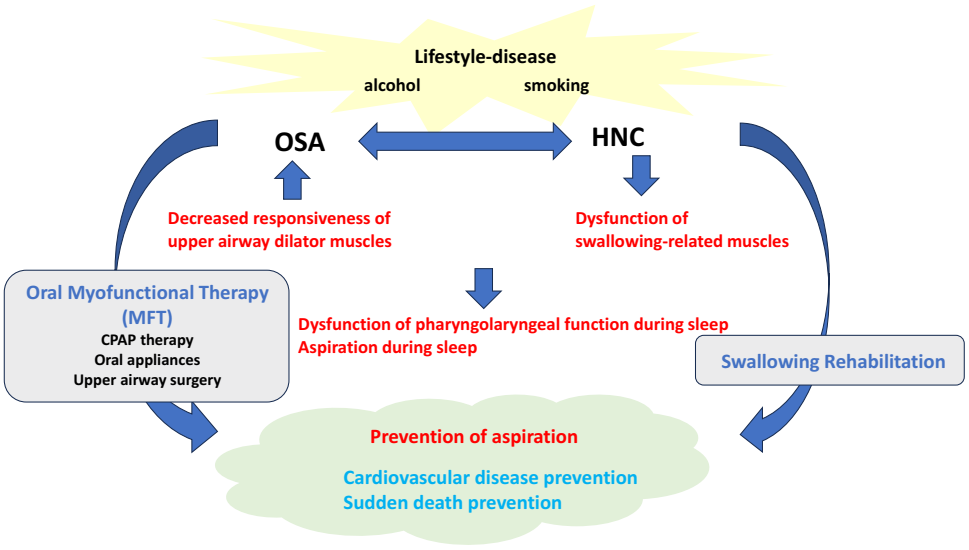

Figure 5 Conceptual model linking HNS, OSA, and swallowing dysfunction This figure illustrates the proposed relationships among head and neck cancer (HNC), obstructive sleep apnea (OSA), and nocturnal swallowing impairment. HNC-related factors (eg, tumor burden, lifestyle risks) and OSA both contribute to reduced upper airway dilator muscle responsiveness, including the genioglossus and geniohyoid, which in turn increases aspiration risk. PSG-based evaluation of coexisting OSA is recommended in patients with HNC. When OSA is present, treatments such as CPcontinuoud airway pressure (CPAP) or oral appliances may help maintain swallowing muscle function and reduce the risk of aspiration pneumonia or sudden death. For patients undergoing HNC treatment, in whom daily-use devices may be difficult to adopt, daytime interventions such as swallowing rehabilitation and myofunctional therapy may support pharyngeal muscle strength and provide adjunctive benefit.

of the present study, these clinical implications should be interpreted with caution, and future studies with larger cohorts are needed to further validate these findings.

Limitations

This study has some limitations. First, differences in swallowing function based on tumor location or clinical stage of HNC were not examined. Second, swallowing assessment relied solely on submental EMG, which may have underestimated actual swallowing activity or strengths. Although two expert raters performed the scoring, the absence of formal inter-rater reliability analysis represents an additional methodological limitation. Third, the sample size was relatively small, and all participants were male, which may limit the generalizability of the findings. In addition, post-treatment evaluation was not performed because many patients were unable to undergo the assessment due to treatment-related pain and other adverse effects. However, longitudinal follow-up after treatment is highly important for understanding changes in swallowing function over time, and future studies should address this issue. Larger studies incorporating more diverse populations and more comprehensive assessment methods are needed to validate these results.

Conclusion

Our findings indicate a possible association between HNC and reduced swallowing function during sleep. Because the pharyngeal muscles involved in swallowing overlap with those implicated in OSA, sleep-disordered breathing may also contribute to swallowing impairment in this population. These results suggest that swallowing rehabilitation and appropriate management of coexisting OSA may help reduce aspiration risk in patients with HNC. Although our results suggest that sleep evaluation, including PSG, could be informative, further research is warranted before considering routine use in clinical practice. The present study was limited by its small sample size, all-male cohort, and evaluation of pre-treatment status only; therefore, the conclusions should be interpreted as hypothesis-generating rather than definitive. Future studies with larger and more diverse samples, as well as longitudinal post-treatment follow-up, are warranted to clarify these associations and determine whether targeted interventions could help reduce the risk of aspiration.

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Data Sharing Statement

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki and its later amendments. The study was approved by the ethics committee of the Juntendo University Faculty of Medicine (E24-0351).

Consent to Participate

This study employed an opt-out consent process approved by the institutional review board. Participants were informed about the study and had the opportunity to decline participation.

Author Contributions

Erina Ishimizu: Conceptualization; Methodology; Investigation; Data curation; Formal analysis; Writing – original draft; Writing – review & editing; Ayako Inoshita: Conceptualization; Methodology; Investigation; Data curation; Formal analysis; Writing – review & editing; Fusae Kawana: Formal analysis; Writing – review & editing; Nanako Shiroshita: Formal analysis; Writing – review & editing; Shinichi Ohba: Conceptualization; Writing – review & editing; Takatoshi Kasai: Conceptualization; Supervision; Writing – review & editing; Fumihiko Matsumoto: Conceptualization; Supervision; Writing – review & editing.

All authors approved the final manuscript and agreed on the journal to which the article has been submitted and agree to be accountable for all aspects of the work.

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References

1. He J, Kryger MH, Zorick FJ, et al. Mortality and apnea index in obstructive sleep apnea. *Chest*. 1988;94:9–14. doi:10.1378/chest.94.1.9
2. Newman AB, Javier Nieto F, Guidry U, et al. Relation of Sleep-disordered breathing to cardiovascular disease risk factors. *Am J Epidemiol*. 2001;154(1).
3. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328:1230–1235. doi:10.1056/NEJM199304293281704
4. White DP. Pathogenesis of obstructive and central sleep apnea. *Am J Respir Crit Care Med*. 2005;172:1363–1370. doi:10.1164/rccm.200412-1631SO
5. Payne RJ, Hier MP, Kost KM, et al. High prevalence of obstructive sleep apnea among patients with head and neck cancer. *J Otolaryngol*. 2005;34:304. doi:10.2310/7070.2005.34502
6. Lin H-C, Friedman M, Chang H-W, et al. Impact of head and neck radiotherapy for patients with nasopharyngeal carcinoma on sleep-related breathing disorders. *JAMA Otolaryngol Head Neck Surg*. 2014;140:1166–1172. doi:10.1001/jamaoto.2014.2953
7. Inoshita A, Sata N, Ohba S, et al. Impact of radiotherapy for head and neck cancer on obstructive sleep apnea: a prospective study. *Ann Palliat Med*. 2022;11:2631–2640. doi:10.21037/apm-22-267
8. Inoshita A, Matsumoto F, Ohba S, et al. Severe obstructive sleep apnea after concurrent chemoradiotherapy for laryngeal and hypopharyngeal cancer managed by CPAP. *Auris Nasus Larynx*. 2022;49:1078–1082. doi:10.1016/j.anl.2021.05.003
9. Owen S, Puvanendran M, Meikle D, et al. Baseline swallowing measures predict recovery at 6 weeks after transoral robotic surgery for head and neck cancer. *Clin Otolaryngol*. 2017;42(2):366–372. doi:10.1111/coa.12731
10. Kikuchi R, Watabe N, Konnno T, et al. High incidence of silent aspiration in elderly patients with community-acquired pneumonia. *Am J Respir Crit Care Med Jul*. 1994;150(1):251–253. doi:10.1164/ajrccm.150.1.8025758

11. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. deliberations of the sleep apnea definitions task force of the American Academy of Sleep Medicine. *J Clin Sleep Med*. 2012;8:597–619. doi:10.5664/jcsm.2172
12. American Academy of Sleep Medicine. *International Classification of Sleep Disorders*. 3rd. American Academy of Sleep Medicine; 2014.
13. Sato K, Nakashima T. Human adult deglutition during sleep. *Ann Otol Rhinol Laryngol*. 2006;115:334–339. doi:10.1177/000348940611500503
14. Yagi K, Lowe AA, Ayas NT, et al. Swallowing and breathing patterns during sleep in patients with obstructive sleep apnea. *Sleep Breath*. 2015;19:377–384. doi:10.1007/s11325-014-1031-8
15. Homma A, Ando M, Hanai N, et al. Summary of Japanese clinical practice guidelines for head and neck cancer - 2022 update edited by the Japan society for head and neck cancer. *Auris Nasus Larynx*. 2024;51:174–188. doi:10.1016/j.anl.2023.07.003
16. Lear CS, JB F, Moorrees CF. The frequency of deglutition in man. *Arch Oral Biol*. 1965;10:83–100. doi:10.1016/0003-9969(65)90060-9
17. Lichter I, Muir RC. The pattern of swallowing during sleep. *Electroencephalogr Clin Neurophysiol*. 1975;38:427–432. doi:10.1016/0013-4694(75)90267-9
18. Sato K, Chitose S-I, Sato K, Umeno H. Deglutition and respiratory patterns during sleep in the aged. *Acta Otolaryngol*. 2016;136:1278–1284. doi:10.1080/00016489.2016.1203991
19. Sato K, Nakashima T. Sleep-related deglutition in patients with sleep apnea-hypopnea syndrome. *Ann Otol Rhinol Laryngol*. 2009;118:30–36. doi:10.1177/000348940911800106
20. Uchida M, Yamaguchi I, Tsukioka R, Sakurazawa T, Manabe Y, Katou M. Ultrasound treatment for swallowing difficulty in Parkinson's disease - thermotherapy to the infrahyoid muscles improves the deglutition efficiency. *Keishin J Life Health*. 2018;2(2):35–40. doi:10.24759/vetrdi.2.2_35
21. Sato A, Taniai S, Akiyama N, et al. Determining the function of infrahyoid muscles based on temporal parameters of activities of the submental and infrahyoid muscles during swallowing. *Drglutition*. 2023;12:180–188.
22. Paydarfar D, Gilbert RJ, Poppel CS, Nassab PF. Respiratory phase resetting and airflow changes induced by swallowing in humans. *J Physiol*. 1995;483:273–288. doi:10.1113/jphysiol.1995.sp020584
23. Furuie H, Hamamoto T, Chikuie N, et al. Evaluation of role of prophylactic swallowing rehabilitation in chemoradiotherapy for advanced head and neck cancer using novel software analysis of videofluorography images. *Hiroshima J. Med. Sci*. 2019;68;(2–3):27–34
24. Guiu Hernandez E, Gozdzikowska K, Jones RD, Huckabee M-L. Pharyngeal swallowing during wake and sleep. *Dysphagia*. 2019;34:916–921. doi:10.1007/s00455-019-09989-6
25. Sato K, Chitose S-I, Sato K, et al. Sleep-related deglutition and respiratory phase patterns in the aged with obstructive sleep apnea under CPAP therapy. *Acta Otolaryngol*. 2020;140:861–868. doi:10.1080/00016489.2020.1772504
26. Orr WC, Heading R, Johnson LF, Kryger M. Review article: sleep and its relationship to gastro-oesophageal reflux. *Aliment Pharmacol Ther*. 2004;20(9):39–46. doi:10.1111/j.1365-2036.2004.02239.x
27. Sturgeon KM, Deng L, Bluethmann SM, et al. A population-based study of cardiovascular disease mortality risk in US cancer patients. *Eur Heart J*. 2019;40:3889–3897. doi:10.1093/eurheartj/ehz766
28. Pierce R, White D, Malhotra A, et al. Upper airway collapsibility, dilator muscle activation and resistance in sleep apnoea. *Eur Respir J*. 2007;30:345–353. doi:10.1183/09031936.00063406