Title

Masseter Muscle Size in Chronic Parotid Sialadenitis.

Journal

Laryngoscope Investigative Otolaryngology, 10(3)

ISSN

2378-8038

Authors

Stephens, Erika
Nesmith, William
Ogden, M
et al.

Publication Date

2025-06-01

DOI

10.1002/lio2.70160

Laryngoscope
Investigative Otolaryngology

ORIGINAL RESEARCH OPEN ACCESS

Masseter Muscle Size in Chronic Parotid Sialadenitis

Erika M. Stephens[1] | William E. Nesmith[1] | M. Allison Ogden[2] | Jolie L. Chang[1,3]

[1]Department of Otolaryngology-Head and Neck Surgery, University of California-San Francisco, San Francisco, California, USA | [2]Department of Otolaryngology- Head and Neck Surgery, Washington University, St. Louis, Missouri, USA | [3]Department of Veterans Affairs Medical Center, Surgery Service, San Francisco, California, USA

Correspondence: Jolie L. Chang (jolie.chang@ucsf.edu)

Received: 11 November 2024 | **Revised:** 7 February 2025 | **Accepted:** 2 May 2025

Keywords: masseter size | salivary glands | salivary stones | sialadenitis | sialendoscopy | stenosis

ABSTRACT

Objective: The relationship between masseter muscle thickness and parotid duct obstruction resulting in recurrent sialadenitis is not well defined. This study aims to evaluate masseter muscle size in patients with chronic sialadenitis of the parotid and compared to patients without parotid sialadenitis.

Methods: Data was collected retrospectively from patients with symptomatic chronic sialadenitis of the parotid or submandibular glands. Measurements of the masseter and lateral pterygoid muscles were performed on CT or MRI imaging by two investigators who were blinded to the symptomatic gland location. Masseter thickness in the region of the parotid duct trajectory was measured.

Results: Out of 94 total patients, 45 (48%) had chronic sialadenitis of the parotid gland and 49 (52%) had sialadenitis of the submandibular gland without parotid symptoms. There was a statistically significant difference in masseter thickness between patients with symptomatic parotid versus submandibular sialadenitis (*parotid*: 15.8 mm; *submandibular:* 14.5 mm; $p < 0.001$). Patients with parotid sialolithiasis compared to duct stenosis had no difference in masseter thickness size. Higher BMI, male sex, and parotid gland symptoms were significantly associated with increased masseter thickness. Logistic regression analysis showed that female sex and masseter muscle thickness were both significantly associated with parotid gland sialadenitis.

Conclusion: Masseter muscle thickness is significantly larger in patients with symptomatic obstructive parotid sialadenitis. Our findings demonstrate an association between increased masseter size and obstructive parotid sialadenitis that may have implications in disease pathophysiology and considerations for therapeutic paradigms.

Level of Evidence: Level 4.

1 | Introduction

Chronic sialadenitis presents with facial swelling and pain during meals over the affected major salivary gland(s) [1]. Chronic sialadenitis is most often due to obstructive etiologies, including duct stenosis and salivary stones, which can be caused by autoimmune disease, radioactive iodine therapy, trauma, infections, or idiopathic causes [1–3]. Parotid sialadenitis is most commonly related to salivary duct stenosis and submandibular gland sialadenitis is associated more with sialolithiasis [1, 3, 4]. A potential relationship between masseter muscle size and chronic parotid sialadenitis has been described; however, there has been minimal work to better characterize the relationship between the masseter muscle and chronic parotid sialadenitis. Masseter muscle size can alter and affect the trajectory of the parotid duct and cause external compression on the duct. Case reports have described difficulty with parotid duct dilation and duct kinking at the anterior border of the masseter approximately 2 cm from Stensen's duct papilla [2].

Masseteric muscle hypertrophy (MMH) refers to a progressive increase in the size of the masseter muscle [5, 6]. MMH often

presents cosmetically with a characteristically rectangular/square face with fullness over the angle of the mandible unilaterally or bilaterally. The etiology of masseter muscle enlargement is debated but is primarily thought to result from use-incited hypertrophy [5]. Consequently, MMH has historically been associated with conditions such as bruxism and temporomandibular joint disorder (TMJ), as well as maladaptive excessive bruxism and chewing activity or habits [5–7]. Among the documented cases of MMH, associated clinical disorders of the nearby parotid salivary glands have neither been well described nor explored. Notably, recent studies have highlighted a potential relationship between MMH and chronic sialadenitis [2, 7, 8]. Capaccio et al. demonstrated an increase in parotid duct diameter on MR sialography in parotid sialadenitis [8] and in a case report demonstrated potential anatomic changes to the duct course due to masseter muscle dysfunction [7]. A non-significant increase in masseter muscle size was seen in patients with recurrent parotitis [8].

We aim to better characterize the relationship between masseter muscle size and chronic obstructive symptoms of the parotid glands. We hypothesize that masseter muscle thickness will be greater in patients with symptomatic parotid sialadenitis compared to patients without parotid sialadenitis symptoms.

2 | Methods

This study was approved by the University of California-San Francisco Institutional Review Board.

Data was collected from patients who presented to the UCSF Otolaryngology—Head and Neck Surgery clinic between 2013 and 2021 with symptoms of chronic sialadenitis in the parotid or submandibular glands defined as repeated episodes of swelling of a major salivary gland occurring for longer than 3 months. Patients with 3-dimensional CT or MRI imaging of the head and neck were included. Electronic health record data, including pertinent demographic information, affected gland(s), laterality of symptoms, and the presence of sialolithiasis and/or stenosis on imaging and sialendoscopy, were abstracted and stored in Microsoft Excel (Microsoft Corporation, Redmond, WA). Submandibular sialadenitis patients were used as the control group. We chose a comparison group within a cohort of patients evaluated for sialadenitis in order to classify patients specifically as those with parotid sialadenitis symptoms versus patients without parotid-related symptoms (control). A specific assessment of salivary gland symptoms and location was performed. Each patient was queried about their symptom location using a salivary symptom survey and diagnosed by an otolaryngologist who specializes in obstructive salivary disorders. Patients with symptoms in both submandibular and parotid glands were excluded.

3 | Measurement Methods and Statistical Analysis

Measurements were performed on CT or MRI imaging by two different investigators who were blinded to the patient's symptomatic gland(s) or symptom laterality. For each participant, left and right maximum dimensions (length, width) of the masseter and lateral pterygoid muscles were measured on axial CT or MRI images. Masseter thickness was measured from the surface of the mid-belly of the masseter to the mandibular ramus perpendicular to the plane of the skin and muscle surface (Figure 1). Each side was measured independently on the image that captured the parotid gland hilum and the course of the parotid duct. The lateral pterygoid muscle was measured in the thickest midportion of the muscle. The lateral pterygoid muscle was chosen as the comparator muscle of mastication that differed in action from the masseter. Average masseter thickness was also compared to a literature review of similar measurement locations for normative values.

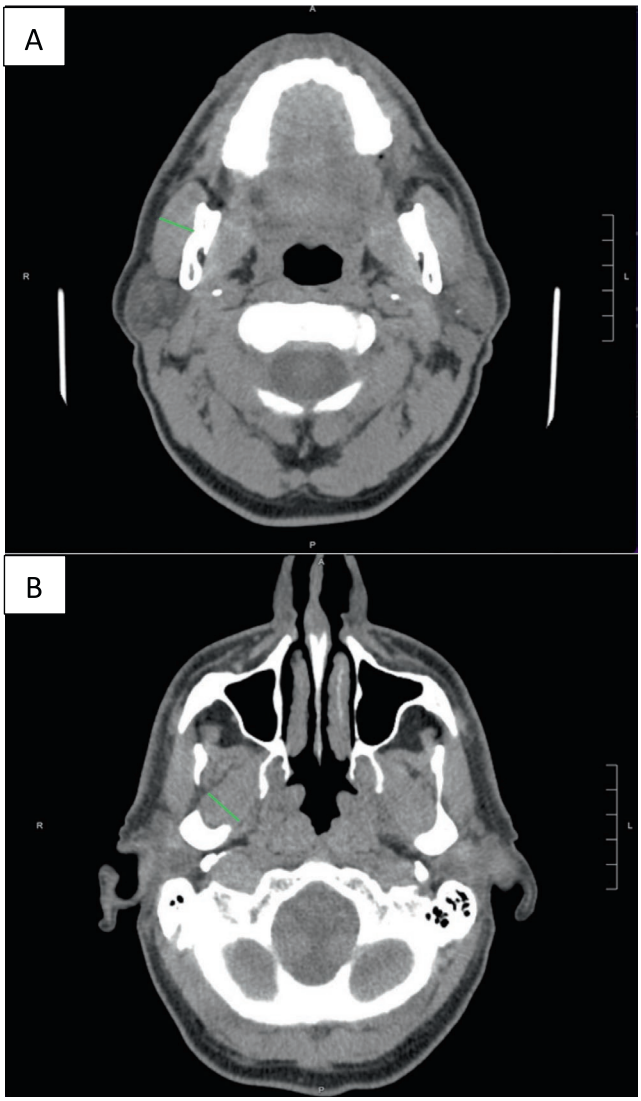

FIGURE 1 | Masseter and lateral pterygoid measurements (A) Right masseter muscle thickness (B) Right lateral pterygoid muscle thickness.

Statistical analysis and figure design were performed using the Stata Statistical Software (Release 17, StataCorp LLC, College Station, TX). Comparisons between the right and left masseters and the right and left lateral pterygoids were performed using the Student's *t*-test. Chi square analysis was used to examine the relationship between masseter thickness, sialoliths, and stenosis. Multivariate regression analysis was used to evaluate predictors for masseter muscle size (BMI, sex, and age) and separately for parotid sialadenitis presence (BMI, sex, masseter thickness, and age).

4 | Results

4.1 | Patient Demographics

A total of 99 patients with imaging were analyzed; 5 were excluded for symptoms in both submandibular and parotid glands. Out of 94 total patients, 45 (48%) had parotid gland symptoms, and 49 (52%) had submandibular gland symptoms without parotid obstruction. Unilateral symptoms were most common ($n = 80$, 81%). A majority of patients were women ($n = 59$, 63%), with a mean age of 59 ± 13.2 years. The total number of symptomatic glands in the cohort was 122. Of those, 64 (52%) glands had sialolithiasis and 58 (48%) glands had duct stenosis (Table 1).

4.2 | Muscle Thickness Characteristics

The mean masseter thickness in the full cohort was 15.1 mm (SD: 2.3). Patients with parotid gland sialadenitis had significantly greater mean masseter thickness compared to the submandibular sialadenitis cohort (*parotid group*: 15.8 mm; *submandibular group*: 14.5 mm; $p < 0.001$, Table 2). The parotid sialadenitis group had significantly larger masseters on both right and left sides (Table 2). The mean lateral pterygoid thickness was 14 mm (SD: 2.1), with no significant difference between patients with parotid versus submandibular sialadenitis (*parotid group*: 13.6 mm; *submandibular group*: 14.4 mm; $p = 0.05$).

Analysis of the parotid sialadenitis cohort (Table 3) showed that 29 (64%) patients had parotid duct stenosis, and 16 (36%) had parotid sialolithiasis. There was no significant difference in masseter thickness between those with stenosis or sialolithiasis ($p = 0.22$). In 33 patients with unilateral parotid symptoms, there was no significant difference in masseter thickness between the symptomatic versus non-symptomatic sides (mean difference + 0.29 mm (SD 2.65)).

Inter-rater reliability of right and left masseter thickness measurements was calculated using a two-way mixed effects intraclass correlation coefficient (Right masseter: ICC = 0.77, 95%

TABLE 1 | Demographics.

		Parotid symptoms	Submandibular symptoms	*P**
Patients, *N* (%)	94	45 (48%)	49 (52%)	
Imaging Modality, *N* (%)		CT: 35 (78%) MRI: 10 (22%)	CT: 45 (92%) MRI: 4 (8%)	
Female: *N* (%)	59 (63%)	34 (76%)	25 (51%)	0.01*
Age, mean (SD); years	59 (13.2)	58 (13)	59 (13)	0.08
BMI, mean (SD); kg/m^2	27 (6.2)	28 (7)	27 (5)	0.51
Laterality, *N* (%)				
Unilateral	80 (81%)	32 (40%)	47 (59%)	
Bilateral	19 (19%)	13 (68%)	2 (11%)	
Glands, *N*	122	55	67	
Cause, *N* (% of glands)				
Sialolithiasis	64 (52%)	15 (27%)	49 (73%)	
Stenosis (non-sialolithiasis)	58 (48%)	40 (73%)	18 (27%)	

*$p < 0.05$ indicates statistical significance, comparisons made between parotid versus submandibular symptom groups.

TABLE 2 | Masseter thickness.

Masseter thickness (mm), Mean (SD)	Parotid Sialadenitis ($N = 45$)	Submandibular Sialadenitis ($N = 49$)	*p*-value
All	15.8 (2.4)	14.5 (2)	< 0.001*
Right	16.1 (2.8)	14.7 (2.9)	0.02*
Left	15.4 (2.6)	14.3 (2.3)	0.02*
Lateral Pterygoid thickness (mm), Mean (SD)			
All	13.6 (2.1)	14.4 (2)	0.05
Right	13.7 (2.5)	14.5 (2.4)	0.09
Left	13.5 (2.1)	14.3 (2.3)	0.07

*$p < 0.05$ indicates statistical significance.

TABLE 3 | Characteristics of patients with parotid sialadenitis.

Symptomatic parotid glands	Sialolithiasis	Stenosis	*p*-value
Patients, *N* (%)	16 (36%)	29 (64%)	
Masseter thickness (mm), Mean (SD)	16.4 (2.6)	15.3 (2.4)	0.22
Patients with Unilateral Symptoms (Parotid), *N*	**15**	**18**	
Difference in Masseter thickness (*R* vs. L side, mm), Mean (SD)	0.5 (3.14)	0.3 (2.8)	0.63
Difference in Masseter thickness (Symptomatic vs. Non-symptomatic side, mm), Mean (SD)	0.7 (3.8)	0.7 (2.9)	0.39

TABLE 4 | Logistic regression analysis: Predictors for parotid sialadenitis.

Variable	Odds ratio	Standard error	Wald	*p*-value
Female sex	8.51	5.03	3.63	<0.001*
Masseter thickness	1.67	0.23	3.70	<0.001*
BMI	0.96	0.04	−1.07	0.29
Age	1.01	0.02	0.33	0.74

*$p < 0.05$ indicates statistical significance.

CI = 0.61–0.87; Left masseter: ICC = 0.81, 95% CI = 0.67–0.89), indicating good reliability.

4.3 | Multivariate Regression Analysis

Multivariate linear regression was used to examine associations for masseter thickness using BMI, age, sex, and affected gland (parotid or submandibular). Mean masseter thickness in men was 16.1 mm (SD: 2.2), and mean masseter thickness in women was 14.5 mm (SD: 2.1). Multivariate analysis showed that higher BMI, male sex, and parotid gland sialadenitis were significantly associated with increased masseter thickness (all $p < 0.001$).

Logistic regression analysis examined the predictors BMI, age, sex, and masseter thickness for the outcome parotid sialadenitis (Table 4). Both female sex and increased masseter thickness were significant predictors of parotid gland sialadenitis ($p < 0.001$). Larger masseter size was associated with a 67% increase in the odds of parotid sialadenitis per 1 mm unit increase in thickness. BMI and age were not associated with parotid versus submandibular sialadenitis.

5 | Discussion

This study demonstrated that masseter thickness measured at the mid-masseter was significantly greater in patients with parotid gland sialadenitis compared to submandibular gland sialadenitis patients without parotid symptoms. There was no difference in masseter size in patients with parotid sialolithiasis versus stenosis and no difference between symptomatic and asymptomatic sides. Logistic regression analysis showed both increased masseter size and female sex were each significant predictors of symptomatic parotid sialadenitis when controlling for BMI and age. These findings raise interesting questions about the possible relationships between parotid gland sialadenitis and increased masseter muscle size and generate potential opportunities for novel treatment paradigms. This is the first study to examine how masseter muscle size may play a role in the pathophysiology of obstructive parotid sialadenitis.

Using values in this study, parotid sialadenitis patients had greater masseter muscle thickness (average 15.8 mm) compared to normative masseter thickness values in the literature with average 13.6 mm (9–13 mm in women, 10–15 mm in men) in the relaxed state [9]. The causes of masseter muscle enlargement are varied, and the etiology is not always clear. Potential causes include bruxism, TMJ disorders, malocclusion, clenching, and over-use [6]. The condition can develop insidiously over a number of years. Various imaging modalities, including CT, MRI, and ultrasound (US) can be used to measure the masseter muscle [10]. While US has the advantage of portability, real-time evaluation, and cost-effectiveness, proper protocol is essential to minimize discrepancies due to varied transducer design, variation of technique between different operators, dependence of pressure altering the masseter thickness, and difficulty recognizing muscle borders [9–11]. Given these limitations, our study utilized CT and MRI imaging to measure masseter thickness, but point-of-care use of ultrasound for masseter muscle assessments may enhance access to measurements.

This study did not find a significant difference in masseter size in patients presenting with parotid gland obstruction due to sialolithiasis versus stenosis. Interestingly, no difference was found between symptomatic and asymptomatic sides; however, the analysis was limited by the number of patients with unilateral disease. On average, the symptomatic masseter side was larger than the asymptomatic side, but the difference was not significant. One explanation for the lack of difference in muscle size between sides may be related to the paired masseter muscle activity with jaw function. If baseline bruxism or TMD occurs over many years, masseter muscle hyperactivity and hypertrophy may occur together bilaterally. A few patients reported preferential chewing or use of one side of the jaw, which may be related to parotid disease in some cases, but this was not queried or captured in the study cohort. Prior studies have also failed to find a difference between right and left masseter muscle thickness in individuals who chew unilaterally [12].

Our finding that masseter thickness is significantly larger in patients with parotid symptoms suggests that larger masseter size may play a pathophysiologic role in obstructive parotid disease. The relationship may be multimodal. Anatomically, the main portion of the mid parotid duct travels superficial to the masseter muscle. Hypertrophy of the masseter muscle may contribute to obstruction of Stenson's duct by altering duct trajectory over an enlarged muscle and slowing normal salivary flow. Certain individuals prone to salivary duct scarring or stenosis as a consequence of salivary stasis, may further lead to exacerbation of obstruction and development of chronic disease. In a 2011 case report, Reddy et al. found that in two patients, masseter muscle size resulted in difficulty reaching the parotid hilum during sialendoscopy [2]. In a third patient, Stenson's duct illustrated dilation as it passed over a hypertrophic masseter muscle. In all three cases, decreasing the masseter muscle size with either a debulking procedure or botulinum toxin injections improved symptoms. This study examines the association between masseter muscle size and obstructive parotid symptoms in a larger cohort. The difference in masseter muscle thickness on the order of 1–1.5 mm, similar to this study, has been associated with different facial profiles and represents the average change in thickness after masseter botulinum injections [12, 13]. These findings suggest that in patients with masseter muscle enlargement and parotid sialadenitis, alterations in masseter size may impact sialadenitis symptoms. The effectiveness of masseter muscle treatment strategies requires further investigation.

The association of masseter enlargement and parotid sialadenitis may be bidirectional. Inflammation of the parotid duct and gland may contribute to muscle hyper-function and hypertrophy. Alternatively, several of the postulated theories of MMH (such as bruxism, mastication, and tooth clenching) may trigger salivary stimulation through the activation of the muscles of mastication and the temporomandibular joint. Salivary secretion is also affected by stress and anxiety [14, 15]. These factors may result in MMH and also concurrently lead to dysfunction of the parotid gland through chronic over-stimulation. The submandibular glands may be less affected by the chronic stimulation from excessive masseter activation. Further study is warranted to evaluate the effect of treatments to address the MMH and bruxism on salivary stimulation and parotid function. Future studies could also evaluate whether the masseter size is a predictor for treatment outcomes after parotid sialendoscopy assisted surgeries. Anatomic relationships between masseter muscle, parotid duct angle, and duct anatomy may be evaluated with traditional and MR sialography, with traditional sialography capable of evaluating for dynamic changes of the flexible ductal system [16]. The differential diagnosis of recurrent parotitis in the absence of stone or stricture warrants an evaluation of the potential extrinsic impact of the masseter muscle along with other difficult-to-diagnose causes such as eosinophilic sialodochitis and juvenile recurrent parotitis.

We acknowledge a number of limitations to our study. Our cohort size is modest, and the study was retrospective. The control group was chosen with submandibular sialadenitis patients to ensure detailed query of parotid versus non-parotid sialadenitis; however, a normative control group without sialadenitis may be more ideal. Future studies could assess the impact of managing masseter hypertrophy in the integrated management of chronic parotid sialadenitis and the utility of ultrasound in measuring masseter size. Finally, our study did not correlate masseter size with symptom severity or capture risk factors for masseter muscle hypertrophy such as temporomandibular joint muscle dysfunction. We also did not correlate sialendoscopy findings to the masseter muscle size, as we found reporting of endoscopic exams to be not consistent at the masseteric bend.

6 | Conclusions

Masseter thickness was significantly larger in patients with obstructive parotid sialadenitis compared to those with submandibular sialadenitis. Our findings demonstrate an association between increased masseter size and obstructive parotid sialadenitis.

Conflicts of Interest

J.L.C. is a consultant for Inspire Medical Systems and Nyxoah, she is also supported by the Veterans Affairs Medical Center, San Francisco, CA. The contents do not represent the views of the U.S. Department of Veterans Affairs or those of the U.S. government. The authors have no other funding, financial relationships, or conflicts of interest to disclose.

References

1. A. Gulati, J. Kidane, and J. L. Chang, "Patient Reported Outcome Measures for Salivary Function: A Systematic Review," *Laryngoscope* 133, no. 8 (2023): 1796–1805, https://doi.org/10.1002/lary.30550.

2. R. Reddy, D. R. White, and M. B. Gillespie, "Obstructive Parotitis Secondary to an Acute Masseteric Bend," *ORL* 74, no. 1 (2012): 12–15, https://doi.org/10.1159/000334246.

3. K. A. Plonowska, E. Ochoa, W. R. Ryan, and J. L. Chang, "Sialendoscopy in Chronic Obstructive Sialadenitis Without Sialolithiasis: A Prospective Cohort Study," *Otolaryngology and Head and Neck Surgery* 164, no. 3 (2021): 595–601, https://doi.org/10.1177/0194599820957256.

4. L. Mandel and F. Surattanont, "Bilateral Parotid Swelling: A Review," *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* 93, no. 3 (2002): 221–237, https://doi.org/10.1067/moe.2002.121163.

5. D. Z. Rispoli, P. M. Camargo, J. L. Pires, V. R. Fonseca, K. K. Mandelli, and M. A. C. Pereira, "Benign Masseter Muscle Hypertrophy," *Brazilian Journal of Otorhinolaryngology* 74, no. 5 (2008): 790–793, https://doi.org/10.1016/S1808-8694(15)31393-8.

6. R. R. Addante, "Masseter Muscle Hypertrophy: Report of Case and Literature Review," *Journal of Oral and Maxillofacial Surgery* 52, no. 11 (1994): 1199–1202, https://doi.org/10.1016/0278-2391(94)90546-0.

7. P. Capaccio, M. Gaffuri, L. Pignataro, F. Assandri, P. Pereira, and G. Farronato, "Recurrent Parotid Swelling Secondary to Masseter Muscle Hypertrophy: A Multidisciplinary Diagnostic and Therapeutic Approach," *Cranio* 34, no. 6 (2016): 388–394, https://doi.org/10.1080/08869634.2016.1142694.

8. P. Capaccio, M. Lazzeroni, F. Lo Russo, et al., "MR Sialographic Assessment of the Masseter Muscle and the Ductal Kinking in Patients With Recurrent Parotitis," *La Radiologia Medica* 129, no. 5 (2024): 785–793, https://doi.org/10.1007/s11547-024-01802-1 Epub 2024 Mar 21. PMID: 38512620; PMCID: PMC11088540.

9. A. P. Reis Durão, A. Morosolli, J. Brown, and R. Jacobs, "Masseter Muscle Measurement Performed by Ultrasound: A Systematic Review,"

Dento Maxillo Facial Radiology 46, no. 6 (2017): 20170052, https://doi.org/10.1259/dmfr.20170052.

10. P. H. Chang, Y. J. Chen, K. V. Chang, W. T. Wu, and L. Özçakar, "Ultrasound Measurements of Superficial and Deep Masticatory Muscles in Various Postures: Reliability and Influencers," *Scientific Reports* 10 (2020): 14357, https://doi.org/10.1038/s41598-020-71378-z.

11. A. C. Dupont, E. E. Sauerbrei, P. V. Fenton, P. C. Shragge, G. E. Loeb, and F. J. R. Richmond, "Real-Time Sonography to Estimate Muscle Thickness: Comparison With MRI and CT," *Journal of Clinical Ultrasound* 29, no. 4 (2001): 230–236, https://doi.org/10.1002/jcu.1025.

12. N. G. Ispir and M. Toraman, "The Relationship of Masseter Muscle Thickness With Face Morphology and Parafunctional Habits: An Ultrasound Study," *Dento Maxillo Facial Radiology* 51, no. 8 (2022): 20220166, https://doi.org/10.1259/dmfr.20220166.

13. D. Erdil, N. Bagis, H. Eren, M. Camgoz, and K. Orhan, "The Evaluation of the Relationship Between Changes in Masseter Muscle Thickness and Tooth Clenching Habits of Bruxism Patients Treated With Botulinum Toxin A," *Journal of Medical Ultrasound* 31, no. 1 (2022): 22–28, https://doi.org/10.4103/jmu.jmu_51_22.

14. N. M. Graham, R. C. Bartholomeusz, N. Taboonpong, and J. T. La Brooy, "Does Anxiety Reduce the Secretion Rate of Secretory IgA in Saliva?," *Medical Journal of Australia* 148, no. 3 (1988): 131–133, https://doi.org/10.5694/j.1326-5377.1988.tb112773.x.

15. D. R. Morse, G. R. Schacterle, J. V. Esposito, M. L. Furst, and K. Bose, "Stress, Relaxation and Saliva: A Follow-Up Study Involving Clinical Endodontic Patients," *Journal of Human Stress* 7, no. 3 (1981): 19–26, https://doi.org/10.1080/0097840X.1981.9936829 PMID: 7276547.

16. P. A. Wenzel, R. K. Thorpe, J. E. Maley, et al., "Stensen's Duct Dynamic Anatomy Assessed With Sialography," *Ear, Nose & Throat Journal* 27 (2024): 1455613241272451, https://doi.org/10.1177/01455613241272451 Epub ahead of print. PMID: 39192625.