Effects of tongue position on mandibular muscle activity and heart rate function

John E. Schmidt, PhD,[a] Charles R. Carlson, PhD,[b] Andrew R. Usery, MD,[c] and
Alexandre S. Quevedo, DDS, PhD,[d] Rochester, MN, Lexington, KY, and Winston-Salem, NC
MAYO CLINIC, UNIVERSITY OF KENTUCKY, AND WAKE FOREST UNIVERSITY

Objectives. A primary goal of pain management for muscle-related pain is to reduce masticatory muscle activity. This study aimed to investigate masticatory muscle group activity and heart rate variability change when the tongue was placed on the palate or the floor of the mouth in a healthy pain-free sample.

Study design. Participants were 23 females and 18 males with a mean age of 19.6 years (standard deviation = 1.5). Muscle activity was measured using surface electromyography and heart period were measured using electrocardiography. The experimental protocol consisted of 3 periods: baseline, tongue placement on the floor of mouth, and tongue placement on palate.

Results. Results indicated significantly more activity in the temporalis and suprahyoid muscle regions as well as a significant reduction in heart rate variability when the tongue was positioned on the palate compared with tongue position on the floor of the mouth.

Conclusions. Instructions to place the tongue on the roof of the mouth are not instructions that will promote reduced physiological functioning (i.e., relaxation) but rather promote small, but potentially important increases in overall activity as indexed by muscle tone and cardiac function. **(Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108:881-888)**

Recently there has been strong interest in developing and applying strategies for health care treatments that have demonstrated effectiveness according to the methods of science. For the management of orofacial pain conditions, this is particularly salient because of the potential for significant iatrogenic consequences to emerge when treatment strategies are applied without evidence from controlled clinical studies.[1,2] One of the more common reversible strategies that dental providers offer to patients with temporomandibular disorders is to monitor and control tongue position as a part of an overall effort to reduce unnecessary parafunctional activity.[3] Generally the goal of these instructions is to minimize muscle activity for pain management. The assumption here is that maintaining a particular "rest" position where muscle activity is minimized will help control muscle overuse, and facilitate reduction in muscle-related pain.

In the dental literature, there are several examples of instruction sets that tell the patient where to place the tongue for achieving a rest position.[4,5] These instructions are typically part of comprehensive treatment approaches, and instruct the patient to place the tongue against the palate with slight pressure.[6,7] However, there is scant evidence that placing the tongue against the palate is indeed a position of "rest" if rest refers to reduction of muscle activity.

There are 2 recent studies that provide data demonstrating increased muscle activity when the tongue is placed against the palate.[8,9] Carlson and colleagues[8] reported data from healthy, pain-free participants that showed significantly higher electromyographic (EMG) activity in the right temporalis and suprahyoid muscles when the tongue was placed against the palate with slight pressure compared with resting the tongue on the floor of the mouth. In a similar investigation, Takahashi and colleagues[9] used an intraoral appliance to evaluate tongue positions and muscle activity. This device included 2 pressure transducers to detect contact with the tongue, as well as the placement of 3 external EMG sensors on the right masseter, right anterior temporalis, and suprahyoid muscles.[9] The results of this study found significant differences between rest and anterior tongue placement for masseter muscle activity, and significant differences between rest and both anterior and superior tongue placements for activity in both the temporalis and suprahyoid muscle groups.

[a]Assistant Professor of Psychology, Department of Psychiatry and Psychology, Department of Anesthesiology, Mayo Clinic.
[b]Professor and Chair, Department of Psychology, University of Kentucky.
[c]Medical Resident, College of Medicine, University of Kentucky.
[d]Research Fellow, Department of Neurobiology, Wake Forest University.
Received for publication Oct 10, 2008; returned for revision Jun 18, 2009; accepted for publication Jun 26, 2009.
1079-2104/$ - see front matter

doi:10.1016/j.tripleo.2009.06.029

Results from these studies provide corroborating evidence that placing the tongue against the roof of the mouth results in increased muscle activity in the suprahyoid and temporalis regions.[8,9] Despite these data-based findings, the question of the appropriate rest position for the tongue remains controversial.[7] For our purposes, we define "rest" as a position in which muscle activity is at a minimum value, as measured by electromyography. The importance of maintaining a rest position is illustrated by a series of studies by Glaros and colleagues[10-12] demonstrating that maintaining small levels of masticatory muscle activity can result in significant pain and dysfunction. These findings support the value of reduced masticatory muscle activity as a goal for orofacial pain management. Furthermore, recent functional magnetic resonance imaging (fMRI) studies have shown that tongue position maintained on the roof of the mouth results in higher cortical activity compared with the tongue resting on the floor of the mouth, and that there are significant differences in areas of cerebral activation associated with tongue position and movement.[13-15]

The fMRI studies showing cortical in addition to midbrain and brainstem control of tongue motor function are consistent with studies showing how reversible cortex cooling extinguished some tongue behaviors while having minimal effect on some evoked chewing/swallowing jaw muscle activity.[13-19] Recent findings that tongue nociception and learned tongue protrusion tasks induce neuroplastic changes in the cortex illustrate how brain control of tongue activity can be modified by environmental stimuli.[20,21] Tongue movements are complex and represent a portion of the highly integrated continuum of behaviors mediated by the cranial nerves, and the hypoglossal motor nuclei, like other cranial nerve motor nuclei, receive neural input from widespread cortical, midbrain, and brainstem regions.[22-25] These regions help process the entire spectrum of sensory input so that the brain can effectively coordinate feeding behaviors, breathing, and speech and modulate cardiovascular and endocrine functions. They also form the central autonomic nervous system and greater limbic system, which contribute to the learning process.[26,27] The therapeutic suggestion to practice elevated or protruded tongue rest positions may instill learned, but functionally irrelevant postures that are assumed when stressors stimulate the brain. Such postures defy gravity, activate muscle metaboreceptors, and, with fatigue summation, produce sympathetic demands that may have adverse effects on orofacial pain patients.

The likelihood that the tongue position on the roof of the mouth results in less muscle activity is thus not supported by available research findings. Further, experimental studies raise the question of whether placement of the tongue against the palate is associated with changes in other physiologic systems beyond the specific musculature and cortical structures associated with tongue activity. If such an effect does occur with tongue activity, habitual placement of the tongue against the palate may contribute to disruption of integrated physiologic system balance. These disruptions could be indexed by measures of autonomic nervous system functioning.

Change in the autonomic nervous system can be quantitatively assessed by examining the change in the beat to beat (NN) interval of an electrocardiogram (ECG). This index of heart rate variability (HRV) represents the heart's ability to respond to normal regulatory impulses that affect heart rhythm.[28,29] For the present study, the time domain index of root mean square of successive differences (RMSSD) of the NN intervals will be used. The RMSSD value is considered to be a strong indicator of parasympathetic activity, often referred to as cardiac vagal tone.[29]

Possible association between change in cardiac vagal tone and tongue position–related muscle activity may help increase understanding of the physiologic reactivity found in chronic orofacial pain patients. This patient population has consistently demonstrated higher reactivity compared with pain-free controls in studies of pain thresholds, emotional and cardiovascular reactivity, psychological distress, fatigue, and sleep dysfunction.[30-32] These characteristics suggest compromised self-regulatory processes are important factors in patients suffering from chronic orofacial pain. Increased knowledge regarding the associations between facial muscle activity and cardiac vagal tone in a healthy pain-free sample will further our understanding of possible mechanisms contributing to and sustaining orofacial pain.

The present study had 2 general aims. The first aim was to investigate activity in masticatory muscle groups when the tongue was placed on the palate or the floor of the mouth in a healthy pain-free sample. The second aim was to investigate cardiac vagal tone response to placing the tongue on either the palate or the floor of the mouth.

Two specific hypotheses were forwarded:

1) It was predicted that positioning the tongue with slight pressure on the palate would involve an increase in temporal, masseter, and suprahyoid muscle activity, compared with measured muscle activity when resting the tongue on the floor of the mouth.[8,9]
2) It was predicted that positioning the tongue with slight pressure on the palate would be associated with decreased cardiac vagal tone (diminished HRV) compared with cardiac vagal tone when the tongue is placed on the floor of the mouth (elevated

HRV). Change in cardiac vagal tone was quantitatively assessed by measuring change in the RMSSD index of HRV.

METHODS

Participants

This study was approved by the University of Kentucky Institutional Review Board and all participants provided written informed consent. Inclusion criteria were as follows: (1) age 18 years or older; (2) no current or past injury or pain in the jaw, mouth, or tongue; (3) no current or past chronic pain condition; (4) no current or past history of hypertension or heart disease; (5) not taking any cardiovascular control medication; (6) no history of asthma or other chronic respiratory conditions; (7) no history of diabetes; (8) not pregnant at time of study participation; (9) before participation, resting blood pressure must meet the following criteria: systolic blood pressure lower than 140 mm Hg, diastolic blood pressure lower than 90 mm Hg.[33] Power analysis to determine sample size was based on previous studies, indicating a sample size of 40 would provide adequate power at a beta of 0.80 and an alpha of 0.05.[3,8,9,34] Study participants were 23 females and 18 males with a mean age of 19.6 years (SD = 1.5), a mean weight of 163.4 pounds (SD = 35.5), and a mean height of 68.4 inches (SD = 3.8). Ethnic distribution of the study sample was as follows: Caucasian (n = 33, 80.5%), African American (n = 4, 9.8%), Hispanic (n = 2, 4.9%), and other (n = 2, 4.9%). All participants received class credit for completion of this study.

Experimental setting

All procedures were conducted in a sound-attenuated room in the Psychophysiology Laboratory at the Department of Psychology of the University of Kentucky. Participants were seated in a cushioned chair with head support. Once informed consent was obtained, all participants completed a health history questionnaire. Physiological recording devices were then attached and checked for accuracy. During all study procedures, participants were asked to sit as quietly and comfortably as possible and to refrain from any unnecessary movements.

Current stage of menstrual cycle

Day of menstrual cycle was recorded for female participants by asking for the last day of their previous period. The menstrual cycle is divided into 4 phases: menstruation (days 1 to 5), proliferative phase (days 6 to 13), ovulation (day 14), and luteal phase (days 15 to 28). Autonomic regulation of the heart fluctuates during the menstrual cycle with HRV being lower in the luteal phase than in the other phases; thus, sympathetic activity is dominant during the luteal phase.[35,36]

Physiological measures

The physiological measures were recorded using the MP150 Biopac data acquisition system (Biopac Systems, Inc., Goleta, CA). The configuration for this study included the electromyographic, carbon dioxide, and electrocardiogram amplifier modules. Electromyographic activity at each muscle site was recorded using Ag/AgCl electrodes with shielded leads connected to an EMG100C electromyographic amplifier module. Module settings were as follows: sampling rate = 100 samples per second, amplifier gain = 1000, low pass filter = 500 Hz, notch interference filter 60 Hz = on. The EMG electrodes were placed on the right masseter, suprahyoid, and right and left temporalis muscles according to procedures described by Cram and Kasman.[37] The raw EMG signal was integrated using the BIOPAC software, and the mean EMG values in microvolts were calculated for each recording period. Breathing rate was recorded by placing a nasal cannula under the participant's nose. The cannula tubing was connected to a CO2100C amplifier module. This module provides a continuous measure of end tidal carbon dioxide level and breathing rate in breaths per minute. Owing to equipment problems, end tidal carbon dioxide data were not available for all participants; therefore, these data are not reported. Breathing rate was successfully recorded for all study participants. Cardiovascular activity was recorded using Ag/AgCl electrodes with shielded leads connected to an ECG100C electrocardiogram amplifier module. Module settings were as follows: sampling rate = 1000 samples per second, amplifier gain = 1000, low-pass filter = 35 Hz, high-pass filter = 0.05 Hz, notch interference 60 Hz = on. The electrodes were placed in the Lead I configuration, with the positive and negative electrodes connected to the inside of the forearms.[38] To calculate the RMSSD heart rate variability index, the ECG signal was first filtered and transformed into NN intervals using the Biopac Acquire system software followed by variability analyses using HRV Analysis Software version 1.1 SP1 by Biomedical Signal Analysis Group, Department of Applied Physics, University of Kuopio, Finland. This software package is a standalone HRV analysis program that provides a variety of HRV indices including time-domain and frequency-domain values. For this study, because of the short time length for each study period (baseline = 5 minutes, tongue position on floor and roof, post = 1 minute for each trial), the time-domain HRV index of RMSSD was reported. The RMSSD HRV index was used to quantify cardiac vagal tone as this technique has proven to be accurate, valid, and clinically useful for short time periods of ECG data.[39,40]

Procedure

Once informed consent and health history were obtained, height and weight were recorded. Blood pressure was assessed using a Paramed 9200 automated blood pressure cuff to ensure resting blood pressures were within study criteria. The participant was seated in a comfortable chair and the physiological recording leads were attached and tested, followed by a 5-minute baseline recording period. During all recording periods, participants were instructed to sit as quietly and comfortably as possible and to refrain from unnecessary movements.

Immediately preceding the baseline recording period, all participants were asked where the tongue was in the mouth. The reported placement was recorded as his or her "normal" tongue position. Participants were told they would be instructed to alternate the position of the tongue between the roof and the floor of the mouth for 1-minute periods. Participants were then instructed to either place the anterior third of the tongue on the roof of the mouth according to modified guidelines explained by Rocabado,[6] or to rest the tongue on the floor of the mouth. The order of tongue placement was randomly assigned, with 19 participants beginning with tongue placement on the roof of the mouth. During all trial periods, participants were asked to keep lips and teeth slightly apart to control for facial positioning and to minimize extraneous movements. All participants alternated tongue position until 2 trials were completed in each position. Instructions for each trial are given in Appendix 1. Upon completion of all trials, the sensors were removed and participants were debriefed and excused from the study.

Analytic strategy

Before completing the statistical analyses, mean physiological activity was calculated by averaging integrated EMG, breathing rate, heart rate, and RMSSD variables for all study trials. These values were averaged for the 2 roof position trials and for the 2 floor position trials, resulting in 1 set of values for each tongue position. These 2 composite trials will be noted as the TF period (tongue position on the floor) and TR period (tongue position on the roof). Overall repeated measures analyses of variance (ANOVAs) were performed on the physiological variables using the 3 trial periods: baseline, TF period, and TR period. Focused contrasts were used to evaluate a priori hypotheses. The advantage of using a repeated measures design with physiological recordings is that each participant acts as his or her own control.[41] This approach controls for other factors that can influence physiological recordings such as skin preparation, age, and site placement.[42] Statistical analyses were completed with the Statistical Package for the Social Sciences, Release 15.0 (SPSS Inc., Chicago, IL). The criterion for statistical significance was set at P less than .05. Effect sizes for hypothesized analyses are reported using Cohen's d.

RESULTS

Comparison of physiological variables based on self-reported normal tongue position

Participants were asked to state tongue position after the baseline recording period. Reported "normal" tongue position was floor ($n = 22$), roof ($n = 12$), and not sure ($n = 7$). Univariate analyses on baseline, floor, and roof EMG variables showed no differences between the self-reported floor and roof groups. No differences were detected between the self-reported floor and roof groups on HR or RMSSD. These preliminary analyses were followed by repeated measures analyses comparing the main effect of study period on physiological variables.

Muscle activity

The overall ANOVA for EMG activity in the right masseter muscle indicated no main effect for period (Wilks' Lambda $(2,39) = 2.35$, $P = .109$). Repeated measures univariate was not completed on these data because of no significant main effect detected for the period. The overall ANOVA was followed by focused contrasts to evaluate the a priori hypothesis that positioning the tongue with slight pressure on the palate would result in an increase in masseter muscle activity. Focused contrasts showed a marginally significant difference between the tongue on the roof (TR) and tongue on the floor (TF) periods with higher muscle activity during the TR period; TR = 0.992 versus TF = 0.855, $P = .078$. EMG and other physiological results are displayed in Table I.

In contrast to the finding with the right masseter muscle, the ANOVA for EMG activity in the suprahyoid muscle was highly significant for the effect of period (Wilks' Lambda $(2,39) = 16.08$, $P < .001$). Repeated measures univariate analyses were significant for suprahyoid muscle activity ($F(2,39) = 17.56$, $P < .001$) across the 3 study periods. As predicted, focused contrasts showed a much higher level of suprahyoid muscle activity in the TR period compared with the TF period; TR = 2.09 versus TF = 1.42, $P < .001$.

The ANOVA for EMG activity in the right temporalis muscle also indicated a significant effect for period (Wilks' Lambda $(2,39) = 7.59$, $P = .002$). Repeated measures univariate analyses were significant for right temporalis muscle activity ($F(2,39) = 8.24$, $P = .001$) across the 3 study periods. As predicted, focused contrasts showed significantly higher muscle activity in the

Table I. Electromyographic activity and breathing rate repeated measures ANOVA

	Baseline	*Floor*	*Roof*	*F (2,39)*	*P*	*Cohen's d*
Right masseter	0.95 (0.54)	0.86 (0.31)	0.99 (0.49)	—	—	0.34
Suprahyoid	1.62 (0.88)	1.42 (0.92)	2.09 (1.12)	17.56	.001	0.65
Right temporalis	1.62 (0.94)	1.18 (0.71)	1.35 (0.67)	8.24	.001	0.25
Left temporalis	1.46 (0.71)	1.13 (0.53)	1.34 (0.65)	6.37	.003	0.36
Breathing rate	15.24 (2.79)	15.47 (2.10)	16.15 (2.20)	4.48	.014	0.32

Standard deviation is denoted within parentheses. Cohen's d notes effect sizes between Floor and Roof conditions only. Repeated measures ANOVA was not completed for the right masseter data because no main effect was detected for the period. EMG values are shown in microvolts. Breathing rate is shown in breaths per minute.
ANOVA, analysis of variance; *EMG,* electromyographic; -, no data for these analyses.

Table II. Heart rate and HRV analyses

	Baseline	*Floor*	*Roof*	*F(2,39)*	*P*	*Cohen's d*
Heart rate	68.44 (11.16)	67.30 (10.80)	66.73 (11.374)	—	—	0.05
RMSSD	71.25 (44.53)	**68.60*** **(40.67)**	63.38 (37.82)	5.49	.006	0.10

Standard deviation is denoted within parentheses. Cohen's d notes effect sizes between Floor and Roof conditions only. Repeated measures ANOVA was not completed for the heart rate data because no main effect was detected for the period. Heart rate is shown in beats per minutes.
ANOVA, analysis of variance; *HRV,* heart rate variability; *RMSSD,* root mean square of successive differences; -, no data for these analyses.
*Bold indicates increase in vagal tone compared with tongue position on roof of mouth.

TR period compared with the TF period; TR = 1.35 versus TF = 1.18, *P* = .001.

The final muscle group tested was the left temporalis muscle, which also indicated a significant effect for period (Wilks' Lambda (2,39) = 8.97, *P* = .001). Repeated measures univariate analyses were significant for left temporalis muscle activity (F(2,39) = 6.37, *P* = .003) across the 3 study periods. As predicted, focused contrasts showed significantly higher muscle activity in the TR period compared with the TF period, TR = 1.34 versus TF = 1.13, *P* = .010.

Breathing rate

The overall ANOVA for breathing rate indicated a significant main effect for period (Wilks' Lambda (2,39) = 4.37, *P* = .02). This was followed by a repeated measures univariate analysis, which was significant for breathing rate (F(2,39) = 4.48, *P* = .014) across the 3 periods. Focused contrasts showed significantly higher breathing rate during the TR period compared with the TF period, TR = 16.15 versus TF = 15.47, *P* = .023.

Heart rate and RMSSD

The overall ANOVA for heart rate was not significant for the main effect for period (Wilks' Lambda (2,39) = 2.62, *P* = .086). As with the right masseter results, repeated measures analyses were not completed because no main effect was detected. The overall ANOVA for RMSSD indicated a main effect for period (Wilks' Lambda (2,39) = 4.15, *P* = .023). The repeated measures univariate analysis was significant for RMSSD (F(2,39) = 5.49, *P* = .006) across the 3 study periods. Focused contrasts to evaluate the a priori hypothesis that tongue position on the roof of the mouth would be associated with lower RMSSD compared with tongue position on the floor of the mouth was significant, TR = 63.39 versus TF = 68.60, *P* = .016. Heart rate and RMSSD values are displayed in Table II.

Current stage of menstrual cycle

Menstrual stage distribution for the female participants was as follows: menstruation (n = 1), proliferative phase (n = 4), ovulation (n = 0), and luteal phase (n = 8). The remaining female participants (n = 10) were taking oral contraceptives. Before the completing the physiological analyses, participants in the luteal phase were compared with the remaining female participants on HR and RMSSD during all study periods. No significant differences were found between the 2 female subgroups on HR or RMSSD, *P*'s greater than .05.

DISCUSSION

The results of the study replicated and extended the findings of 2 previous studies that demonstrated muscle activation of temporalis and suprahyoid groups when tongue position is maintained on the roof of the mouth.[8,9] Furthermore, the results of this study showed that tongue position has a significant effect on heart rate variability in that when the tongue was elevated to the roof of the mouth there was a significant reduction in cardiac vagal tone. This finding suggests that there is integration between tongue activity and heart function shown by the increase in muscle activity corresponding with a measureable change in heart function. It is clear that instructions to place the tongue on the roof of the mouth with slight pressure are not instructions that bring about relaxation (i.e., reduction in sympathetic activity) but rather promote small, but potentially important changes in overall activity as indexed by increased muscle tone and reduced cardiac vagal tone.

Because there is evidence that even small increases in muscle activity for extended periods can result in the development of pain and dysfunction, the importance of allowing the tongue to rest as frequently as possible seems self-evident.[10] The present findings replicate and extend the results of earlier studies demonstrating reduced EMG activity in several muscle groups when the tongue is resting on the floor of the mouth. Therefore, instructions for helping individuals obtain a rest position for muscles associated with the jaw should encourage the assumption of this rest position and not promote a strategy whereby the tongue is placed up against the palate.

There are numerous reports in the literature demonstrating the importance of HRV as an index of self-regulatory control.[43-46] It has been found that higher HRV is associated with higher self-regulatory ability whether after a laboratory stressor or coping with distressing life events.[43,46,47] Conversely, individuals with low HRV appear to have reduced self-regulatory ability after experiencing a stressor, as quantitatively indexed by the sustained reduction in HRV indices during post-stressor laboratory assessment.[43] The difference in HRV among individuals in laboratory assessments does not appear to be attributable to one specific variable, but instead is likely a multifactorial issue. It has been suggested that cognitive (e.g., racing thoughts, rumination) as well as behavioral (e.g., hyperventilation) patterns may contribute to sympathetic activation with the end result being a less adaptive functioning for the autonomic nervous system.[48,49] Thus, for individuals who regularly engage in a cognitive or behavioral process associated with elevated sympathetic activity and reduced cardiac vagal tone, an acute stressor may result in faster, more intense, and more sustained arousal. Findings from the present study suggest maintaining the tongue on the palate with slight pressure may be a contributing factor to increases in physiological activation that were evidenced by the observed decrease in cardiac vagal tone.

Our study design cannot provide direct evidence for the contribution of sympathetic activity to the observed changes in HRV owing to the use of the RMSSD index. This HRV index is considered to be a quantitative index of cardiac vagal tone. Further work in this area should attempt to assess both sympathetic and parasympathetic activity in the HRV spectrum by using frequency domain indices. Our study design used short-term ECG recording, the length of which is not recommended for frequency domain analyses that are required to examine sympathetic and parasympathetic functioning more closely.[29] An additional possible limitation is the use of self-report regarding tongue position. We did not use technical sensors such as the one used by Takahashi et al.[9] to assess actual tongue position, but given the pattern of physiological findings are consistent with their work, it is reasonable to conclude that tongue position was likely consistent with self-reports that were obtained.

The design of the study was intended to minimize the potential role that "normal" rest positions of the tongue might have on muscle activity. We believe that the findings in this study provide a consistent picture of muscle activity increase when tongue position is maintained on the roof of the mouth. Given the attention to rigorous experimental controls in order to rule out alternative interpretations, the present findings are likely a reliable representation of muscle activity in the larger population. Although it is the case that some individuals may report that resting the tongue on the floor of the mouth "doesn't feel relaxed," the evidence is consistent that muscle activity is lowest when such a position is maintained.[8,9] What may be important to focus on with such individuals is the necessary "internal recalibration" that is needed because they have become accustomed to heightened muscle activity as a part of their normal habit patterns. That is why if an individual presents with pain or discomfort in the muscles of the face and the tongue positioned against the palate, one of the first lines of interventions may be to alter tongue position to the floor of the mouth.[3]

In summary, the present study provides evidence that tongue position influences both motor and cardiac vagal activity in small, but significant and potentially important ways. Researchers now have additional opportunities to explore the potentially far-reaching implications of tongue position on self-regulatory functioning. Further, clinicians have an opportunity to teach patients a rest position of the tongue that truly fosters physiological rest. It is hoped that the field of orofacial pain uses this information in the service of patient care that is based on the results of scientific study.

The authors thank the editor and reviewers for their helpful comments on earlier versions of this manuscript. We especially thank Dr. Peter Bertrand, Orofacial Pain Center, Bethesda Naval Hospital, for his assistance in providing information on central nervous system control of tongue activity and its implications for clinical outcomes.

REFERENCES

1. Ozcelik O, Haytac MC, Akkaya M. Iatrogenic trauma to oral tissues. J Periodontol 2005;76:1793-7.
2. Abdel-Fattah RA. Diagnosis and prevention of temporomandibular joint (TMJ) or adontostomatognathic (OSG) injury in dental practice. Todays FDA 1990;2:6C-8C.
3. Carlson CR, Bertrand PM, Ehrlich AD, Maxwell AW, Burton RG. Physical self-regulation training for the management of temporomandibular disorders. J Orofac Pain 2001;15:47-55.
4. Caine A. Beyond chewing. Cranio View 1995;4:33-41.
5. Kotsiomiti E, Kapari D. Resting tongue position and its relation to the state of the dentition: a pilot study. J Oral Rehabil 2000;27:349-54.
6. Rocabado M. Arthrokinematics of the temporomandibular joint. Dent Clin North Am 1983;27:573-94.
7. Fricton JR, Schiffman EL. Managment of masticatory myalgia and arthralgia. In: Sessle BJ, Lavigne GJ, Lund JP, Dubner R, editors. Orofacial pain: from basic science to clinical management. Hanover Park: Quintessence; 2008. p. 179-85.
8. Carlson CR, Sherman JJ, Studts J, Bertrand PM. The effects of tongue position on mandibular muscle activity. J Orofac Pain 1997;11:291-7.
9. Takahashi S, Kuribayashi G, Ono T, Ishiwata Y, Kuroda T. Modulation of masticatory muscle activity by tongue position. Angle Orthod 2005;75:35-9.
10. Glaros AG, Burton E. Parafunctional clenching, pain, and effort in temporomandibular disorders. J Behav Med 2004;27:91-100.
11. Glaros AG, Forbes M, Shanker J, Glass EG. Effect of parafunctional clenching on temporomandibular disorder pain and proprioceptive awareness. Cranio 2000;18:198-204.
12. Glaros AG, Williams K, Lausten L. The role of parafunctions, emotions and stress in predicting facial pain. J Am Dent Assoc 2005;136:451-8.
13. Watanabe J, Sugiura M, Miura N, Watanabe Y, Maeda Y, Matsue Y, et al. The human parietal cortex is involved in spatial processing of tongue movement—an fMRI study. NeuroImage 2004;21:1289-99.
14. Corfield DR, Murphy K, Josephs O, Fink GR, Frackowiak RSJ, Guz A, et al. Cortical and subcortical control of tongue movement in humans: a functional neuroimaging study using fMRI. J Appl Physiol 1999;86:1468-77.
15. Martin RE, MacIntosh BJ, Smith RC, Barr AM, Stevens TK, Gati JS, et al. Cerebral areas processing swallowing and tongue movement are overlapping but distinct: a functional magnetic resonance imaging study. J Neurophysiol 2004;92:2428-43.
16. Murray GM, Sessle BJ. Functional properties of single neurons in the face primary motor cortex of the primate. I. Input and output features of tongue motor cortex. J Neurophysiol 1992;67:747-58.
17. Murray GM, Sessle BJ. Functional properties of single neurons in the face primary motor cortex of the primate. II. Relations with trained orofacial motor behavior. J Neurophysiol 1992;67:759-74.
18. Murray GM, Sessle BJ. Functional properties of single neurons in the face primary motor cortex of the primate. III. Relations with different directions of trained tongue protrusion. J Neurophysiol 1992;67:775-85.
19. Yao D, Yamamura K, Narita N, Martin RE, Murray GM, Sessle BJ. Neuronal activity patterns in primate primary motor cortex related to trained or semiautomatic jaw and tongue movements. J Neurophysiol 2002;87:2531-41.
20. Adachi K, Murray GM, Lee JC, Sessle BJ. Noxious lingual stimulation influences the excitability of the face primary motor cerebral cortex (face MI) in the rat. J Neurophysiol 2008;100:1234-44.
21. Sessle BJ, Adachi K, Avivi-Arber L, Lee J, Nishiura H, Yao D, et al. Neuroplasticity of face primary motor cortex control of orofacial movements. Arch Oral Biol 2007;52:334-7.
22. Fay FA, Norgren R. Identification of rat brainstem multisynaptic connections to the oral motor nuclei using pseudorabies virus, I. Masticatory muscle motor systems. Brain Res Rev 1997;25:255-75.
23. Fay RA, Norgren R. Identification of rat brainstem multisynaptic connections to the oral motor nuclei in the rat using pseudorabies virus, II. Facial muscle motor systems. Brain Res Rev 1997;25:276-90.
24. Fay RA, Norgren R. Identification of rat brainstem multisynaptic connections to the oral motor nuclei using pseudorabies virus, III. Lingual muscle motor systems. Brain Res Rev 1997;25:291-311.
25. Ter Horst GJ, Copray J, Leim R, Van Willigen J. Projections from the rostral parvocellular reticular formation to pontine and medullary nuclei in the rat: involvement in autonomic regulation and orofacial motor control. Neuroscience 1991;40:735-58.
26. Groenewegen HJ, Uylings HBM. The prefrontal cortex and the integration of sensory, limbic and autonomic information. Progr Brain Res 2000;126:3-28.
27. Nieuwenhuys R. The greater limbic system, the emotional motor system and the brain. Progr Brain Res 1996;107:551-80.
28. Akselrod S. Components of heart rate variability: basic studies. In: Malik M, Camm AJ, editors. Heart rate variability. Armonk: Futura Publishing; 1995. p. 147-63.
29. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation 1996;93(5):1043-65.
30. Carlson CR, Reid KI, Curran S, Studts J, Okeson J, Falace D, et al. Psychological and physiological parameters of masticatory muscle pain. Pain 1998;76:297-307.
31. Maixner W, Fillingim R, Booker D, Sigurdsson A. Sensitivity of patients with painful temporomandibular disorders to experimentally evoked pain. Pain 1995;63:341-51.
32. Curran S, Carlson CR, Okeson J. Emotional and physiological responses to laboratory challenges: patients with temporomandibular disorders versus matched control subjects. J Orofac Pain 1996;10:141-50.
33. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. The JNC 7 Report. JAMA 2003;289:2560-71.
34. Lipsey MW. How to estimate statistical power. Design sensitivity: statistical power for experimental research. Newbury Park: Sage; 1990. p. 69-89.
35. Landen M, Wennerblom G, Tygesen H, Modigh K, Sorvik K, Ysander C, et al. Heart rate variability in premenstrual dysphoric disorder. Psychoneuroendocrinol 2004;29:733-40.
36. Sato N, Miyake S, Akatsu J, Kumashiro M. Power spectral analysis of heart rate variability in healthy young women during the normal menstrual cycle. Psychosom Med 1995;57:331-5.
37. Cram JR, Kasman GS. Electrode placements. Introduction to surface electromyography. Gaithersburg (MD): Aspen; 1998. p. 237-384.
38. Guyton AC. Textbook of medical physiology. 8th ed. Philadelphia: W.B. Saunders; 1991. p. 119-23.
39. McNames J, Thong T, Goldstein B. Reliability and accuracy of heart rate variability metrics versus ECG segment duration. Med Biol Eng Comput 2003;44:747-56.
40. Hamilton RM, Mckechnie PS, Macfarlane PW. Can cardiac vagal tone be estimated from a 10-second ECG? Intl J Cardiol 2004;95:109-15.
41. Fridlund AJ, Cacioppo JT. Guidelines for human electromyographic research. Psychophysiol 1986;23:567-89.
42. Lund JP, Widmer CG. An evaluation of the use of surface electromyography in the diagnosis, documentation, and treatment of dental patients. J Craniomandib Disord 1989;3:129-37.

43. Schmidt JE, Carlson CR. A controlled comparison of emotional reactivity and physiological response in chronic orofacial pain patients. J Orofac Pain 2009;23:230-42.
44. Carney RM, Blumenthal JA, Stein PK, Watkins L, Catellier D, Berkman LF, et al. Depression, heart rate variability, and acute myocardial infarction. Circulation 2001;104:2024-8.
45. Thayer JF, Lane RD. A model of neurovisceral integration in emotion regulation and dysregulation. J Affect Disord 2000;61:201-16.
46. Segerstrom SC, Solberg Nes L. Heart rate variability reflects self-regulatory strength, effort, and fatigue. Psychol Sci 2007; 18:275-81.
47. Cohen H, Benjamin J, Geva AB, Matar MA, Kaplan Z, Kotler M. Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. Psychiatry Res 2000;96:1-13.
48. Thayer JF, Lane RD. Perseverative thinking and health: neurovisceral concomitants. Psychol Health 2002;17:685-95.
49. Sullivan GM, Kent JM, Kleber M, Martinez JM, Yeragani VK, Gorman JM. Effects of hyperventilation on heart rate and QT variability in panic disorder pre- and post-treatment. Psychiatry Res 2004;125:29-39.

Reprint requests:

John E. Schmidt, PhD
Mayo Clinic
Anesthesia Research
Joseph Building 4-184W
1216 Second Street SW
Rochester, MN 55902
Schmidt.john1@mayo.edu

APPENDIX 1

Initial instructions:

"I'm going to ask you to move your tongue to the top of your mouth and to the bottom of your mouth. We will alternate position 4 times. Each time will last for 1 minute, and then I will tell you to move your tongue again.

'Up' instructions:

"Begin by placing your tongue against the roof of your mouth, near the top front teeth and make a clicking sound. Now maintain the front third of your tongue against the palate with slight pressure until I tell you to move it, so that the tongue stays comfortable at the roof of the mouth. Also keep your lips and teeth slightly apart so that the mouth stays in a relaxed position, and close your eyes."

'Down' instructions:

"Begin by placing your tongue on the floor of the mouth. Make sure you are not pushing against the back of the teeth. The tongue should stay comfortably on the floor of the mouth. Just let the tip of the tongue 'flop' to the floor of the mouth and let it lie there until I tell you to move it. Also, keep your lips and teeth slightly apart so that the mouth is in a relaxed position, and close your eyes."