Quantifying orofacial muscle stiffness using damped oscillation

By: Nancy Pearl Solomon& **Heather M. Clark**

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ABSTRACT

Muscle stiffness can reflect muscle tone, often presumed to be aberrant in persons with dysarthria. This exploratory study used the Myoton-3 to assess stiffness of the lateral tongue and mid-cheek in 10 participants with various neurologic disorders--primarily lower motor neuron (n = 6), primarily upper motor neuron (n = 4), and neurolsgically normal adults (n = 4). The Myoton delivered a 25-ms pulse perturbation to the surface of the structure of interest and sensed the response with an internal accelerometer. The resulting acceleration curve was used to determine frequency of oscillation and decrement of damping; stiffness was derived from the linear displacement of tissue perforce of the perturbation. Tongue stiffness was significantly lower for the LMN group than for the normal control group, consistent with the assumption that hypotonia accompanies flaccidity. Tongue stiffness did not differ for the UMN group, nor did cheek stiffness, oscillation frequency or decrement differ between any groups. These preliminary findings indicate that stiffness can be determined from the surface of the tongue and cheek, and may be indicative of low muscle tone in LMN lesions. Although methodologic challenges remain, this novel approach has the potential to quantify orofacial muscle stiffness and document potential changes in muscle tone with disease and treatment.

Normal muscle tone reflects a well-functioning neuromuscular system, with balanced central nervous system (CNS) inhibition over peripheral nervous system (PNS) reflexes and tonic activity. Abnormal muscle tone underlies motor dysfunction, such that PNS impairment interrupts normal reflex loops, and CNS impairment releases inhibition to lower motor neuron pools. Thus, hypotonia accompanies flaccidity, and hypertonia accompanies spasticity. Disorders of the basal ganglia and cerebellar control circuits are associated with disrupted muscle tone as well (hypotonia in ataxia, hypertonia in hypokinesia, and hypertonia or variable tone in hyperkinesia). Disordered muscle tone is thought to underlie many types of dysarthria (Darley, Aronson, & Brown, 1975; Duffy, 2005), yet empirical data addressing the influence of muscle tone on speech production are largely lacking.

Clinical methods for assessing muscle tone in the orofacial musculature are rarely used. Only two clinical assessments for muscle tone exist for clinical use in speech-language pathology, to our knowledge. Both are subjective in nature, and neither is accompanied by supporting data (Beckman, 1988; Dworkin & Culatta, 1996). Generally, they involve passively stretching or palpating the structure of interest and rating whether resistance to the perturbation is lower or higher than normal.

Tone is operationally defined as passive resistance to stretch or palpation, and can be quantified as stiffness. Two instruments, currently only available for research applications in the United States, have been applied to the assessment of orofacial stiffness. One, the OroSTIFF, was designed to assess stiffness of the perioral structures (tissues around the mouth opening, including the orbicularis oris muscles) (Chu, Barlow, Kieweg, & Lee, 2010). A pneumatically operated scissor cantilever stretches the corners of the relaxed mouth laterally, and dynamic stiffness is calculated as the change in force across a series of displacements. Preliminary data support the reliability and validity of the assessment for neurologically normal adults, and the presence of increased stiffness (rigidity) in an adult with Parkinson's disease.

A second instrument, the Myoton, operates according to a damped oscillation model. The Myoton delivers a brief pulse perturbation with a thin probe to the surface of the structure of interest, and senses the resulting oscillation with an accelerometer (Vain, 1995). Veldi et al. (Veldi, Vasar, Hion, Kull, & Vain, 2001; Veldi, Vasar, Hion, Vain, & Kull, 2002; Veldi, Vasar, Vain, & Kull, 2004) used the Myoton-2 to generally reveal increased stiffness and decreased elasticity of the tongue and soft palate in middle-aged adults with obstructive sleep apnea compared to adults with no symptoms (i.e., non-snorers). Stiffness was inferred from the frequency of oscillation (in Hz) in response to an 8-ms pulse perturbation, and elasticity was inferred from the logarithmic decrement of damped oscillation; both were calculated from the acceleration curve. Although the current Myoton output includes direct stiffness values (in N/m), these were not reported in the studies by Veldi et al.

This preliminary study explores the utility of the Myoton for assessing orofacial muscle tone in persons with neurological disorders that can result in dysarthria or dysphagia. Purposes of this investigation were to establish standard procedures for the assessment of lateral tongue and cheek stiffness in persons with normal and disordered neurologic systems. Specifically, we sought to demonstrate that measures of tissue stiffness could be obtained and reliably repeated

from the tongue and cheeks of neurologically normal individuals. Further, we explored tongue and cheek stiffness in persons with upper motor neuron (UMN) or lower motor neuron (LMN) lesions to examine whether resting muscle stiffness varies across disorders as predicted. Finally, in participants with unilateral impairments, we explored differences in tissue stiffness between the weak and normal sides. In persons with LMN damage, we expected to find lower stiffness on the affected side, and in cases of UMN lesions, we expected higher stiffness on the affected side.

METHOD

Participants

Thirteen men with a variety of neurologic impairments and nine neurologically normal adults originally enrolled in this exploratory study. Disordered participants were recruited after referral to the Speech Pathology Clinic for speech and/or swallowing evaluation at Walter Reed Army Medical Center (WRAMC). During data collection and reduction, data were screened for accuracy and eliminated if they failed to meet measurement criteria. Table 1 lists demographic information, etiologies, and primary site of lesion for the 10 disordered participants who ultimately contributed data to this study. Of the nine originally enrolled neurologically normal participants, two men and two women (ages 22-35, M = 29.8 years) provided adequate valid data. One of the neurologically normal women returned for four sessions to assess day-to-day variability. All participants provided informed consent according to the rules and regulations for human-subjects research at WRAMC.

TABLE 1. Demographic and Diagnostic Information for Participants with Neurological Disorders.

Participant	Age	Weak Side & Structure	Etiology
LMN 1	43	bilateral tongue	fibrous dysplagia
LMN 2	42	bilateral tongue	blast injury
LMN 3	23	left tongue	gun-shot wound
LMN 4	39	left tongue	blast injury
LMN 5	72	left face	tumor
LMN 6	24	right lip	blast, shrapnel to neck
UMN 1	44	right lower face	left stroke
UMN 2	21	generalized	severe TBI (motor-vehicle accident)
UMN 3	31	generalized	demyelinating disease
UMN 4	48	generalized	demyelinating disease

Instrumentation and Procedures

The Myoton-3 (V6.7, 2005; Mtiomeetria, Estonia, EU) was outfitted with a 7-cm cylindrical probe (5- or 3-mm diameter) with a flat end. With the participant lying comfortably on his or her side, a tongue blade was positioned under the relaxed tongue or cheek such that the blade was parallel to ground (Figure 1). The probe was lowered slowly onto the surface of the tissue, perpendicular to ground, until the instrument triggered a pulse perturbation (25-ms tap time). If the resulting acceleration curve failed to meet measurement parameters (at least three positive phases), an internal algorithm returned an error message. The procedure was repeated until five error-free trials were obtained for each tissue site.

Data Reduction and Analysis

The Myoton generated an acceleration curve and numeric results for frequency, decrement, and stiffness for each trial. All curves were visually examined for integrity and were eliminated from further analysis if they contained any perturbations or irregularities that could affect the validity of the calculated results. Thus, the results were based on curves that resembled damped sinusoids, consistent with measurement theory. Nonparametric statistics evaluated differences between groups and across structures (tongue, cheek). Because of the exploratory nature of this study, a was set at 0.10.

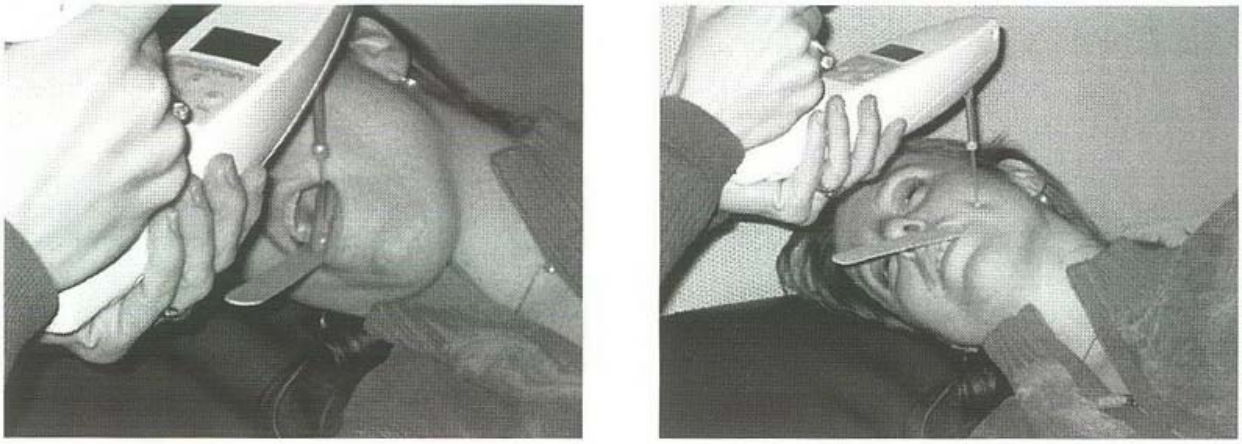

Figure 1. Positioning of Participant and Myoton-3 for Assessment of Lateral Tongue and Mid-Cheek Stiffness During Relaxation; Structural Support is Provided by a Tongue Blade

RESULTS

Table 2 lists summary statistics for the myometric variables (stiffness, frequency, decrement) for 6 LMN, 4 UMN, and 4 normal participants. Stiffness differed significantly across groups for the tongue [[x.sup.2] (2) = 5.51, p = .063]. Follow-up testing (Wilcoxon Rank-Sum) revealed lower tongue stiffness for the LMN than normal groups (W = 21.0, p = .011); no other comparisons differed significantly. No significant differences were found across groups for the cheek, nor for

frequency and decrement for either structure. Wilcoxon Signed-Rank tests revealed that, compared to the tongue, the cheek was significantly stiffer (Z = -3.18, p = .001), oscillated at a higher frequency (Z = -2.34, p = .019), and had a smaller decrement (Z = -3.18, p = .001). No group by structure interactions met criterion for significance.

TABLE 2. Mean (and Standard Deviation) Myometric Variables for Neurologically Normal Participants (n = 4), and for Participants with Primarily Lower Motor Neuron (LMN; n = 6) or Upper Motor Neuron (UMN; n = 4) lesions.

Stiffness (N/m)								
Group	*Tongue Right*		*Tongue Left*		*Cheek Right*		*Cheek Left*	
Normal	224.5	(17.5)	197.8	(33.7)	248.0	(35.1)	258.8	(72.3)
LMN	177.3	(10.0)	185.3	(9.4)	235.0	(33.7)	243.8	(25.2)
UMN	201.6	(23.9)	206.2	(49.5)	240.6	(18.1)	253.4	(45.2)
Frequency (Hz)								
Group	*Tongue Right*		*Tongue Left*		*Cheek Right*		*Cheek Left*	
Normal	16.7	(2.0)	15.9	(2.3)	18.3	(0.6)	15.7	(3.2)
LMN	13.9	(1.8)	13.8	(1.9)	16.4	(3.3)	15.7	(2.1)
UMN	13.7	(0.5)	15.3	(1.4)	15.2	(2.1)	15.3	(2.3)
Decrement								
Group	*Tongue Right*		*Tongue Left*		*Cheek Right*		*Cheek Left*	
Normal	1.5	(0.2)	1.2	(0.3)	1.1	(0.2)	1.2	(0.3)
LMN	1.3	(0.2)	1.5	(0.3)	1.3	(0.2)	1.1	(0.1)
UMN	1.5	(0.2)	1.5	(0.1)	1.0	(0.1)	1.1	(0.1)

Repeated assessments for one neurologically normal participant revealed relatively steady results across 4 days. The percentage difference between each individual measure and the mean for that structure was, on average (and min-max), 4.3% (0.3%-10.7%) for stiffness, 4.4% (0.6%-10.7%) for frequency, and 11.5% (3.1%-25.7%) for decrement.

Five neurologically disordered participants had unilateral impairment of either the tongue or cheek. Stiffness was greater on the affected side for four, despite two being related to LMN and two UMN lesions. All differences were within the range of asymmetry seen in normal participants.

DISCUSSION

Myometric assessment of the tongue and cheek was accomplished in selected individuals with LMN, UMN, and no neurologic disorders. Overall, the cheeks were found to be stiffer than the tongue, consistent with the findings of increased frequency and decreased decrement

(interpreted as elasticity). This finding was expected because the cheek is thin compared to the tongue bulk, leaving less tissue to respond to the perturbation.

The finding that the tongue's stiffness was lower for the participants with LMN impairments than for UMN or no impairments is consistent with the prediction that LMN damage leads to flaccidity With this small number of participants, however, this observation must be made with extreme caution. Nonetheless, it is promising in that it suggests the clinical applicability to objectively document disordered muscle stiffness in the tongue. That increased stiffness in participants with UMN damage was not found, however, leaves the notion of spasticity of the tongue with UMN lesions unanswered. Tongue asymmetry was only testable for two participants with LMN lesions, and surprisingly, tongue stiffness was greater on the affected side for one and not asymmetric for the other. This is contrary to what the group results would indicate. Three participants presented clinically with facial asymmetry (1 LMN, 2 UMN). All three tested as having stiffer cheeks on the affected side. Again, these data must be interpreted cautiously because of the small sample size and because the neurologically normal participants also demonstrated asymmetry for stiffness of the tongue and cheeks.

The Myoton is based on a damped-oscillation model, such that frequency and decrement are determined based on the second wave of the acceleration curve resulting from a brief mechanical pulse. Trials are analyzed from three cycles of the acceleration curve. During protocol development, we determined that increasing the duration of the pulse increased the chance of obtaining the required number of cycles for these soft, thin structures. Therefore, although Veldi et al. used an 8-ms pulse and the Myoton's default is 15 ms, this study used a 25-ms pulse. The longer tap duration allowed for less error-prone data collection, but appears to have contributed to problems noted during subsequent examination of the acceleration curves. In 2009, the company changed the Myoton's internal algorithm (V3.23.4) to require only two acceleration peaks. This new configuration allows for easier data collection, and has led us to reduce tap duration from 25 to 8-10 ms. With this new method, tissue perturbation is cleaner and more specific.

Another important issue to consider is the potential contribution of cutaneous reflexes to stiffness measures. When the tongue or face is tapped, a reflex occurs at that site. The latency of this reflex is approximately 17 ms for the lower lip and 31 ms for the tongue (Weber & Smith, 1987). These latencies are shorter than or close to the timing of the first acceleration peak of the Myoton response. EMG monitoring during the pulse perturbation is needed to confirm that muscles are relaxed during testing and to determine the extent to which perioral and lingual reflexes contribute to overall tissue stiffness.

This exploratory study revealed that myometric assessment procedures are well tolerated by neurologically normal and disordered adults, but that obtaining analyzable trials was sometimes difficult, especially for certain persons. This study should be replicated and extended with the Myoton's new data-reduction algorithm, which appears to be more successful and appropriate, especially for pliable and thin muscles like those of the tongue and cheek. The Myoton appears to have potential utility for quantifying stiffness in the orofacial system, but careful evaluation of

data-collection parameters is needed to reduce analysis errors and variability, and to improve data interpretation.

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