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Indirect Estimates of Jaw Muscle Tension in Children With Suspected Hypertonia, Children With Suspected Hypotonia, and Matched Controls

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Abstract

Purpose—In this study, the authors compared indirect estimates of jaw-muscle tension in children with suspected muscle-tone abnormalities with age- and gender-matched controls.

Method—Jaw movement and muscle activation were measured in children (ages 3 years, 11 months, to 10 years) with suspected muscle-tone abnormalities (Down syndrome or spastic cerebral palsy; $n = 10$) and controls ($n = 11$). Two measures were used to infer jaw tension: a kinematic index of mass-normalized stiffness and electromechanical delay (EMD). The kinematic index used video-based kinematics to obtain the slope of the peak velocity-displacement relationship. The EMD was derived from the interval between the onset of suprahyoid muscle activity and the onset of jaw depression.

Results—Neither measure differentiated the groups. The kinematic index revealed differences between stressed and unstressed syllables in 3-syllable productions by the participants with cerebral palsy and controls, but not in 2-syllable productions by the participants with Down syndrome and controls.

Conclusion—This preliminary investigation included the novel application of 2 measures to infer the jaw-muscle tension of children with suspected tone abnormalities. Although the results do not support the hypothesis that suspected muscle-tone abnormalities affect jaw movement sufficiently to influence speech production, considerations for interpreting the findings include methodological limitations and possible compensatory muscle coactivation.

Keywords

cerebral palsy; Down syndrome; children; speech disorders

Low-level tonic agonist–antagonist muscle activity balanced against intrinsic muscle properties (e.g., elasticity) and joint properties is believed to maintain muscle length and tension in a state of movement readiness. Although the level of tonic activity required to maintain this normal level of muscle tension (i.e., muscle tone) has not been quantified, individuals presenting with diminished (hypotonia) or excessive (hypertonia) tone are readily identified and distinguished during routine clinical examination. Despite its prominence in virtually any clinical evaluation of neuromuscular integrity, however, a standard measure of muscle tone has not been widely adopted. The valid and reliable quantification of muscle tone is critical to answering basic theoretical and experimental questions that persist with respect to disorders characterized by abnormal tone: What is the

relationship of abnormal muscle tone to functional movement? Does abnormal muscle tone affect different systems (e.g., the limbs vs. oral articulators) by similar or shared mechanisms and to similar degrees? What are the ranges of normal/abnormal muscle tone for various motor systems?

Our understanding of abnormal tone and movement disorders is essential to advances in theoretically based treatment. Patients are categorized, treatment is planned, and progress is measured according to ratings of muscle tone (Pomeroy et al., 2000). Even more to the point, clinical treatments of motor speech disorders may focus on the normalization of muscle tone (see Clark, 2003; Ruscello, 2008), despite the fact that the nature and degree of muscle-tone abnormalities are indeterminate (Abbs, Hunker, & Barlow, 1983) and the impact of abnormal muscle tone of the oral articulators on speech production is unclear.

Recently, techniques to measure stiffness in the oral articulators, including the lips (Chu, Barlow, Kieweg, & Lee, 2010), cheek, and tongue (Solomon & Clark, 2010), have been reported. Thus far, these techniques have been restricted to the measurement of articulatory stiffness at rest in adult populations, although Chu and colleagues (2010) reported the future application of their technique to difficult-to-test populations, such as children, and during speech production.

Potential Measures of Muscle Tension

Because, to date, direct measurement of muscle tone in pediatric populations remains impractical, the current investigation used two physiologic measures to indirectly estimate jaw-muscle tension: the kinematic index and electromechanical delay (EMD). Although these measures are not new, their application for the express purpose of measurement of muscle tension in the oral articulators of children is novel.

Kinematic index

One indirect estimate of muscle tension in the speech musculature is the *kinematic index of mass-normalized stiffness* (Munhall, Ostry, & Parush, 1985; Ostry, Feltham, & Munhall, 1984; Ostry, Keller, & Parush, 1983; Ostry & Munhall, 1985). This measure is based on the model in which the limbs (Cooke, 1980) and oral articulators (Ostry et al., 1984; Ostry et al., 1983; Ostry & Munhall, 1985; Perkell & Zandipour, 2002; Perkell, Zandipour, Matthies, & Lane, 2002; van Lieshout, Bose, Square, & Steele, 2007; Vatikiotis-Bateson & Kelso, 1993) are described as mass-spring systems. This model has been contested (Burdet, Osu, Franklin, Milner, & Kawato, 2001; Franklin et al., 2007; Fuchs, Perrier, Hartinger, 2011; Gomi & Honda, 2002), but without disputing or supporting the applicability of the mass-spring model, this measure can be used as an indicator of a musculoskeletal system's mechanical state. Specifically, the slope of the linear regression of maximum velocity to movement amplitude has been very usefully and broadly applied as a kinematic index of mass-normalized stiffness. This approach to musculoskeletal tension associated with movement yields higher stiffness values for shorter duration movements with greater peak velocity/movement amplitude ratios (Ostry & Munhall, 1985; Perkell et al., 2002; Perkell & Zandipour, 2002; van Lieshout et al., 2007). Somewhat remarkably, linguistic structure is related to the slope of the relationship between displacement and maximum velocity of the oral articulators, which has shown that changes in this measure are related to prosodic structure. Greater stiffness (i.e., greater peak velocity/movement amplitude ratio) has been associated with unstressed syllables in investigations of the tongue (Ostry et al., 1984; Ostry et al., 1983) and lower lip and jaw (Jancke, Bauer, Kaiser, & Kalveram, 1997; Vatikiotis-Bateson & Kelso, 1993). Although this stress-stiffness relationship has been observed in the tongue for children 6 years of age and older (Ostry et al., 1984), it is not clear whether this relationship is consistent in the jaw for children.

EMD

A second potential independent and cross-validating measure of muscle tension and muscle tone is the *EMD*, defined as the interval between the onset or change in muscle activation and the onset of movement (Winter & Brookes, 1991) or force generation (Granata, Ikeda, & Abel, 2000). EMD may comprise several processes: excitation–contraction coupling, contraction of the contractile component (Asai & Aoki, 1996), and stretching of the series elastic components of the muscle (Asai & Aoki, 1996; Winter & Brookes, 1991). The stretching of the series elastic component is modeled as the primary factor underlying the EMD, because the relatively inactive and nontensed muscle/tendon system takes time to “take up the slack” prior to the exterior generation of force and/or movement (Nordez et al., 2009). Described as an index of musculotendinous stiffness (Mora, Quinteiro-Blondin, & Pérot, 2003), the EMD has been used to distinguish muscle tension in children with spasticity from controls with typical development on a knee-flexion task (Granata et al., 2000).

Disorders of Suspected Abnormalities of Muscle Tone

Two disorders frequently associated with excessive and reduced muscle-tone abnormalities are spasticity secondary to cerebral palsy (CP) and Down syndrome (DS). Spasticity, the most common form of hypertonia, is associated with damage to the upper motor neuron, brain stem, or spinal cord (Ronan & Gold, 2007) and is most commonly caused by CP (Delgado et al., 2010). Children with dysarthria associated with spastic CP exhibit a prominent constellation of speech characteristics: hypernasality, breathy voice, voice quality change, inappropriate voice stoppage/release, inappropriate phrasing, dysrhythmia, monoloudness, reduced stress, and slowed rate (Workinger & Kent, 1991).

On the other hand, the characterization of DS includes hypotonia as the most frequently implicated motor deficiency (Davis & Kelso, 1982); nearly all infants with DS exhibit hypotonia at birth (Cooley & Graham, 1991). Typical of the dysarthric speech associated with DS are consistent speech sound production impairments and reduced intelligibility (Bunton, Leddy, & Miller, 2007; Miller & Leddy, 1998), which arise from anomalous cognitive, auditory, structural, and phonological development, hypotonia (Stoel-Gammon, 1997), cerebellar anomalies, and fine-motor coordination deficits (Leddy, 1999). In addition to segmental impairment, likely contributors to this reduced intelligibility include atypical suprasegmental features associated with fundamental frequency modulation, fluency, phrasing, speech rate, and placement of sentential stress (Stoel-Gammon, 1997).

Purpose of the Study

The relationship between abnormal muscle tone and functional movement in motor disorders remains a question of great interest and effort. With regard to orofacial structures, it is not clear that abnormal muscle tension parallels the effects in limb structures or that it affects speech production, despite the fact that techniques aimed at normalizing muscle tone are used clinically. In the present investigation, we sought to employ a novel approach to provide preliminary data to inform (a) whether children with suspected muscle-tone abnormalities demonstrate muscle tension differences in their orofacial structures and (b) whether differences in muscle tension are associated with aberrant speech production. These questions were addressed experimentally using the following design:

1. Jaw-muscle tension was indirectly measured (i.e., using both the kinematic index and EMD) and compared across three groups of speakers: children with suspected hypertonia (CP group), children with suspected hypotonia (DS group), and age- and gender-matched controls.

2. The degree and modulation of estimated jaw muscle tension (i.e., the kinematic index) were compared across production of stressed and unstressed syllables.

Method

Participants

Twenty-two children participated in this investigation, although the data from one participant were not usable and therefore not reported. The participants' groups and ages are shown in Table 1. The final data set included samples from seven children diagnosed with DS (suspected hypotonicity), three children diagnosed with spastic CP (suspected hypertonicity), and 11 age- and gender-matched control participants. All participants fell within the age range of 3;11 (years;months) to 10;0. In addition to the experimental and control groups, one adult without motor speech disorder (a 35-year-old woman) completed the experimental tasks to demonstrate the feasibility of the experimental measures and illustrate the adult model. Participants were seen for a single experimental session, which ranged from 1 to 1.5 hr. All recruitment and experimental procedures were approved by the University of Washington Division of Human Subjects. Control participants were recruited through the Human Subjects Recruitment Research Core at the University of Washington (P30 DC04661).

Participant inclusion criteria included the ability to follow simple directions, hearing reportedly within normal limits and sufficient to complete the experimental tasks, and no reported or observable jaw abnormalities (e.g., symptoms of temporomandibular dysfunction, such as clicking or popping noises or complaints of jaw pain).

Age- and gender-matched control participants—Each control participant was gender and age matched (to within 6 months) to an experiment participant. Based on caregiver report and experimenter observation, control participants had negative histories of neurological, neuromuscular, speech, language, and/or developmental deficits.

Experiment participants—The participants with DS and spastic CP were recruited through speech-language clinicians likely to treat children with motor speech disorders. Additional participants with DS were recruited through the Down Syndrome Subject Pool at the Center for Human Development and Disability at the University of Washington. Inclusion in one of the experiment groups required the presence of either spastic CP or DS, moderate to severe dysarthria, and indications of possible hyper- or hypotonicity affecting the oral/speech mechanism. The presence of either spastic CP or DS was ascertained by parent report of a medical diagnosis and the experimenter observation of characteristics consistent with these disorders and concomitant speech disorders. Structure-function and motor speech examinations were conducted by the first author, a certified speech-language pathologist.

For DS, inclusionary characteristics included physical appearance consistent with DS, parental report of atypical oral-motor development, and structural-function and motor speech exams revealing at least one of the following: the absence or reduction of reflexes, weakness of structures, and slow maximum syllable repetition rates (Brown-Sweeney & Smith, 1997). For spastic CP, inclusionary characteristics included movement abnormalities (e.g., limb contracture, paresis, or scissor gait), oral motor characteristics including weakness or limited range of motion of articulators, and at least one of the following: decreased range and slow nonspeech alternating motion rates and the presence of abnormal reflexes. Individuals were excluded if other neuromuscular disorders were reported or suspected.

Confirmation of suspected hypo- or hypertonicity was obtained through the judgments of two speech-language pathologists with expertise in childhood motor disorders (i.e., a doctoral degree in speech and hearing sciences; academic responsibilities including teaching and research related to motor speech disorders in children). The expert judges were provided with a written summary of the parent report of each child's history, medical diagnosis, and observations and a variety of audio clips for each experiment participant, including the intelligibility test, samples of the experimental tasks, samples from the structural-functional examination, and spontaneous speech samples. The experts rated the presence of hypotonia/hypertonia "likely affecting the child's oral motor functioning and speech performance" on a 7-point scale. Based on mean ratings of the two experts and experimenter, experimental participants received mean ratings ranging from mild to moderate hypo- (DS group) or hypertonia (CP group).

The presence of dysarthria was established by the experimenter through a brief nonstandard/noncommercial structural-functional examination and motor speech examinations derived from established protocols (e.g., Robbins & Klee, 1987), parent report, and clinical observation. The severity of dysarthria was determined by the judgments of the experimenter, the two experienced speech-language pathologists with expertise in childhood motor speech disorders, and nonstandardized intelligibility testing. Intelligibility testing included the production of words and phrases elicited from pictures from the Index of Augmented Speech Comprehensibility in Children (Dowden, 1997) and from Webber Articulation Cards by Super Duper Publications (see www.superduperinc.com/products/view.aspx?stid=348#.UPb7vCc71XE) presented on a computer. These productions were subsequently transcribed by unfamiliar listeners (speech-language pathology graduate students), following a brief training and familiarization with the task. An intelligibility score based on the transcriptions of three transcribers was calculated for each participant. Interjudge reliability across the listeners was .95 (Cronbach's alpha). A rating of severity of the speech disorder was obtained from the mean score on a 9-point scale provided by the expert judges and the experimenter.

Table 2 presents the characteristics of the participants with DS and CP. The summary includes parent report of the participant's medical/behavioral background and history of feeding/oral motor difficulties, results of the structural-function exam conducted by the experimenter, and intelligibility ratings. Ratings revealed that each participant with DS exhibited mild-to-moderate hypotonicity, 0%–10% intelligibility ratings, and severe-to-profound speech disorder. The participants with CP presented with mild-to-moderate hypertonicity. Their intelligibility ranged from 30%–64%, although one child (CP1) with severe speech impairment did not comply with intelligibility testing. CP1 and CP2 were judged to present with severe-to-profound speech disorder, and CP3 was judged to present with a moderate-to-severe speech disorder. The control group had intelligibility scores ranging from 78%, for one of the younger participants, to 100%, with a mean intelligibility score of 96%.

Procedure

Data acquisition of physiologic measures—Each child was accompanied into the testing booth by a caregiver and was seated upright in a chair. Physiologic data collection followed a period of familiarization for the child and the caregiver, and application of electrodes and transducers. Data streams included the acoustic signal, video capture of jaw kinematics, and mandibular electromyography (EMG). The participants' audio recordings were obtained using either a miniature, omnidirectional microphone (Sony ECM-77B) attached to the participant's forehead or a dynamic omnidirectional handheld microphone (Electro-Voice 635A) and coupled to a digital audio recorder (Panasonic SV-3800). The

audio signal was filtered ($f_{\text{lowpass}} = 5000 \text{ Hz}$, $f_{\text{highpass}} = 350$) and digitized at 10,989 samples per second.

Jaw kinematics—Mandibular position was tracked using an infrared camera (Burle TC351A) in the sagittal (profile) view and recorded using a video recorder (Panasonic AG-1980). One reflective marker (~2 mm) was placed on the chin, at the inferior-most midsagittal point over the mandible; another was inferior to the angle of the mouth; and two reference markers were placed in a vertical line on the ear, allowing for head movement correction by aligning the axis to the line defined by these markers.

Cartesian coordinates for each marker were extracted automatically from the videorecordings using a commercially available computer-based movement tracking system (Motus Version 6.0, Peak Performance). The inferior-superior and anterior-posterior positions of the jaw were sampled at 60 samples per second and displacement signals were digitally filtered ($f_{\text{lowpass}} = 15 \text{ Hz}$) in MATLAB using a zero-phase-shift digital filter (Butterworth, 8 pole). The accuracy of the movement tracking system has been assessed to be better than 0.1 mm (see Green, Moore, Higashikawa, & Steeve, 2000).

EMG—EMG signals were recorded using bipolar placement of miniature Ag–AgCl surface electrodes (Kendall KittyCat Pre-Wired Electrodes) coupled to a Grass Model 15 Neurodata Amplifier System. Activity of the jaw-elevating muscles was recorded from the following sites: (a) right masseter, (b) left masseter, (c) right temporalis, and (d) left temporalis. The activity of the jaw-depressing muscles (Koolstra, 2002) was recorded from electrode placement over the suprahyoid muscles. A single forehead ground was used. Electrodes were placed to span the main mass of each muscle lengthwise, with intrapair spacing of approximately 2 cm. EMG records were digitized online at 10,989 samples per second; oversampling of EMG signals was necessary to capture and coregister the audio signal at an adequate rate. All signals were low-pass filtered ($f_{\text{lowpass}} = 1000 \text{ Hz}$) with digital antialiasing filters (Alligator Technologies, Inc.) and amplified with gain factors of 10,000 to 50,000 and bandpass settings of 30–1000 Hz (Grass Model 15 Neurodata Amplifier System/Telefactor). Analog-to-digital conversion used 14-bit sampling (Windaq Version 2.54, 14-bit maximum resolution, Dataq Instruments).

Tasks Analyses

Kinematic index task—The tokens subjected to measurements using the kinematic index included productions of syllable strings (“MAmama”) with first-syllable stress. An attempt was made to elicit a minimum of 20 productions from each participant. To provide a consistent model across participants, each repetition was prompted by the same adult model presented by computer. Three participants did not respond to the computer model, so productions were elicited using a live model provided by the caregiver or experimenter.

Seven of the experiment participants (six with DS and one with CP) produced between zero and 10 three-syllable tokens. In these cases two-syllable productions were accepted. When possible, the age- and gender-matched control participant also produced two-syllable productions, using tokens from his/her experiment match as a model.

Perceptual judgment of stress—Before comparisons of the stress tokens were made within and across participants, these tokens (e.g., “MAmama”) were judged perceptually for stress placement to evaluate task validity. Individual tokens were extracted from audio recording, randomized across participants, and recorded to a CD for presentation to listeners. Five judges independently rated stress, specifically circling their response to the question “Which syllable is stressed or prominent?” following a brief training session. Twenty

percent of the tokens were repeated to obtain a measure of intrajudge reliability. To ensure the highest intrajudge reliability, the judgments from the four listeners with the highest agreement were used for the analysis. Intrajudge reliability of the four listeners ranged from 83% to 94% ($M = 87\%$, $SD = 5\%$). The tokens were labeled with the stress agreed to by at least three of the four judges. Those tokens that did not elicit at least 75% interjudge agreement were excluded from further analysis.

Kinematic measures—Jaw displacement, velocity, and duration of the opening gesture were extracted from the inferior-superior and anterior-posterior kinematics of the mandible. The *onset of jaw depression* was operationally defined as the point at or following the zero-crossing (of the inferior-superior movement trace) that was followed by at least three consecutive descending points. Displacement was calculated from the superior-inferior and anterior-posterior axes using a custom routine in MATLAB. Peak velocity was obtained from the differential of the displacement signal.

The slope (m) of the linear regression equation ($y = mx + b$) of the scatter plot of peak velocity by displacement has been used to infer stiffness, with greater stiffness indicated by a greater slope (Cooke, 1980; Ostry et al., 1983; Ostry & Munhall, 1985). For example, Figure 1 illustrates the slopes of the peak velocity/displacement relationship of multiple productions of stressed (white circles) and unstressed (black squares) syllables by one participant with CP and his matched control. The linear regression equations for each token type are included on the figure. The greater slope of the regression lines for the unstressed

tokens ($14.39 \frac{cm/s}{cm}$ and $18.93 \frac{cm/s}{cm}$ for the participant with CP and the matched control, respectively) relative to the slope of the stressed regression line ($9.25 \frac{cm/s}{cm}$ and $6.69 \frac{cm/s}{cm}$ for participant with CP and the matched control, respectively) are consistent with greater mandibular tension during production of the unstressed tokens.

EMD and muscle-activation measures—For two participants, EMG measurements were noisy and therefore not included in the analysis. Therefore, the data for the EMD and muscle-activation measures included those tokens produced by 19 of the 21 participants. Two tasks were used to measure the EMD and muscle-activation patterns; the participant was asked to open his or her mouth as wide as possible and to produce a prolongation of the vowel /a/. A minimum of 20 repetitions of each behavior was elicited.

The EMD for each token was measured as the difference between the time of the onset of suprahyoid muscle activation and the onset of jaw depression. The onset times were identified using a custom routine written for MATLAB. The algorithm to determine the onset of muscle activity was adapted from earlier descriptions of EMD (Di Fabio, 1987). The EMG was demeaned, rectified, and low-pass filtered at 20 Hz. A baseline EMG reference region (75–175 ms) was defined in a window surrounding the visually estimated onset of the suprahyoid muscle activity. Infrequently, the user rejected the baseline because of the presence artifact and attempted to locate an alternative region of minimal activity. If no acceptable baseline region was located, the token was rejected. From an acceptable baseline, *EMG onset* was defined as the first point occurring three standard deviations above the mean of the baseline and remaining above this threshold for at least 25 ms. The *onset of jaw depression* was defined as the point following the onset of suprahyoid muscle activation at which the movement achieved 20% of its peak lowering velocity. The *EMD* was measured as the difference between the onsets of suprahyoid muscle activity and jaw movement. The derivation of EMD is shown in Figure 2. The upper trace illustrates the suprahyoid EMG signals, the lower trace shows jaw-lowering movement, and the EMD is indicated by the interval between the two solid lines.

To be included in the EMD measurement, tokens were required to exhibit a clear onset of activation and sufficient EMG modulation. *Sufficient modulation* was defined as at least a doubling of energy following EMG onset compared to the period preceding that onset and quantified by calculating a ratio between EMG energy post- and pre-onset. The energy of EMG was determined by the summed integral of the area falling under the filtered and rectified EMG. A second criterion for tokens to be included in the EMD analysis was a lack of jaw movement prior to the target jaw depression. A *sufficient rest period* was confirmed for the period 150 ms prior to the algorithm-defined EMG onset and was defined as the absence of any displacement greater than 10% of the displacement of the target lowering gesture.

Two further analyses were conducted to account for the presence of additional EMG activity, possibly above and beyond the background tonic activity, related to the experimental tasks that may have influenced the onset of jaw opening. These measures were the relative depth of EMG modulation and the cross-correlation of activity between muscle pairs of a subset of the EMD tokens. To measure the relative depth of EMG modulation of the four antagonist pairs and the suprahyoid muscles, the onsets of activity in the four antagonist muscles were determined by employing the algorithm used to determine the onset of the suprahyoid. *Modulation of muscle activity* was defined as the ratio of EMG energy pre- and post-EMG onset. To limit these analyses to antagonist modulation during the opening movement, only tokens with modulation occurring prior to the onset of the suprahyoid muscle or within a period double the length of the EMD following the onset of the suprahyoid muscle were included.

A second analysis of muscle activation was the zero-lag cross-correlation of muscle pairs, which provided a measure of muscle-activation coupling. The portion of the signal evaluated was a 500-ms window centered on the visually estimated onset of the suprahyoid muscle activity. The EMG channels were detrended, rectified, and low-pass filtered at 15 Hz prior to computing the cross-correlation function. Zero-lag cross-correlation values were calculated for each agonist–antagonist (i.e., suprahyoid and each of the four jaw elevators including the left temporalis [LT], right temporalis [RT], left masseter [LM], and right masseter [RM]) and antagonist–antagonist muscle pair (i.e., $LT \times RT$, $LT \times LM$, $LT \times RM$, $RT \times LM$, $RT \times RM$, $LM \times RM$), yielding 10 correlation coefficients for each token. The coefficients for each muscle pair were averaged across tokens within a participant for statistical analysis.

Results

Perceptual Validation of Stress Production

Each nonsense syllable was perceptually judged for correct stress placement prior to the kinematic analyses. A one-way analysis of variance (ANOVA) indicated a significant difference among the groups (children with DS, children with CP, and controls) on the percentage of tokens excluded because of a lack of listener agreement on stress placement, $F(2, 19) = 13.12$, $p < .001$. A Tukey's test post hoc analysis revealed that significantly more tokens were excluded from the group of children with DS ($p < .001$) because of lack of agreement of judgment on stress placement than from the control group.

Kinematic Analyses

Two- and three-syllable utterances were analyzed separately, because the comparison of jaw displacement of the opening gesture of the first and second syllable of “Mama” and “MAmama” were significantly different, $F(1, 24) = 11.29$, $p = .003$. All further kinematic analysis of two-syllable comparisons included the productions from DS ($n = 6$) and controls

($n = 5$) who produced two-syllable tokens, as only one participant with CP produced two-syllable tokens. Three-syllable comparisons included the productions from the CP group ($n = 2$) and controls ($n = 11$) who produced three-syllable tokens, as only one participant with DS produced three-syllable tokens.

Jaw displacement, peak velocity, and movement duration—Kinematic descriptors of stress production (e.g., displacement, velocity, duration of the opening gesture) were analyzed separately for two- and three-syllable productions using two-way repeated-measures ANOVAs (see Table 3). For two-syllable productions, jaw displacement and peak velocity were significantly different between the DS group and the control group and between stressed and unstressed syllables. The significant interaction between group and stress for displacement and duration indicated that the control participants used increased jaw displacement and duration to mark stressed syllables to at least a greater degree than did participants with DS. For three-syllable productions, increased jaw displacement, peak velocity, and movement duration were observed for stressed relative to unstressed syllables. This difference in peak velocity was greater for the control group than for the CP group, and movement durations were longer for the CP group.

Kinematic index—The bar graph in Figure 3 presents the mean kinematic indices (i.e., slopes) of two-syllable productions by the DS group and matched controls and one participant with CP. Figure 4 presents the mean kinematic indices of three-syllable utterances produced by the CP group and matched controls and one participant with DS. The mean slopes of the stressed and unstressed productions by the DS ($n = 6$) and control ($n = 5$) groups in two-syllable productions and CP ($n = 2$) and control ($n = 11$) groups in three-syllable productions were compared using two-way repeated-measures ANOVAs (see Table 4). For two-syllable productions, no differences were observed between the groups, between stressed and unstressed tokens, or for the interaction between group and stress. In fact, contrary to expectations, the mean kinematic index appeared higher for the stressed than unstressed tokens in the productions of participants with DS. For three-syllable productions, the kinematic index of unstressed tokens was significantly higher than the kinematic index of stressed tokens.

Correlation of peak velocity and displacement—To verify the covariance of displacement and velocity of the jaw (Vatikiotis-Bateson & Kelso, 1993), the strength of the relationship between peak velocity and displacement was evaluated using correlational analysis. The R^2 values from the linear regression for each participant's stressed and unstressed productions ranged from .11 to .90. The values were generally above .50, with 71% falling above .66 and 14% falling below .50, indicating a generally strong linear relationship for peak velocity and displacement.

Electromyographic Measures Related to Jaw Movement

EMD—The average EMD values and standard deviations for the groups are shown in Figure 5. The range of EMD values within the DS group was 12 to 43 ms, within the CP group was 29 to 43 ms, and within the control group was 20 to 52 ms. A one-way ANOVA comparing the mean EMD from each participant revealed no differences between the groups, $F(2, 16) = 1.04, p = .37$.

Modulation of jaw-muscle activation—One criterion for these tokens to be included in the analysis of antagonist modulation was the presence of an identifiable onset of activity of the jaw-elevating muscles in a window surrounding the onset of suprahyoid activation. This criterion, which implied some degree of coactivation of the agonist–antagonist muscles during jaw lowering, resulted in the inclusion of 74% of the tokens produced by the

participants with DS, 58% of the tokens produced by the participants with CP, and 45% of the tokens produced by the controls.

The average modulation ratio for five jaw-muscle groups (RT, LT, RM, LM, suprahyoids) for the participants in the three groups, the group averages, and adult values are seen in Figure 6. Whereas all of the control participants modulated activity in the suprahyoid muscles more than in the jaw-elevating muscles, two participants with DS and one participant with CP modulated the jaw-depressing muscle to a lesser degree during the jaw-opening target behavior. A two-way repeated-measures ANOVA across all three groups did not reveal group modulation ratio differences, $F(2, 16) = 1.78, p = .20$. Although a main effect for muscle was seen, $F(4, 16) = 15.06, p = .001$, the interaction between muscle and group was not significant ($p > .05$). An ANOVA across the three groups comparing the zero-lag cross-correlation between the muscle pairs did not reveal differences, $F(2, 16) = 1.60, p = .23$.

Discussion

In general, measurement tools for muscle tone have been criticized for their lack of objectivity, poor quantification (Pomeroy et al., 2000), and unclear relevance to functional movement. It is encouraging to note that techniques designed to measure stiffness of the oral articulators have been developed to address this concern (Chu et al., 2010; Solomon & Clark, 2010), although these techniques have not yet been demonstrated for use during functional movement and/or with children with motor speech disorders. The development of effective measures to quantify muscle tension in individuals with motor impairment is critical to a comprehensive understanding of both the nature and treatment of neuro-motor disorders. Understanding the role of muscle tone in functional movement such as speech production is necessary given that muscle-tone abnormalities are often cited as contributing to motor speech disorders, and the normalization of these abnormalities may be considered a treatment target (Clark, 2003; Ruscello, 2008). If evidence does support this treatment approach, valid and reliable measures of muscle tone have the potential to inform assessment and provide measures of progress of treatment. The current investigation was designed to uniquely apply two measures to obtain preliminary data for understanding orofacial structure differences during speechlike movements in children with suspected muscle-tone abnormalities.

Kinematic Index

For the kinematic index, the production of three-syllable tokens by participants with CP and controls demonstrated the expected difference across stress contrasts (i.e., steeper slope in the unstressed condition). To our knowledge, this investigation is the first to reveal the distinction of stressed and unstressed syllables using the kinematic index for jaw movement in young children and for children with motor speech impairment. A previous investigation of the tongue (Ostry et al., 1984) had demonstrated that older children (6+ years) and adults consistently demonstrated greater slope of unstressed than stressed tokens, although the effect was not consistent in younger children.

Similar kinematic slope differences across stress were not observed in the two-syllable productions in the current investigation. Among the number of possible interpretations of this finding, a likely contributor was the role of final syllable lengthening (Lehiste, 1972), because stress effects demonstrated by the kinematic index may be obscured in instances of final lengthening (Edwards, Beckman, & Fletcher, 1991). The potential final lengthening effects may have been more robust for the DS group, which demonstrated a trend of a higher kinematic index in the stressed rather than unstressed tokens (see Figure 3), contrary to expectations.

Evidence from the adult population suggests that kinematic index differences may vary with the specific disorder. Reduced kinematic indices have been observed in the arm movements (Brown, Hefter, Mertens, & Freund, 1990) and oral articulators of adults with cerebellar dysfunction (Ackermann, Hertrich, Daum, Scharf, & Spieker, 1997; Ackermann, Hertrich, & Scharf, 1995) or who have suffered traumatic brain injury (Jaeger, Hertrich, Stattrop, Schonle, & Ackermann, 2000). However, the results of investigations with adults with apraxia of speech (McNeil & Adams, 1990; Robin, Bean, & Folkins, 1989) and Parkinson disease (Ackermann et al., 1997; Forrest & Weismer, 1995) have been conflicting.

EMD

A previous investigation of the lower limbs of children with spasticity found that the EMD of the children with spasticity was significantly shorter than the EMD of the control group (Granata et al., 2000). The investigators interpreted these findings as providing quantification of the increased muscle tension in the children with spasticity. Contrary to the expected finding of a shorter average EMD for participants with CP and longer average EMD for participants with DS in the current study, however, the DS group range of EMD values included the shortest EMD overall. This finding was somewhat consistent with the results of Devanne, Gentil, and Maton (1995), who compared jaw-opening EMD in participants with Friedreich's ataxia and in unaffected adults. These investigators did not find significant differences between the EMDs of the participants with ataxia ($M = 43.0$ ms) and the control participants ($M = 42.7$ ms), although these EMD values were similar to those obtained in the current study. They also noted that the individuals with ataxia consistently demonstrated tonic antagonist activation of the masseter muscle prior to suprahyoid muscle activation for jaw depression.

Jaw-Muscle Activation

Muscle coactivation may be an additional influence on inferred muscle tension. Coactivation of antagonistic muscles increases joint stiffness, and mandibular coactivation has been shown to typify activities requiring fine control of the jaw, including speech in both adults (Moore, Smith, & Ringel, 1988) and children (Moore & Ruark, 1996). Although the motor development of many skills is characterized initially by coactivation of antagonistic muscles, with the mature form revealing a pattern of reciprocal activation (Damiano, 1993), a number of motor disorders are characterized by the atypical persistence of these patterns. Excessive muscle cocontraction has been documented in spasticity (Berger, 1998; Damiano, Martellotta, Sullivan, Granata, & Abel, 2000; Dietz & Berger, 1995) and DS (Almeida, Corcos, & Latash, 1994). In fact, perturbation studies have revealed an increased reliance on a coactivation strategy by participants with DS (Latash, 1992).

In the current investigation, although shallow modulation was observed in the jaw-elevating-muscle EMG, substantially deeper modulation of suprahyoid muscle activity was observed for the control participants and the adult, but it was not necessarily exhibited by children with suspected muscle-tone abnormalities. Two of the six participants with DS and one of the two participants with CP produced greater relative modulation of the jaw-elevating muscles compared with the suprahyoid muscles. Although this finding is not conclusive, it is consistent with the possibility of greater cocontraction of antagonist muscle groups, possibly superimposed on increased tonic activation levels, by children in the experimental groups. Furthermore, in this preliminary data set, the DS group tended to demonstrate jaw-muscle coactivation more frequently during jaw lowering than did the other groups. This muscle-activation pattern may explain the unexpected finding of relatively short EMD values among the participants with DS. Although the muscle coactivation analysis in the current investigation did not reflect significant findings between groups and was limited to a relatively short time frame in an isolated task, it is possible that children with DS rely on

muscle cocontraction to a greater extent than do age-matched controls, possibly as a compensatory mechanism for reduced muscle tension.

Conclusion

In this investigation, we sought to quantify the association of abnormally high or low mandibular muscle tone with speech impairment (Barlow & Abbs, 1984; Dietz & Berger, 1983) by employing two measures to estimate muscle tension not previously applied to the oral articulators for this purpose. The use of a novel approach that did not directly test muscle tension, coupled with other methodological limitations—such as small sample sizes and heterogeneous populations—limits the interpretation of the absence of expected differences between the groups. Future investigation will include a focus on establishing the validity and reliability of the experimental approach to estimate muscle tension, as well as multiple production targets. Given the preliminary nature of this investigation, it is unclear if the results reflect the lack of a long-suspected (and clinically expected) relationship between tone abnormalities and jaw movement during speech and nonspeech tasks, the above-mentioned study design limitations, or the differential impairment of the articulators—because different speech motor systems comprise a broad range of biomechanical and neuromuscular characteristics, and neurological disorders can variously affect speech motor systems (Abbs et al., 1983; Ackermann et al., 1997; Jaeger et al., 2000).

Despite these factors, concerns persist regarding both the inability to differentiate speakers using tasks and measures designed to quantify muscle tone and the lack of a clear relationship between muscle tone and dysarthria in neuromuscular disorders. This uncertainty calls into question the validity and role of muscle tone in the underlying pathophysiology and subsequent treatment of speech impairment. Further investigations to determine the physiological basis of motor speech disorders are essential to our models of speech motor control, our understanding of speech pathophysiology, and the design of appropriate treatments, including those that seek to address articulatory impairment arising from putative disruptions of muscle tone.

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Figure 1.

The peak velocity–displacement relationship of multiple productions of “Mamama” produced by one child with cerebral palsy (CP; CP2) and one age- and gender-matched control (CPM2). The black squares indicate the unstressed syllables, and open circles indicate the stressed syllables. The regression equation for the stressed and unstressed syllables is provided, with the slope representing the kinematic index.

Figure 2. Derivation of electromechanical delay (EMD) from suprahyoid electromyography (EMG; upper trace) and jaw movement (lower trace). The solid vertical lines represent the onset of EMG (left line) and onset of jaw movement (right line). The EMD is the time between the onsets.

Figure 3. Group mean (+ *SD*, indicated by error bars) of the kinematic index for two-syllable productions by children with Down syndrome (DS), children with CP, and age- and gender-matched controls.

Figure 4. Group mean (+ *SD*, indicated by error bars) of the kinematic index for three-syllable productions by children with DS, children with CP, and age- and gender-matched controls.

Figure 5. Group mean (+ *SD*, indicated by error bars) EMD by children with DS, children with CP, and age- and gender-matched controls.

Figure 6.

Modulation ratios of the five muscles during jaw lowering for each participant, and group averages. The top panel represents the individual and mean data for the participants with DS, the middle panel represents the individual and mean data for the participants with CP, and the bottom panel represents the individual and mean data for the age- and gender-matched controls and data from one adult. rtemp = right temporalis; ltemp = left temporalis; rmass = right masseter; lmass = left masseter; abd = anterior belly of the digastric (a suprahyoid) muscle.

Table 1

Participant characteristics.

Participant code	Group	Age (years;months)	Gender	Control match	Age
DS1	Down syndrome	4;0	Female	DSM1	3;11
DS2	Down syndrome	4;7	Female	DSM2	5;0
DS3	Down syndrome	4;11	Male	DSM3	4;11
DS4	Down syndrome	5;4	Male	DSM4	4;10
DS5	Down syndrome	5;6	Male	DSM5	5;11
DS6	Down syndrome	7;4	Male	DSM6	7;10
DS7	Down syndrome	8;5	Male	DSM7	8;8
			Male	DSM8	10;0
CP1	Cerebral palsy	4;4	Male	CPM1	4;3
CP2	Cerebral palsy	5;6	Male	CPM2	5;4
CP3	Cerebral palsy	6;0	Female	CPM3	6;0

Table 2

Clinical characteristics of participants with Down syndrome (DS1–DS7) and cerebral palsy (CP1–CP3).

Participant	Parent report of feeding/oral problems	Oral structure–function				Intelligibility (%)	Dysarthria severity rating
		Lips	Tongue	Jaw	Abnormal reflexes		
DS1	*		+	++	gag [–]	7	6
DS2				+	gag [–]	0	7
DS3	*	+	+	++		7	6
DS4		++	++	+	gag [–]	8	7
DS5	*	++		+		3	6
DS6		++			gag [–]	10	7
DS7	*	+	+	++		5	7
CP1	*	Did not comply			suck [*]	Did not comply	7
CP2	*	++	+++	++	gag [*]	3	7
CP3	*	+	+	++		64	5

Note. Dysarthria severity rating based on 9-point scale (0 = none, 2 = mild, 4 = moderate, 6 = severe, 8 = profound).

[–] = absent or reduced;

^{*} = present or excessive;

⁺ = mild impairment;

⁺⁺ = moderate impairment;

⁺⁺⁺ = severe impairment.

Table 3

Analyses of variance (ANOVAs) of jaw displacement, peak velocity, and movement duration.

Comparison	Two syllables DS vs. C		Three syllables CP vs. C	
	<i>F</i> (1, 187)	<i>P</i>	<i>F</i> (1, 356)	<i>P</i>
Displacement (cm)				
Group	32.54	< .00*	0.15	.70
Stress	7.91	< .01*	255.57	< .00
Interaction	14.41	< .00*	5.21	.02*
Peak velocity (cm/s)				
Group	43.85	< .00*	0.23	.63
Stress	17.89	< .00*	71.43	< .00*
Interaction	0.02	.89	5.76	.02*
Duration (s)				
Group	2.29	.14	8.80	.00*
Stress	0.13	.71	388.86	< .00*
Interaction	27.54	< .00*	0.98	.32

Note. Group = children with suspected hypotonia (DS) versus controls (C), or children with suspected hypertonia (CP) versus controls; Stress = stressed versus unstressed; Interaction = Group $\times$ Stress.

*
 $p < .05$.

Table 4

ANOVA of the kinematic index.

Comparison	<i>F</i>	<i>df</i>	<i>P</i>
Two-syllable productions (DS vs. C)			
Group	0.63	1, 9	.45
Stress	4.99	1, 9	.05
Interaction	4.66	1, 9	.06
Three-syllable productions (CP vs. C)			
Group	1.42	1, 11	.26
Stress	32.50	1, 11	< .00*
Interaction	2.30	1, 11	.23

*
 $p < .05$.