
Article abstract—Motor impairments in the fine force control of lips, tongue, and jaw were measured in subjects with congenital spasticity. Because these orofacial motor systems are not uniformly endowed with muscle spindles and monosynaptic reflexes, quantification of these motor impairments addresses the question of whether stretch reflex hypertonus is a positive or negative sign. The results indicated that hyperactive muscle spindle-based monosynaptic reflexes are not a causal factor in these voluntary orofacial motor impairments. These data also indicated that motor impairments were disproportionately greater at finer levels of isometric force control. These fine control measures appear useful as a quantitative index of general voluntary motor deficit.

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Orofacial fine motor control impairments in congenital spasticity: Evidence against hypertonus-related performance deficits

Steven M. Barlow and James H. Abbs

Evidence has accumulated to suggest that the hypertonus of spasticity is not causally related to abnormal motor performance.¹⁻⁵ Specifically, Landau⁴ argues that hyperactive stretch reflexes and hypertonus are, in a Jacksonian sense, positive neuropathologic signs; namely, abnormal resistance to passive stretch is unrelated to voluntary movement impairment. This view has been supported by examining performance deficits after elimination of muscle afferent influences with anesthesia or surgery. Corroborating this interpretation are observations that certain drugs, such as baclofen, reduce hypertonicity but do not improve motor performance.^{2,6,7} However, such drugs seldom have a unitary action.

A further test of this issue is to examine motor deficits in motor systems that do not have conventional muscle spindle actions, such as in the cranial nerves. The facial muscles lack muscle spindles; conventional stretch reflexes and responses to tonic vibration are lacking.⁷⁻¹⁰ By contrast, the jaw closing muscles are densely supplied with spindles, and a monosynaptic stretch reflex can be evoked readily.¹¹⁻¹⁴ The tongue muscles are intermediate between the lip and the jaw muscles; a few spindles have been

found in the intrinsic tongue muscles of primates,^{15,16} but these afferents do not seem to project directly to hypoglossal motoneurons.^{10,17} Predictably, Neilson et al¹⁰ reported jaw muscle hypertonus in congenital spastic patients, but no comparable hypertonus of lip or tongue muscles. In this context, our goal was to compare motor impairments among the jaw, lips, and tongue. Although choosing an index of motor impairment in patients with congenital spasticity is not trivial, most recent opinion and theory suggests some direct measure of fine control.^{4,18} As such, a secondary goal of this study was to examine the potential clinical value of quantifying fine force control impairments.

Methods. Subjects. We studied six men with congenital spasticity (table) and three normal controls. A major consideration in interpreting orofacial fine force control impairments was certainty that these patients presented uncontaminated signs of congenital spasticity. Precautions were taken to eliminate prospective subjects with even mild signs of "mixed" neurologic disease, including two independent neurologic examinations and a complete evaluation by a

From the Speech Motor Control Laboratories, Waisman Center for Human Development, University of Wisconsin-Madison, Madison, WI.

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Address correspondence and reprint requests to Dr. Abbs, Speech Motor Control Laboratories, 1500 Highland Avenue, University of Wisconsin-Madison, Madison, WI 53706.

Table. Neurologic profiles of congenital spastic subjects

Subject 1	Mild left hemiplegia. Visual acuity WNL.
Subject 2	Mild spastic diplegia. Involvement greater on the left than the right. Corrective lenses required.
Subject 3	Spasticity present in the right arm and in both legs. Visual acuity WNL.
Subject 4	Spastic diplegia: mild spasticity in the left arm, severe spasticity in both legs. Visual acuity WNL.
Subject 5	Moderate-severe spastic quadraplegia. Visual acuity WNL.
Subject 6	Moderately involved spastic quadraplegia. Visual acuity WNL.

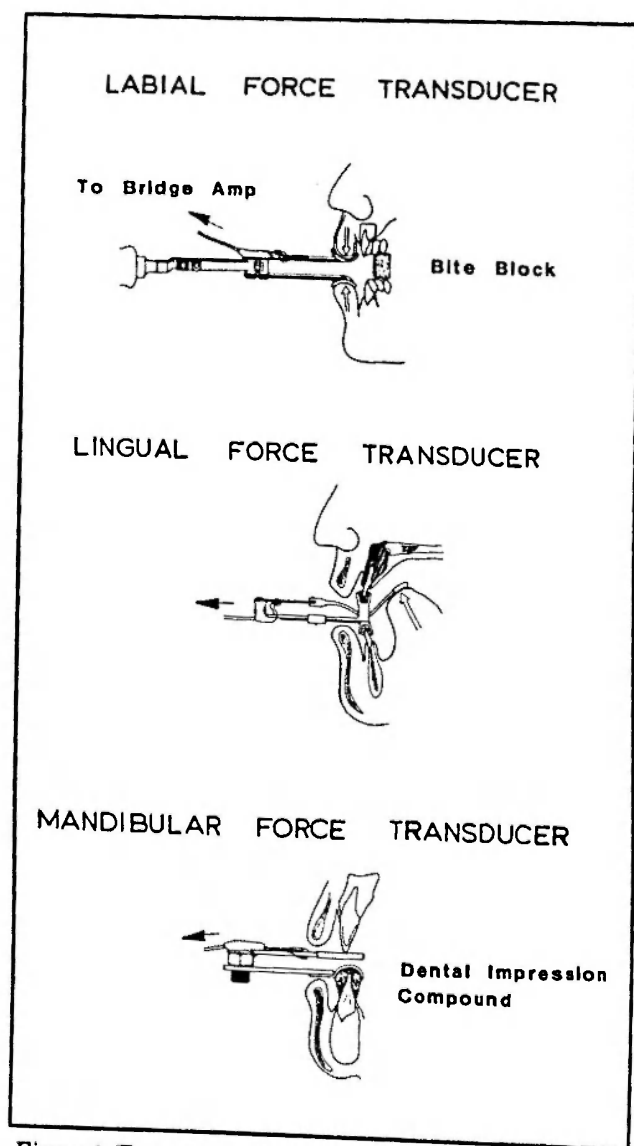


Figure 1. Transducers for monitoring isometric contraction forces in the tongue, lips, and jaw.

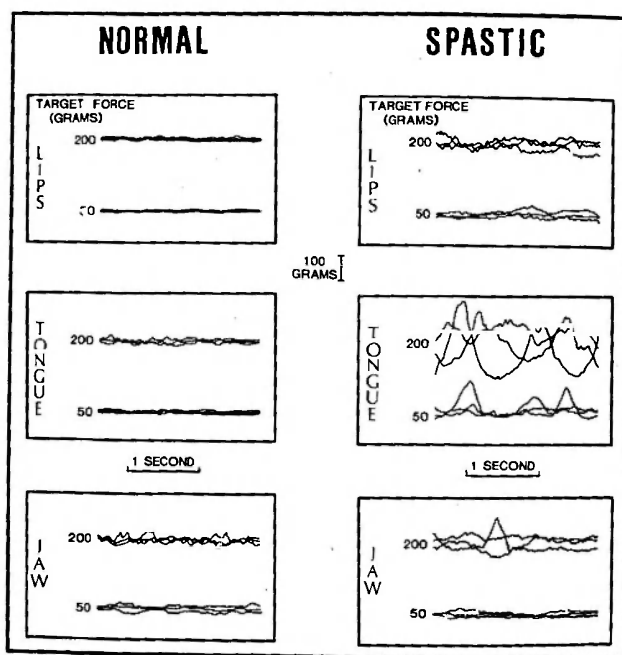


Figure 2. Production of lip, tongue, and jaw isometric forces at two levels (50 and 200 grams) by a normal and congenital spastic subject.

speech pathologist. Particular care was taken to detect indications of differential cranial nerve involvement.

Apparatus. Force control measures from the lips, jaw, and tongue were obtained with three force transducers (figure 1). These strain gauge transducers provided analog signals of the desired muscle forces. A more complete description of these devices is forthcoming.¹⁹

Procedures. All subjects produced submaximal, isometric forces. Ten contractions, 4 to 5 seconds in duration, were obtained at multiple force levels for each orofacial system (50, 100, 200, 300, 400, and 500 grams for the jaw; 50, 100, 150, and 200 grams for the tongue; and 20, 50, 100, 150, and 200 grams for the lips). Each target force level was displayed on an oscilloscope at a slow sweep speed (500 msec/div). A cursor reflecting the subject's force output was provided. The objective of this task was to generate a stable force at each target level. These force signals were computer digitized (300 samples/sec). Control instability was quantified by calculating the standard deviation for 900 samples obtained during 3 seconds of each force trial. This measure provided an index of "fine motor adjustment."⁴

Results. All spastic subjects were less stable in the generation of fine orofacial forces than the normal controls. Figure 2 illustrates force signals from a normal subject and a spastic subject. Clearly, in this spastic subject tongue isometric control is more impaired than that of either the lips or jaw. The overall distribution of fine force control impairments for the six spastic subjects is shown in figure 3. These

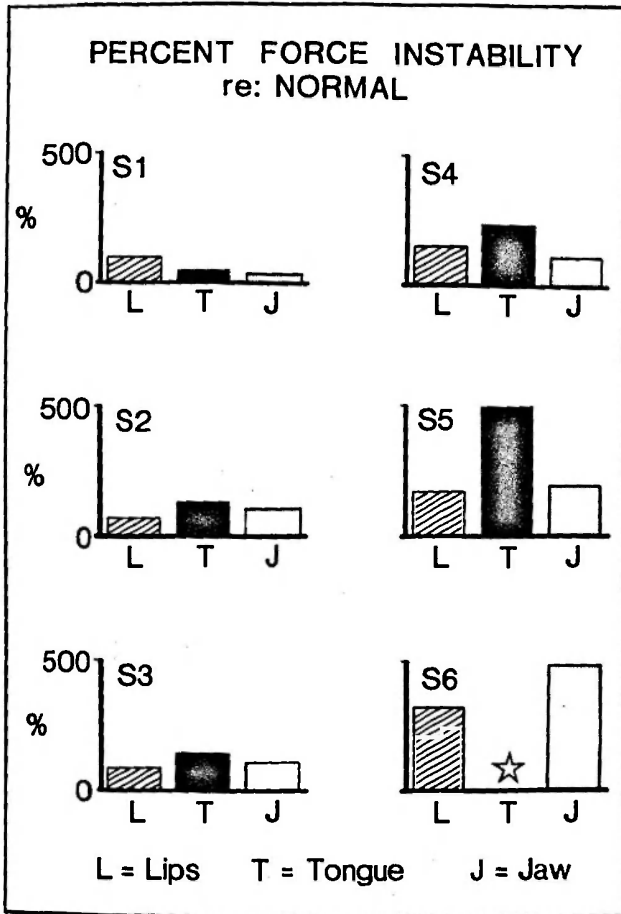


Figure 3. Relative degree of fine force control instability for the lips, tongue, and jaw for the six spastic subjects. Each histogram bar represents the average instability across the force levels observed, expressed as a percentage of normal subject performance.

values reflect force control instability as a percentage of the normal instability, permitting lip, tongue, and jaw comparisons. In these spastic subjects the tongue manifests greater relative instabilities than either the lips or the jaw. Also noteworthy is that within this disordered group, lip, jaw, and tongue impairment is not uniform. Whereas subjects 2 and 3 showed comparable degrees of impairment in these three orofacial systems, subjects 4 and 5 are disproportionately impaired in tongue control. Further, spastic subject 6, with considerable instability in the lips and jaw, was able to generate only minimal tongue forces, indicating lingual weakness. None of the subjects manifested disproportionate impairment in the jaw.

These data allowed us to address whether congenital spastic patients manifest a disproportionate control impairment for finer levels of muscle contraction. Figure 4 illustrates the force signals for subject 5 for two levels of jaw contraction; relative instability was considerably greater at the low force level. As shown in figure 5, this pattern was reflected in the spastic subjects as a group. Relative control instability for the controls was slightly greater at the lowest levels of contraction. By contrast, fine control impairment in the spastic subjects was considerably greater at lower levels of contraction. At higher force levels relative instability for the spastic subjects approached that of the controls.

We also attempted to determine how well these measures reflected impairments of functional motor behaviors. Two experienced speech pathologists scaled the overall severity of speech performance in the spastic subjects by listening to audio recordings. These judgments were then correlated with a com-

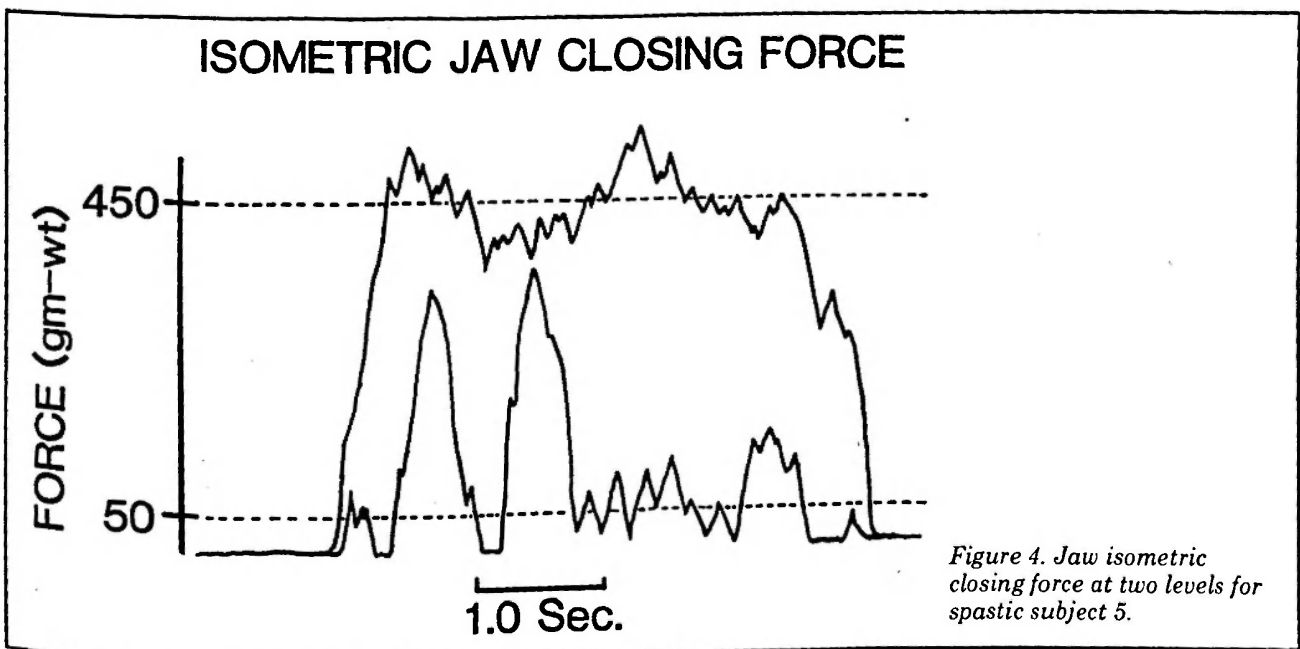


Figure 4. Jaw isometric closing force at two levels for spastic subject 5.

posite index of lip, jaw, and tongue force instability (for each spastic subject, re: normal) using a rank order statistic (Spearman's Rho). The obtained correlation coefficient was 0.886, illustrating the predictive value of these measures. These observations also highlighted the sensitivity of direct force control measures; spastic subject 1 was judged to have nor-

mal speech, but the measures shown in figure 3 indicate otherwise.

To illustrate the relation between fine force control impairments and speech motor deficits more directly, the spastic subjects with the least and greatest control impairments (subjects 1 and 5) were studied further using measures of orofacial movement

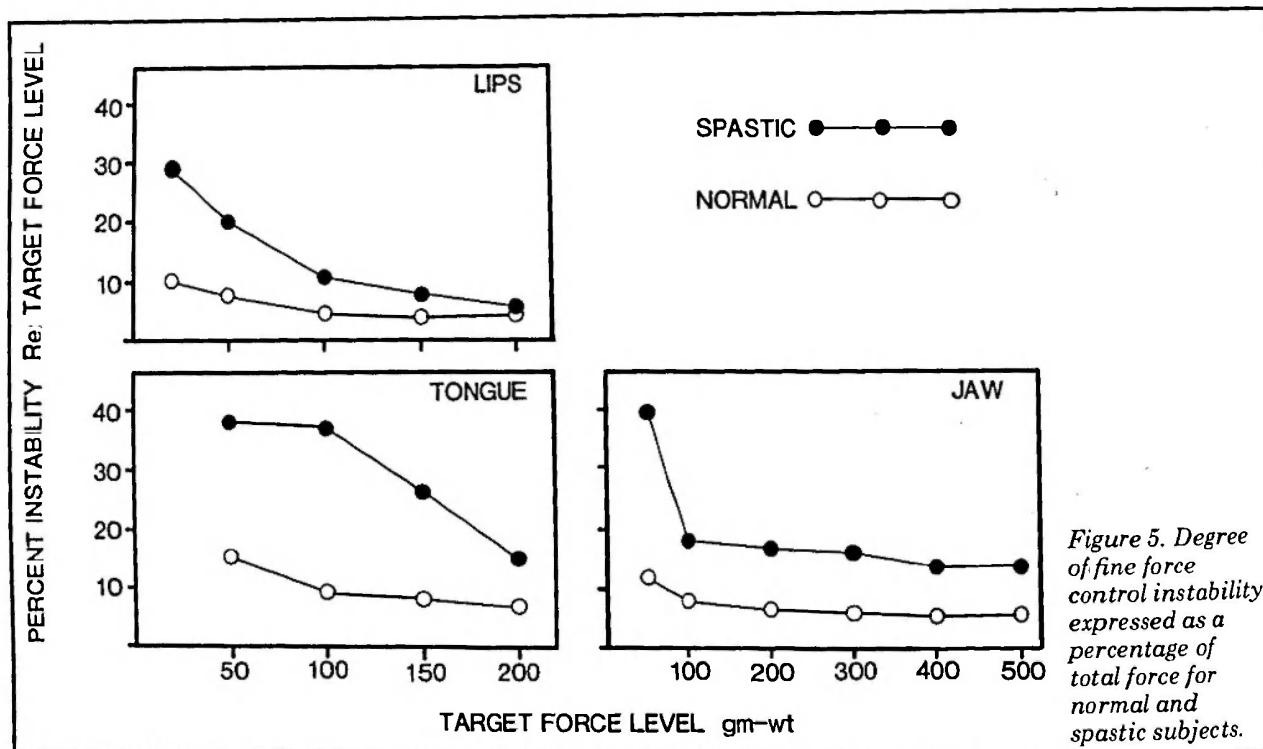


Figure 5. Degree of fine force control instability expressed as a percentage of total force for normal and spastic subjects.

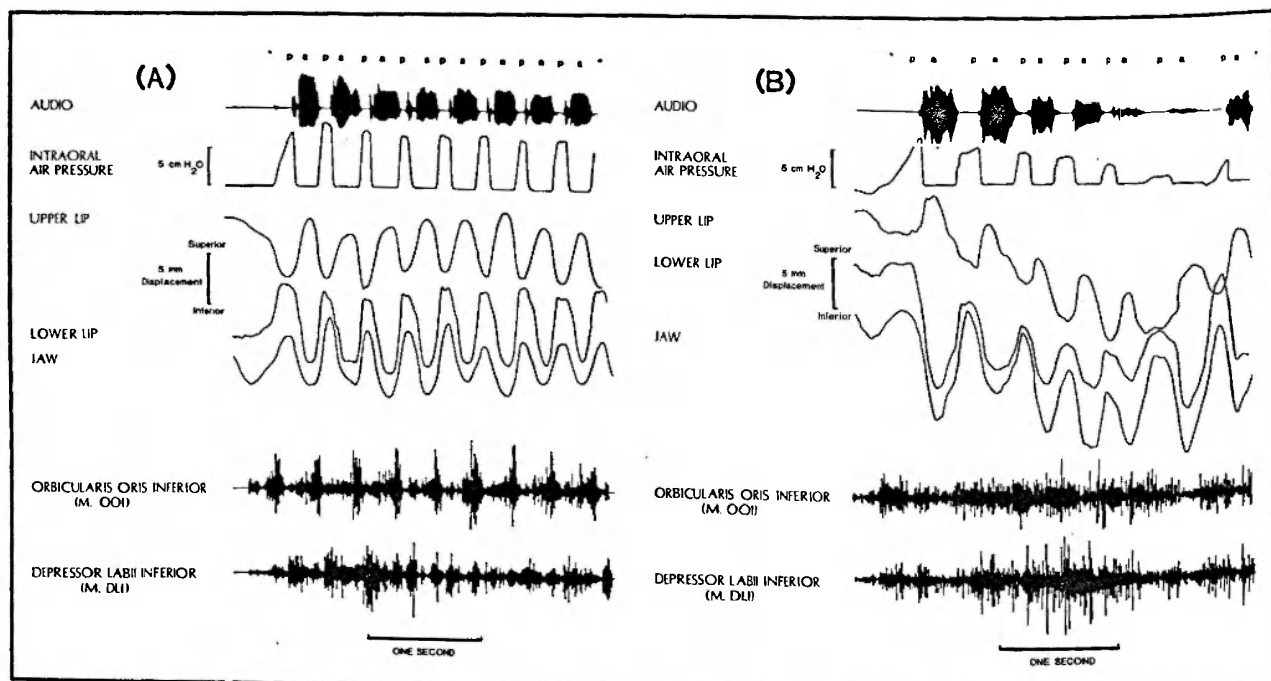


Figure 6. Labial-mandibular movement and lip muscle EMG for a simple speech task (diadochokinetic PaPaPa...) for a mildly impaired spastic subject (subject 1, A) and severely impaired spastic subject (subject 5, B).

and muscle activity. Upper lip, lower lip, and jaw movement were transduced using a head mounted movement transducer system designed for use with abnormal subjects.²⁰ EMG from two lower lip muscles was recorded simultaneously with intramuscular, hooked-wire electrodes. Global measures of speech performance were obtained in parallel from intraoral air pressure and speech acoustic signals. Subjects were instructed to repeat the syllable "pa" at a rate of about three per second. The movement behavior associated with this syllable repetition was reasonably well preserved in the mildly involved subject 1 (figure 6A); lip and jaw movements were smooth, muscle activity was semidiscrete and phasic, intraoral air pressures for the oral closure for "p" were regular, and voicing was adequate. By contrast, in subject 5, a near complete breakdown in movement is apparent (figure 6B). Lip and jaw movement trajectories were irregular, and the movement range was highly variable. Intraoral air pressure and voice signals were likewise irregular. EMG bursts were undifferentiated compared with the discrete, phasic bursts commonly observed in normal or in milder spastic subjects (figure 6A). The differences between the speech performance of these two spastic subjects illustrates the value of fine control measures to predict impairments of general functional performance.

Discussion. These data indicate that motor performance deficits associated with spasticity are not disproportionately severe in motor systems with dense spindle innervation and monosynaptic reflex pathways. Impairments of the tongue (a system without a stretch reflex) were, on the average, most severe. Lip and jaw motor impairments were roughly equivalent in five of the six spastic subjects studied. It appears that in the cranial motor system aberrant actions of stretch reflex mechanisms do not underlie impairments of voluntary motor control. These data augment earlier studies in that potentially confounding surgical or pharmacologic conditions were not utilized. Further, instrumental observation of the distribution and degree of impairment severity permit a quantitative test of this hypothesis. These observations augment Neilson and O'Dwyer's²¹ observations which indicated that hypertonus is not a causal factor in the spastic speech motor impairments.

These data address the value of fine force control measures to assess general motor impairment in spastic subjects. For the subjects in the present experiment there was a high positive correlation between fine force control instability and parallel, independent measures of speech impairment. These measures also confirm the clinical observations that spastic subjects are disproportionately impaired in control of fine motor tasks.²² This interpretation is consistent with impairments of jaw fine force control observed after motor cortex lesions in nonhuman primates²³; eg, the force control impairments shown in figure 3 of Luschei and Goodwin²³ are strikingly

similar to those shown in figure 4 of this paper. Moreover, Evarts and Fromm^{24,25} and Fromm²⁶ indicate that the feedback pathway operating through the precentral motor cells is especially important for voluntary control of fine motor adjustments. Although the nature of cortical abnormality in congenital spastics is not fully understood, available observations²⁷⁻²⁹ and the importance of these cortical areas for fine motor adjustments are consistent with the present findings. Similarly, fine force control impairments should correlate with speech motor deficits. Speech movements do not require large muscle forces, but rely on fine, precise control of submaximal contractions ranging from 10 to 20% of maximum.³⁰

The correlation between impairments in the voluntary generation of small isometric forces and the motor execution for speech is also consistent with Jackson's concepts of "most automatic" and "least automatic" motor behaviors. Both speech and voluntary production of small muscle forces would be considered least automatic with dependence on control functions of precentral cortical neurons.^{26,31} With motor cortex abnormalities, a positive correlation in impairment severity among different "voluntary" motor behaviors is thus expected.

Finally, these data suggest that there is substantial variation in the severity among orofacial motor subsystems in any given spastic subject. These variations provide some insight into the degree and distribution of supranuclear abnormality. Further, nonuniform impairment of orofacial motor systems indicates the value of tests that quantify these differences in performance. Such measures should thus be of value in refining assessment and diagnosis, to evaluate the results of therapeutic intervention, and to quantify changes that occur with progressive motor impairments.

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