A Comparison of Labiomandibular Kinematic Durations, Displacements, Velocities, and Dysmetrias in Apraxic and Normal Adults

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Adult acquired apraxic speakers have been described as sounding slow (Lebrun, Buyssens, and Henneaux, 1973). Acoustic analyses reported by several authors (Shankweiler, Harris, and Taylor, 1968; Collins, Rosenbek, and Wertz, 1983) have verified the extended durations of apraxic speakers, with vowel prolongations sharing the burden of these extensions. Extended intersyllabic durations (Kent and McNeil, 1987) and prolonged consonants (Kent and McNeil, 1987; Kent and Rosenbek, 1983) have also been found and are believed to contribute to the perception of slowed speech. The explanations for these extended movement durations and silent periods have ranged from those attributable to (1) a conscious compensatory strategy to (2) a motor programming disorder to (3) a motor execution disorder in which there is a decrease in movement speed (velocity). Other kinematic variables could, however, account for the finding of slower speech in the apraxic speaker. That is, a gesture with a greater displacement could take longer to execute, especially if the normal peak velocity and displacement relationship were not achieved. Movements could take longer to execute because there is variation in the movement trajectory (dysmetria) in the absence of longer peak velocity or greater displacement. Only through detailed kinematic analyses can these various accounts of speech slowness be investigated.

Although the literature is abundant with quantitative movement studies for normal and dysarthric subjects (Abbs and Netsell, 1973; Hirose, Kiritani, and Sawashima, 1982; Kuehn and Moll, 1976; Sussman, MacNeilage, and Hanson, 1973), few kinematic studies have been employed to evaluate labial, mandibular, lingual, and velar kinematics in apraxic speakers (Fromm, Abbs, McNeil, and Rosenbek, 1982; Itoh and Sasanuma, 1984; Itoh, Sasanuma, Hirose, Yoshioka, Yoshioka, and Ushijima, 1980). Even in these kinematic studies of apraxic speech, data analysis has been perceptual relative to interstructural timing and coordination. To our knowledge, only three studies have reported quantitative kinematic data concerning the rate of movements in apraxic speakers compared to normal subjects or other control groups to determine the actual velocity of the movements. Using an x-ray microbeam device, Itoh, Sasanuma, Hirose, Yoshioka, Yoshioka, and Ushijima (1980) reported peak velocities for one apraxic subject that were ". . . approximately one-half the value of the fast rate of the normal speaker and [were] similar to that of the ALS patient." These values were slightly greater than 100 mm/s for the apraxic speaker producing repetitions of /pa/ at his maximum diadokokinetic rate. Using a light-emitting diode system of measurement, Itoh and Sasanuma (1987) reported peak velocities for five young normal, five aged normal, five Broca (apraxic), and three Wernicke aphasic subjects. They found that both the apraxic and normal speakers produced the previously well documented finding in normal subjects that peak velocities were positively correlated

with the magnitude of the displacement. However, the apraxic subjects produced ranges of peak velocities and displacements that were greater than the normal subjects. They concluded that the apraxic speaker's articulatory speed was not generally slow. Some individual trials were, however, outside the range of the velocity-displacement slopes produced by the normal subjects.

More recently, Robin, Beam, and Folkins (in press) reported lower lip velocities for six apraxic subjects that were within the range of their single normal control subject. Further, these velocities were not different from the normal control as a function of speech rate (normal to fast), correct versus incorrect productions, or as a function of whether the jaw was blocked (lower lip only) or unblocked (lower lip plus jaw).

It was the general purpose of this investigation to expand on the limited information currently available on the speech kinematics in persons with apraxia of speech (AOS). Specifically, we sought to determine if the movements for the lower lip plus jaw were significantly different in peak velocity between normal and apraxic subjects. We also asked whether the movement durations, maximum displacements, and number of dysmetrias were significantly different between the two groups.

METHODS AND PROCEDURES

SUBJECTS

Eight adult males served as subjects for this investigation. All were native speakers of English and had speech discrimination of 70 percent or better at 40 dB HL in at least one ear. Four of these subjects were normal controls, between the ages of 57 and 67 years. All were without a history or evidence of speech, language, cognitive, or neurological deficits as measured by a neurological examination conducted by a board-certified neurologist and by a large battery of standardized speech, language, and cognitive tests administered by a certified speech-language pathologist. The tests relevant to the description of the subjects for this particular study were the Raven Coloured Progressive Matrices (RCPM) (Raven, 1962), the Word Fluency Measure (WFM) (Borkowski, Benton, and Spreen, 1967), the Revised Token Test (RTT) (McNeil and Prescott, 1978), the Porch Index of Communicative Ability (PICA) (Porch, 1967), and the Structural-Functional Speech System Evaluation (S-F) (Veterans Administration Hospital Examination, Madison, WI). In addition all apraxic subjects were given a computed tomographic (CT) scan at the time of the examinations. All scans were later read and interpreted by a board-certi-

fied neurologist experienced in the description and quantification of CT data for speech and language research. Lesions for the apraxic subjects involved a wide range of cortical and subcortical structures, with the only common lesion location among subjects being the inferior portion of the post-central gyrus.

The remaining four subjects were diagnosed as having apraxia of speech without concomitant dysarthria and without aphasia to a degree that would interfere with the accomplishment of the task or to a degree that was detectable with the battery of tests administered. Darley's (1982) definition was used for the conceptual definition of aphasia. These subjects ranged in age from 54 to 72 years.

The presence of apraxia of speech was judged perceptually by two certified speech-language pathologists experienced in the detection of apraxia of speech and its differential diagnosis from aphasia and dysarthria. These judgments included the presence of effortful trial and error groping on the initiation of speech gestures; frequent single feature sound substitutions; articulation and prosody judged at least as accurate on imitation as on spontaneous speech production on the cookie theft picture description from the Boston Diagnostic Aphasia Examination (BDAE); variability of articulation and prosody on repeated trials of the same utterance; articulatory agility, phrase length, and melodic line ratings on the BDAE between 1 and 4, and no evidence of weakness or incoordination of the speech musculature on examination (clinical neurologial examination or the S-F examintion) and when used for reflexive or automatic acts such as chewing, swallowing, and sucking. Other criteria for inclusion in this category were a score at or above the 95th percentile for aphasic subjects on the average of subtests II, III, V, VI, VII, VIII, X, and XI of the PICA and a score of 22 or above on the RCPM. The basic biographical and descriptive data for all subjects are summarized in Table 15-1.

TABLE 15-1. SUBJECT BIOGRAPHIC AND DESCRIPTIVE DATA

	Normal (N = 4)	Apraxic (N = 4)
Gender	Male (4)	Male (4)
Age	$\bar{X}$ = 62.8; R = 57-67	$\bar{X}$ = 61.8; R = 54-72
Neurological examination	Normal	Abnormal
RCPM	$\bar{X}$ = 31.75; R = 29-33	$\bar{X}$ = 28.25; R = 27-30
WFM	$\bar{X}$ = 43.25; R = 30-58	$\bar{X}$ = 17.00; R = 11-31
RTT	$\bar{X}$ = 14.53; R = 14.15-14.83	$\bar{X}$ = 13.08; R = 12.08-14.07
PICA	$\bar{X}$ = 14.68; R = 14.51-14.84	$\bar{X}$ = 14.62; R = 14.33-14.96

R = range.

PROCEDURES

The data for this investigation were collected in the context of a larger study designed to evaluate the relationships among electromyographic, aerodynamic, acoustic, and kinematic measures within and among several subject groups, on multiple experimental tasks chosen for their potential influence on the speech of persons with ataxic dysarthria, conduction aphasia, and/or apraxia of speech. Subjects were seated in a comfortable dental chair, with the head-held movement transducers affixed to the upper lip, lower lip, and jaw. Movements were transduced using a light-weight cantilever beam instrument with resistive strain gauges (Barlow, Cole, and Abbs, 1983). Movements were transduced in the midsaggital plane, and data were recorded on a 12-channel FM tape recorder for later digitization and analysis using a PDP 11/44 laboratory computer. In this condition, the subjects were instructed to repeat 40 different words and phrases after each stimulus delivered from a tape recorder. Each stimulus was randomly presented among the other 39 stimuli. Five randomly presented repetitions of each stimulus for each subject were collected for later analysis. From this corpus of data, one stimulus item, "stop fast," was selected for analysis and comparison between the normal and apraxic subject groups. This utterance was selected because it provided an opening and closing labial gesture and because it required a CC transition in the gesture medial position.

Only the data from the lower lip transducer (i.e., movements transduced from the lower lip plus jaw) were analyzed for this investigation. Data analysis involved displaying the analog movement and concurrent acoustic signals for each trial. A cursor was placed at the onset of the closing gesture for the /ap/ in "stop" and at the maximum lower lip plus jaw excursion for /f/ in "fast." The cursor was aided in placement with reference to the movement by the offset of the acoustic signal from the preceding vowel for the closing gesture and by the onset of the vowel for the opening gesture. The movement signals were then digitally filtered (low pass cutoff of 30 Hz) and differentiated. From these data, four measurements were made: (1) The total duration of the closing plus opening gesture was determined in milliseconds, (2) the peak instantaneous velocity of the movement was determined in millimeters per second, (3) the maximum displacement of the closing gesture was calculated in millimeters, and (4) the number of occurrences in which the velocity changed direction (accelerations or decelerations) was measured and counted. The zero velocity crossings were termed dysmetrias.

The data for each subject group and each of the four measures were analyzed separately with a between groups one-way ANOVA. A .05 alpha level was set for all comparisons. The values for the five trials of the utterance for all of the four subjects in each group were used for each of

the analyses. Spearman rank order correlation coefficients were computed among selected dependent measures for each of the subject groups.

RESULTS

DURATION

The average duration of the defined movement for the lower lip plus jaw for each individual subject (open bars) and for the group (solid bars), along with 1 SD about the mean (error bars) are represented in Figure 15-1. Each group average is presented with the solid bar. The average duration for the apraxic group was 265 ms longer than the average for the normal group. Results of the between group analysis revealed that this difference was statistically significant at the .05 level of confidence. As seen in this figure, the normal group was relatively homogeneous, with one subject (N3) having slightly longer durations than the other three. It can also be seen that one apraxic subject (A2) had an average duration for this speech gesture that was comparable to the normal group. Further, the standard deviations for the normal group were quite small and relatively homogeneous. Contrarily, the variability for each of the apraxic subjects was larger than for the normal subjects except for apraxic subject A2, whose standard deviation was the same as for subject N1.

PEAK VELOCITY

The average peak velocity for each subject and for each group are represented in Figure 15-2. Group means were not statistically significant (p > .05) from each other. As is readily apparent from an inspection of the individual subject average peak velocities and from the error bars, there was considerable within and between group variability in the subject's lower lip plus jaw peak velocity for this speech segment. Within subject variability was generally greater for the apraxic group; however, apraxic subjects A1 and A2 had standard deviations within the ranges of normal subjects. Normal subject 4 produced an average peak velocity that was equivalent to the largest average of the apraxic subjects. However, the variability of peak velocities was smaller for this and all other normal subjects at equivalent peak velocities.

The magnitude of the peak velocity does not describe, in adequate detail, the many potential differences that could exist in the overall velocity profile. A visual inspection of the velocity traces reveals considerably more variability in the morphology of the contour for the apraxic subjects.
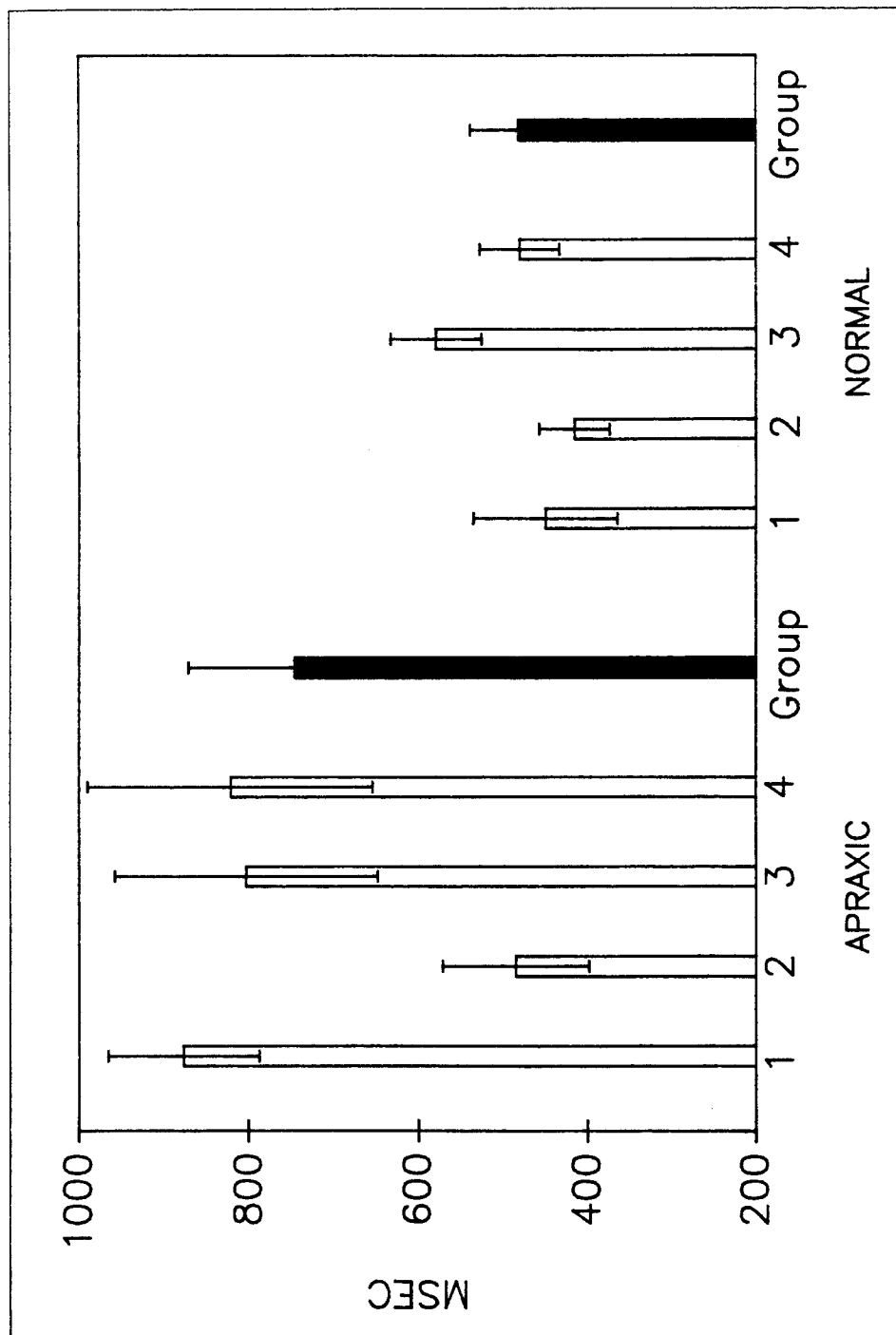
Figure 15-1. Lower lip duration means and standard deviations (*error bars*) for each subject (*open bars*) and for the apraxic and normal groups (*solid bars*).
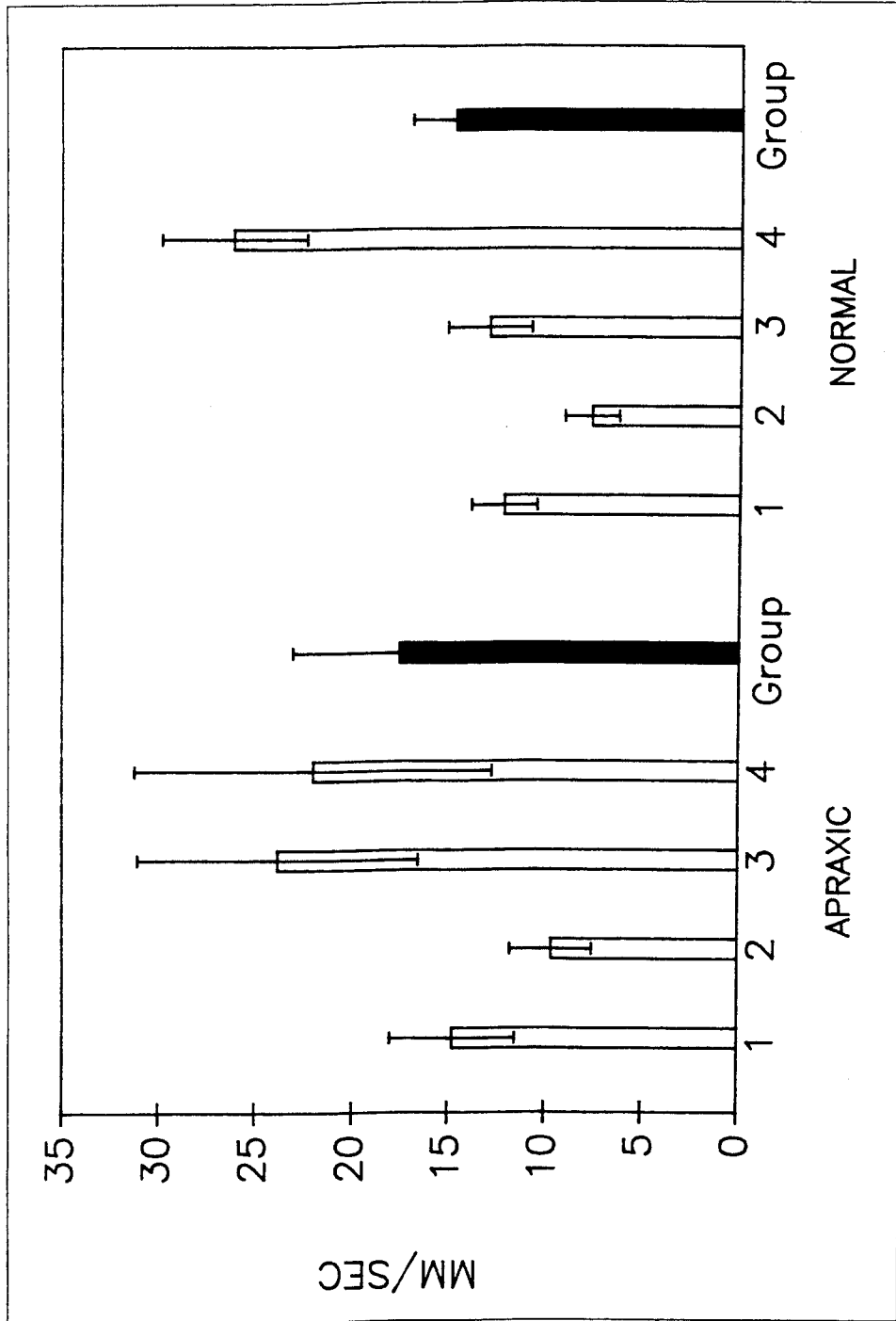
Figure 15-2. Lower lip peak velocity means and standard deviations (*error bars*) for each subject (*open bars*) and for the apraxic and normal groups (*solid bars*).
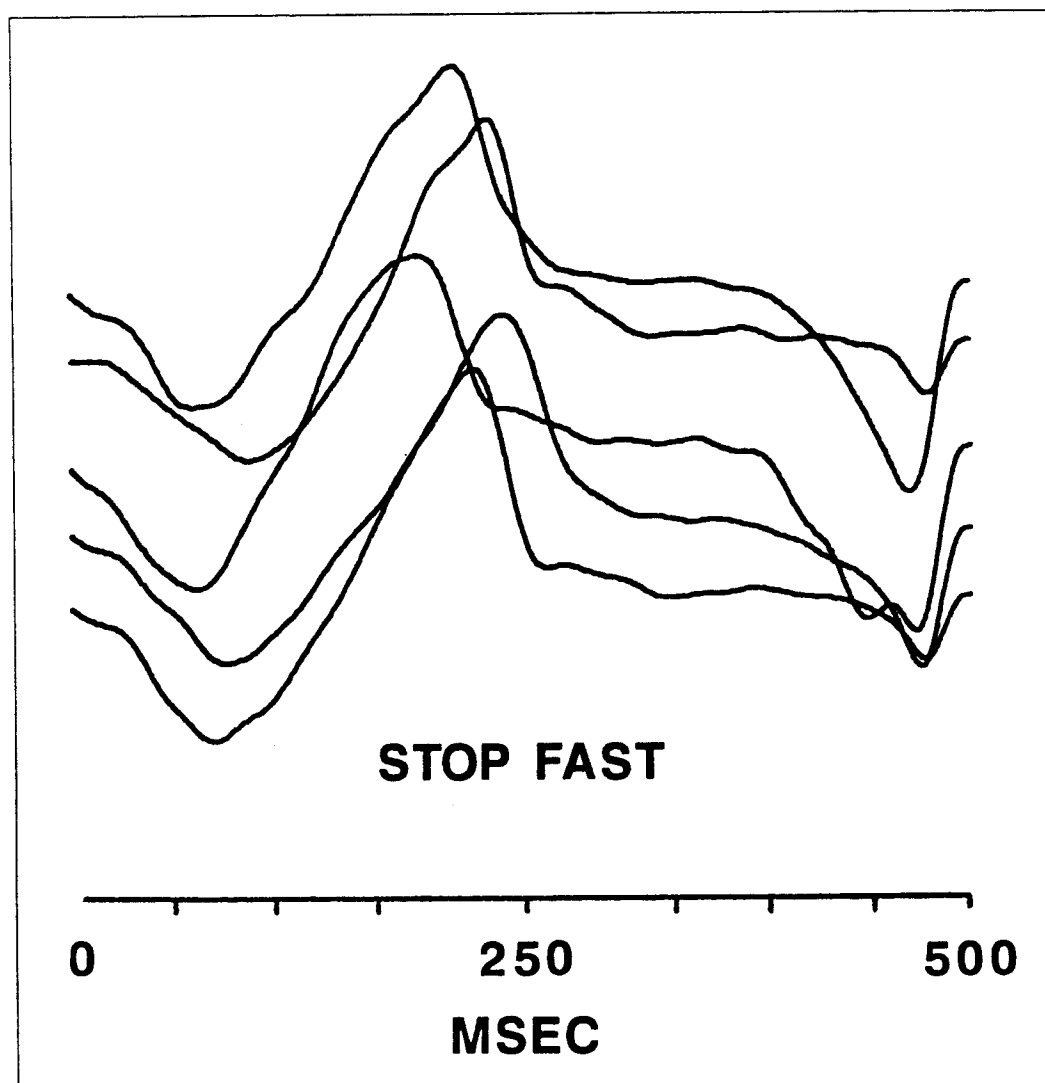
Figure 15-3. Five consecutive velocity traces for one normal subject.

Figure 15-3 shows five consecutive velocity traces for a normal subject. A relative consistency of the peaks in time can be seen, along with a relative smoothness of the trajectory to and from that peak. Figure 15-4 shows five consecutive velocity traces for one apraxic subject. In contrast to the normal traces, there was considerable difference in the timing of the peaks and the smoothness of the trajectory. One means of capturing the differences in these velocity profiles is to count the number of times that the trajectory crosses the zero axis. This measure produces a quantitative method for estimating the smoothness or consistency in time of the velocity produced over the course of the gesture. We have termed these zero crossings dysmetria.
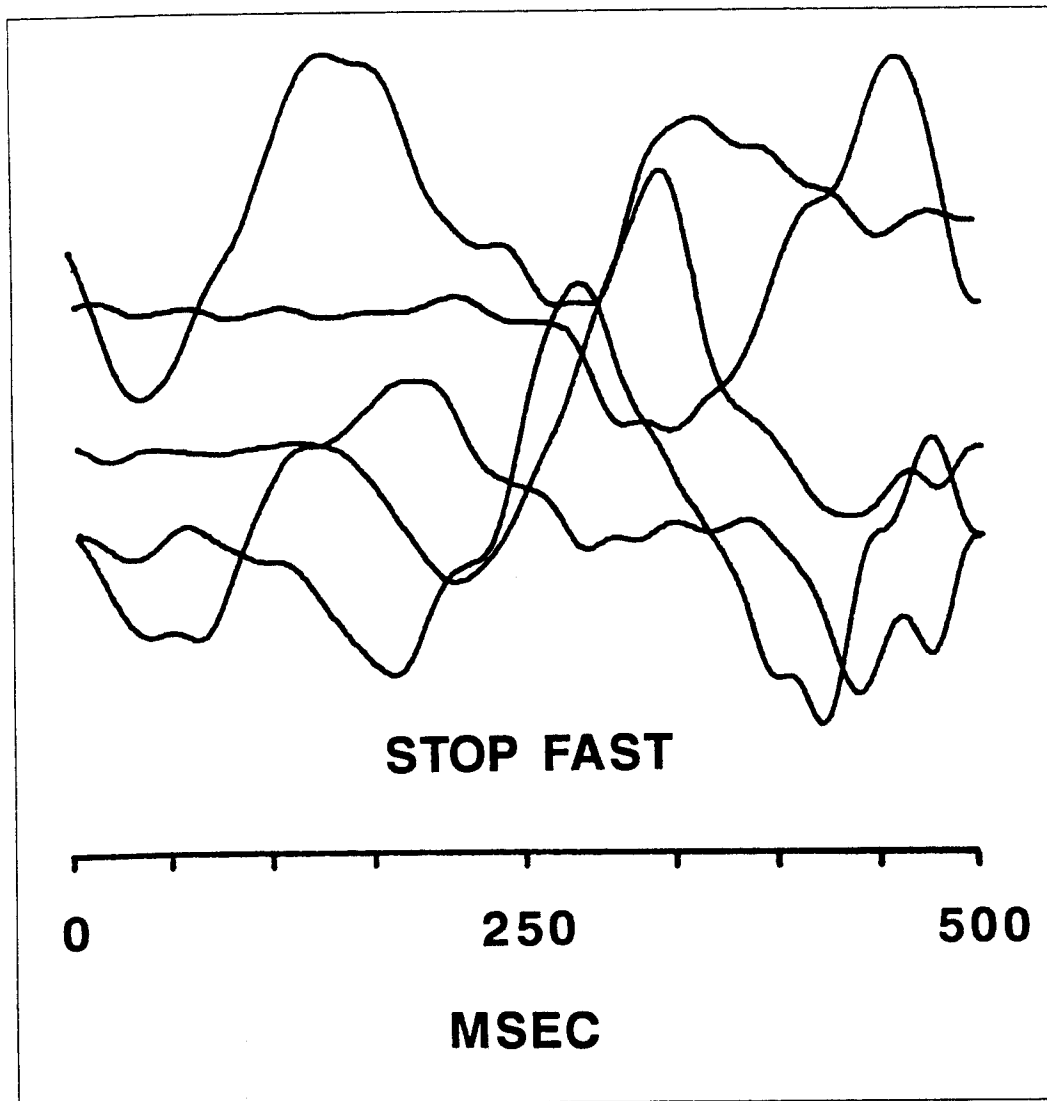
Figure 15-4. Five consecutive velocity traces for one apraxic subject.

DYSMETRIA

The number of velocity changes (zero axis crossings) during the course of the movement were detected and counted using a computer program written for that purpose. The average number of subject and group dysmetrias are summarized in Figure 15-5. It is apparent from this figure that on the average, the normal subjects produced less than half as many dysmetrias as the apraxic group during this gesture. This between group difference was statistically significant at $p \leq .05$. It can also be seen that with the exception of subject A2, the apraxic subjects had considerably larger standard deviations than the normal subjects.
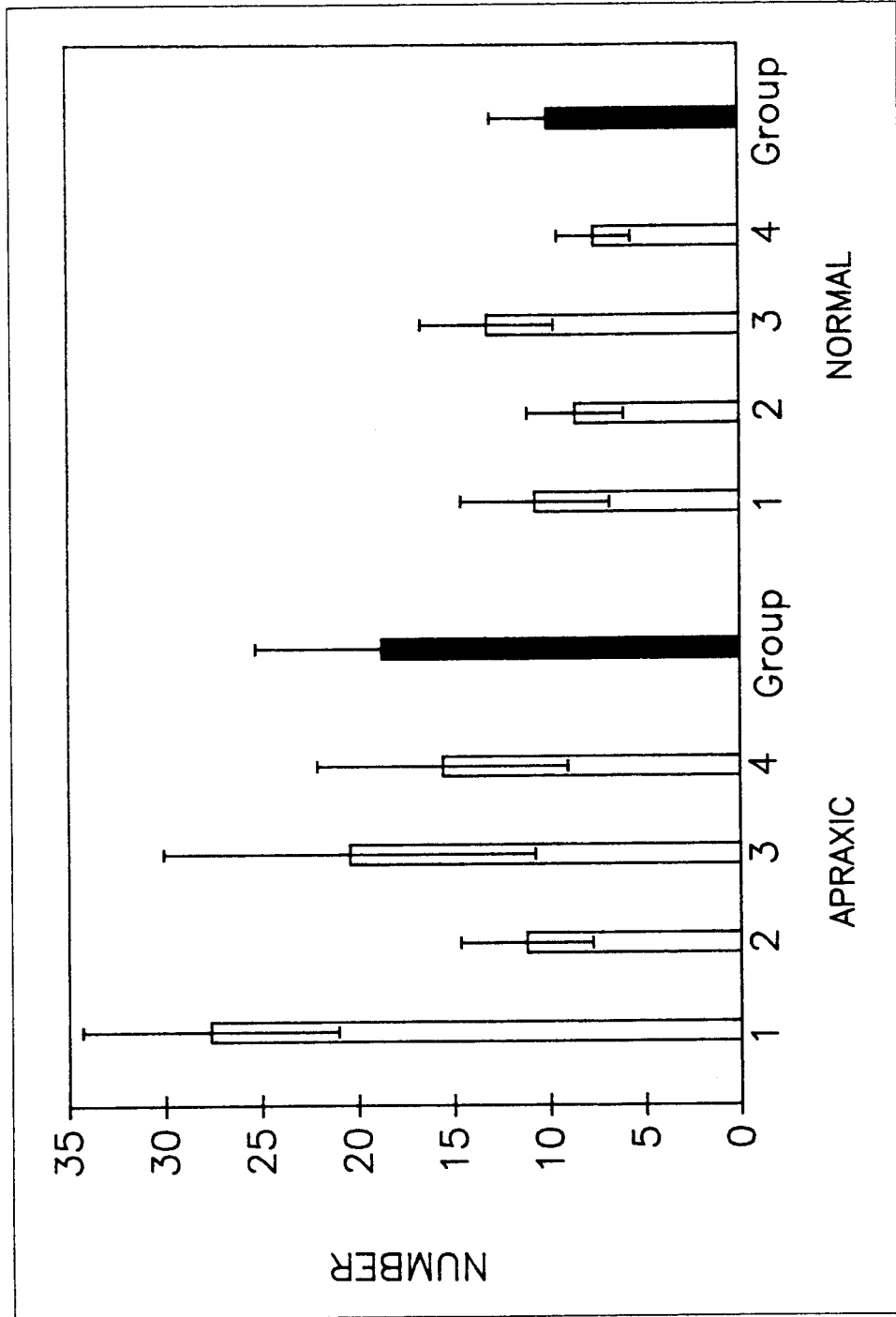
Figure 15-5. Lower lip dysmetria count means and standard deviations (*error bars*) for each subject (*open bars*) and for the apraxic and normal groups (*solid bars*).

Data from three of the four subjects in each of the groups were analyzed for the maximum displacement of the lower lip during the closure phase of the speech gesture (from /a/ to /p/ in "stop"). These data are summarized in Figure 15-6. Results of the between group analysis revealed a significant (p < .05) difference, with the apraxic subjects making greater movement excursions with the lower lip than the normal subjects. In general, the standard deviations were greater for the apraxic subjects. However, as with the other dependent measures, apraxic subject A2 had an average peak velocity and standard deviation that was well within the range of the normal subjects. It should be remembered that A2 also had shorter mean durations and velocities than the other apraxic subjects, and these values were in the ranges of the normal subjects for each of these dependent measures.

To determine if there was a predictable relationship among several of the dependent measures (duration, velocity, dysmetria, and displacement) for each subject group, a series of Spearman Rank order correlation coefficients was computed. Table 15-2 summarizes the results of these correlations. Among the correlation coefficients that were computed, the only one to reach a reasonably high level, accounting for about half of the variance, was the correlation between the duration of the movement and the number of dysmetrias occurring in that speech gesture. All other correlations were low and clinically unimportant. One correlation coefficient that is important to calculate but was not computed was for velocity-displacement. This was not computed because the peak velocity was measured from the greater of either the opening or the closing gesture, and displacement was measured from only the closing phase of the gesture.

DISCUSSION

These results provide the following answers to the experimental questions posed. First, the apraxic subjects were not statistically significantly

TABLE 15-2. SELECTED CORRELATION COEFFICIENTS AMONG THE DEPENDENT MEASURES FOR UTTERANCE "STOP FAST"

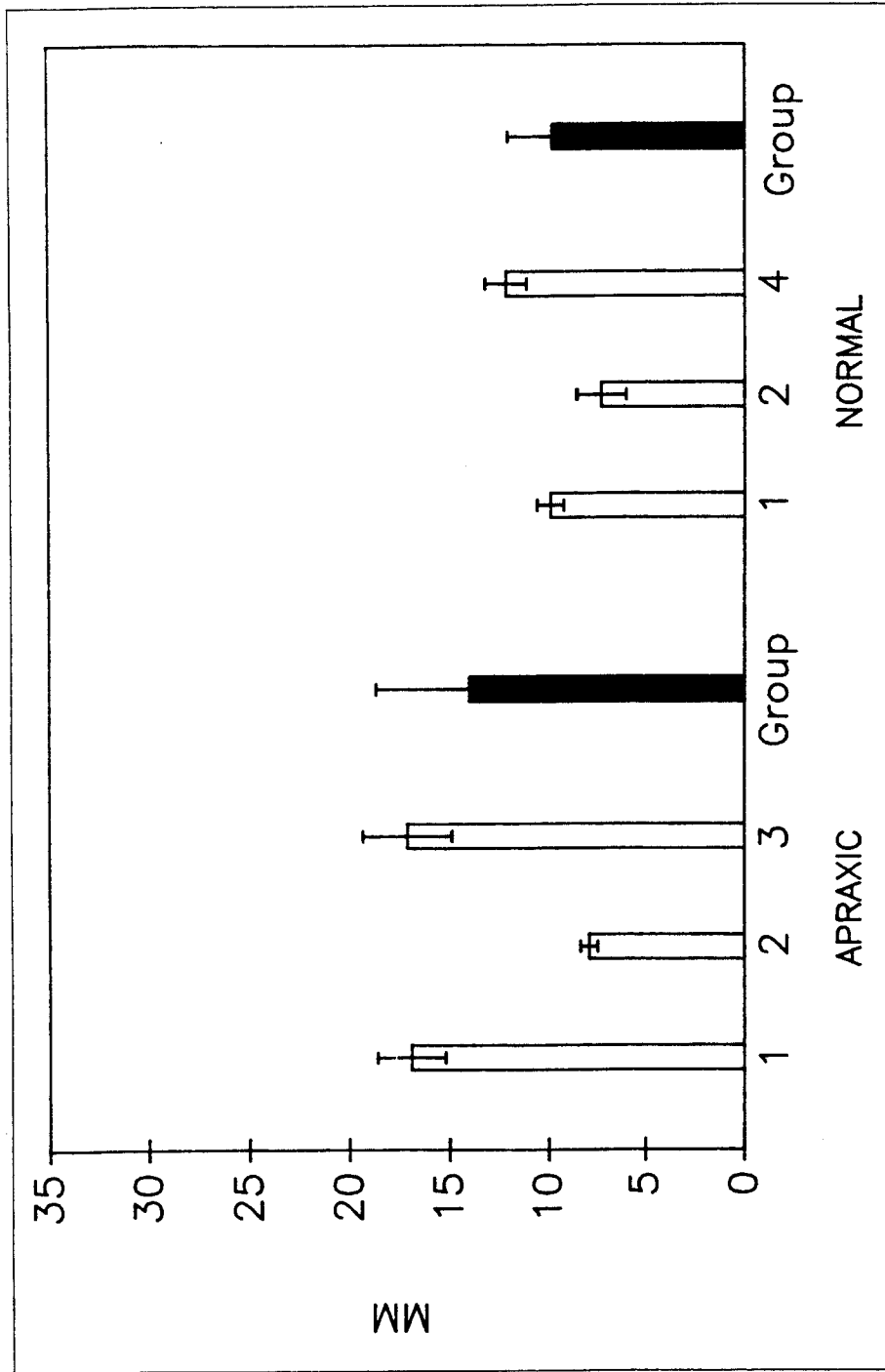|	Groups	
Measures correlated	Apraxic	Control
Duration-velocity	.46	.18
Duration-dysmetria	.70	.53
Velocity-Dysmetria	.14	−.26

Figure 15-6. Lower lip dysmetria count means and standard deviations (*error bars*) for each subject (*open bars*) and for the apraxic and normal groups (*solid bars*).

different from the normal group in terms of peak velocity and thus were not fundamentally slower than the normal group. Like the ataxic subjects reported by Hirose and colleagues (1982), the apraxic subjects in this study were not statistically significantly different in the magnitude of the peak velocities but were more variable in their performance from trial to trial than the normal subjects.

While few velocity studies have been undertaken with apraxic subjects, several such studies have been conducted on normal and dysarthric subjects. In general, these studies have concluded that some types of dysarthria present with articulatory velocities that are within the range of those produced by normal subjects and other dysarthric types that are not. Hirose and colleagues (Hirose, Kiritani, Ushijima, and Sawashima, 1978; Hirose, Kiritani, and Sawashima 1982), for example, have reported that the velocities for amyotrophic lateral sclerosis (ALS) subjects, with presumably mixed spastic and flaccid dysarthria, were slower than for normal subjects. Ataxic dysarthric subjects and subjects with dysarthria secondary to Parkinson's disease, on the other hand, had variable velocities that ranged from slow to normal across repeated productions of a monosyllable. These researchers also stressed the importance of the pattern of velocity variability across repeated productions. In the Hirose and colleagues (1982) study, they reported that ". . . the velocity of lip movements for each consecutive syllable is quite variable and inconsistent in cerebellar ataxia, while in parkinsonism, the velocity declines quickly as the same syllables are produced repeatedly." They interpreted their findings in the ataxic subject with reference to Allen and Tsukahara's model (1974) of cerebrocerebellar function. They suggested that the planning and sensory updating functions of the cerebellar hemispheres and pars intermedia of the cerebellum, along with the planning functions performed by the cerebral association cortex and the basal ganglia account best for the variability of velocities produced in these subjects. The reduced variability of velocity in the ALS subject was also interpreted as support for the motor planning explanation in the ataxic subject. The fact that the apraxic subjects in the current study were not fundamentally slow but took significantly longer to achieve the gesture is cautiously interpreted as support for a movement programming disorder in the sense of the term used by Allen and Tsukahara for the planning and sensory updating functions of the motor programmer. The finding of peak velocities within the normal range are consistent with those of Robin, Bean, and Folkins (in press). While they are consistent with those reported for the single subject by Itoh and colleagues (1980), they are quite consistent with those reported by Itoh and Sasanuma (1987). In this study they reported peak velocities that were inconsistent. They state (pp. 158–159):

> The speed of the articulators of Broca's aphasic patients is not always slow compared to that of normal and Wernicke's aphasic speakers. The performance of Broca's aphasic patients is rather characterized by inconsistency in terms of articulatory velocity .... Coupled with this observation, the finding that the patients with Broca's aphasia do perform normally in the rapid repetition of nonsense monosyllables indicates that their problem is not attributable to paralysis or weakness of the articulatory muscles.

In general, their conclusions regarding the speed of movement is consistent with the conclusion that must be drawn from the present investigation.

The apraxic subjects in the present study took significantly longer achieving the speech gesture than the normal subjects. This extended movement duration could be accounted for by larger movements (greater displacements) and/or by a greater number of movement aberrations during the course of the gesture. Both greater displacements and more dysmetrias were present for the apraxic subjects. It is at this time difficult to determine whether the greater number of dysmetrias occurred as a result of the greater opportunity to vary the course of their movements because of the greater displacement or whether they would have made more dysmetrias than the normal group even with equivalent movement amplitudes. To gain some insight into the relationships among the variables, selected correlation coefficients were computed. A substantively high correlation coefficient was found when the durations were correlated with the number of dysmetrias. While there is a predictive relationship between these two variables, either of the above interpretations remains plausible and awaits additional analyses of other structures and other speech movements from these subjects.

The fact that apraxic subjects moved their lower lip plus jaw to a greater extent than the normal subjects is interesting and could suggest a disorder of spatial programming. However, until simultaneous upper lip movement data have been analyzed to determine the total amount of labial movement and the degree of motor equivalence achieved, these interpretations remain speculative. The finding of a larger than normal displacement is not consistent with the Robin and colleagues (in press) study; however, it is consistent with those reported for two of the five subjects in the Itoh and Sasanuma (1987) study. Our findings are not supportive of the finding that the majority of subject's performance in the Itoh and Sasanuma study had an "... extremely slow lower-jaw peak velocity in the closing transition for some utterances of the /VsV/ and /VzV/ words." While we did not extract the jaw from the lower lip movement in our study, all measurements were made on lower lip plus jaw, and the jaw undoubtedly had an influence on the measurements that were made.

Finally, the nature of the increased number of dysmetrias found in the apraxic subjects is interesting and deserves comment. Dysmetria is a clini-

cal term often associated with movement aberrations seen in ataxic dysarthria. While the dysmetrias were not calculated from the displacement curves, similar aberrations have been found in the velocity profiles of the ataxic dysarthric patient (Hirose et al., 1978). To the degree that these profiles can be explained by the cerebrocerebellar portion of the motor program in the ataxic dysarthric, it is tempting to propose a similar mechanism for those dysmetrias in the apraxic subjects in this study. Given the limited information currently available on this phenomenon and given some preliminary data that we have on the velocity profiles of normal and apraxic subjects on rate controlled utterances, it may be at best speculative and perhaps premature to speculate about mechanisms. Our preliminary data suggest that normal subjects may produce velocity profiles during slow speaking rates that are like the apraxic profiles measured in this investigation produced at their control rate. If this in fact turns out to be the case, it might be that the dysmetrias are an artifact of the slow speaking rate of the apraxic subjects. This slow rate could be caused by a mechanism that is entirely different or perhaps related to the mechanism generating the dysmetria. Further research will surely clarify this very important issue.

ACKNOWLEDGMENTS

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DISCUSSION

Q = question; A = answer; C = comment.

Q. Did I understand you to say that the Sasanuma study was the one study that did not agree with your findings? I want some clarification on that.

A. The first study by Itoh and his colleagues reported the velocities for one subject. They found that the peak velocities were actually decreased in this subject. But on subsequent studies using more subjects, they found exactly what we found.

C. The only point I wanted to make — given that you obviously had variability within English as well — languages do vary pretty markedly in their metrical properties, they are syllable-timed versus different kinds of timing and prosody relationships, and I wonder whether you might not find somewhat different organization of what's slow and what's disrupted in timing as a function of the basic metrical properties of the language.

A. Well, they had normal controls of their own language. I think that would take care of that potential problem. But the point is a good one and I think that there probably are [metrical influences on speech movement velocities], especially as you cross boundaries as we did in our analyses going from a closing to an opening gesture and measuring velocity within all of that. I think that's a real good point. It's a factor that we should all consider. In fact it's such an important factor that I don't think we should have measured all of that. We should have measured the opening *or* closing gesture to reduce those kinds of potential differences that can occur, even within one linguistic system.

Q. I know you ruled out weakness on clinical exam, but I suppose one could ask if that were nonetheless still a potential contributor. I guess the question I have is whether or not the fact that patients had normal peak velocities is evidence that their movements were not influenced by weakness or spasticity, or is my thinking wrong about that?

A. I'm not sure about the relationship between weakness and velocity. I'm not even sure how you define weakness physiologically unless it is defined by force measures. The relationship between velocity and weakness is unclear to me. The kind of a speed problem that I think you are referring to, like in the pseudobulbar kinds of guys, I think we've eliminated that for sure, and I think if we had found lower peak velocities, we would have had to worry that we might in fact

have had spastic subjects in there. I expect spastic people to be slow in terms of peak velocities and the data from ALS, and other pseudo-bulbar patients support that expectation.

Q. One other question, do you have any data on individuals with ataxic dysarthria, and if not, would you care to speculate about what they might look like on these measurements?

A. We have a ton of data on ataxic dysarthrics; however, we haven't looked at it adequately to make sense out of it yet. My equivocation has to do with your question about weakness because some ataxics are weak and some aren't weak. It's not a characteristic of ataxia, but they can be weak. If I have to guess, I'm going t guess that they are going to act like the apraxics on this measure. There are other things, however, on which they will probably perform very differently. If you are just talking about peak velocity, I think that they are going to act like apraxics, which is like normals. The reason is that they [the ataxic speakers] are so bloody variable. Sometimes they are going to be slow and sometimes they are not. There will surely be other kinematic attributes that will differentiate them, but peak velocity probably wouldn't .... It'll be fun to see.

Q. Here's a conundrum. You know the way you collect data may influence the data you collect. Is there any evidence that people may behave differently when their heads aren't strapped in a cage than when their heads are strapped in a cage.

A. I can't think of any evidence on either side of that. In defense of doing this, I think I can say that these guys are strapped in a cage but they are free to move. It isn't a cephalostat. It's a head-held movement transducer that they can bob around in and do all the things they normally do, and all it does is keep the movement transducers moving with their head. So, it is not as invasive as a cephalostat. But, yes, even having a movement transducer affixed to your lip with glue may make you do things differently — maybe.

C. I don't know of any other way to do it.

A. There are other devices for measurement, such as the cell spots or the light-emitting diode system that Itoh and lots of other people are using. There are even better ways of doing this kind of kinematic analysis such as the electromagnetic system that Joe Perkell is working on here in the states and Paul Shonle and some other people in West Germany have developed. Each technique is still invasive. You have to do something to put the cell spot or the radio receiver on the structure to be measured, and that could make them perform differently than they would if it were not there.

Q. Do you see any difference with your data over time? Do people seem to settle down physiologically, say reduce variability?

A. I think that they are terribly variable all the time. One thing that we did do to try and take care of that is that we put them in it (the head-held movement transduction system) and then we would talk to them a long time, and we would do a lot of tasks (e.g., calibration) before we would start collecting the data. But I don't know how long adaptation takes. We know that if we put a false palate in someone's mouth, it takes about 48 hours or longer before they adapt to it. I have no idea! Perhaps some of the speech scientists in the audience would care to comment on that.

C. Or fatigue in the interaction between the two.

A. Yes, yes, I don't know. That's a very good question.

Q. I think you said that basically peak velocities weren't different, and I heard you raise a little bit of caution about the possibility that treatments that are aimed at reducing rate may not be the best thing in the world. I also heard you say that the clinical adage that we have held to our heart may be true, and that is when you mess with rate, you mess with other things. Seems to me that perhaps we ought to think about the perceptual level as well and if we manipulate rates, or if that's what we think we're doing, and if they sound better when we do that, shouldn't we keep doing it?

A. Yes, if the goal is to make someone slow down. But if that slowing down interferes with other things down the line, that we just haven't been careful enough to measure yet, then we may need to do it cautiously. I'm not sure where to go with it. Let me tell you one thing if I'm not out of time, because this is, I think, potentially interesting and important. We're now looking at these kinds of movements in normal speakers, ataxic dysarthrics, and some conduction aphasic speakers during the production of slow speech. While this is preliminary, it looks like (and I have to give the credit to Scott Adams, one of our research assistants) during slowed speech produced by the normals, the velocity traces look remarkably like the apraxic subjects during their normal rate productions. The velocity traces become very different from their [the apraxic] normal velocity traces. That doesn't necessarily translate to bad speech though. It's the relationship between kinematics or acoustics and the perceptual event, and the final arbiter is what you hear. That's always going to be the final arbiter. I guess the answer is yes.

Q. You know I have a lot of respect for your work, and listening to Kevin's comment, one of the things that I wonder is if maybe some of

this slowness might be in compensation for some neuromotor deficit that we are not picking up, so maybe the things you might be measuring kinematically are central adjustments that are made by the patient consciously or unconsciously in compensation for a neuromotor deficit. The one that pops to mind that would be common with UMN [upper motor neuron] lesions would be lack of discrete movement of the various articulators, and what I'm wondering, if you'd care to comment on, are you measuring the direct affect or a compensation affect?

A. Yes. It's a perfectly valid question, and it haunts us all the time. The obvious answer is that I have no idea what it is. I suspect that it might be some of both. The fact that one of the subjects looks like the normals, but is still apraxic, has to tell us something, and we just can't ignore that.