Simultaneous Perceptual-Physiological Method for Studying Apraxia of Speech

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INTRODUCTION

In the last ten years, a considerable effort has been expended upon descriptions of apraxia of speech. These investigations have focused on auditory-perceptual and acoustic manifestations of this disorder with occasional analyses of articulatory movement and speech muscle activity. Typically, however, only one of these variables has been observed in any given investigation. The rationale underlying this single-measure approach appears to reflect the traditional wedding cake conceptualization of the speech production process, shown in Figure 1. This representation carries with it certain assumptions, the major one being that intended phonetic targets, at a phonological/linguistic level, yield stereotyped and consistent muscle activities, movements and vocal tract shapes at the periphery. Given this assumption, many investigators have interpreted single measures in relation to multiple levels of this overall process. However, as our sophistication concerning normal speech motor programming and control has increased, it is apparent that this simple model and the associated critical assumptions are, at best, questionable.

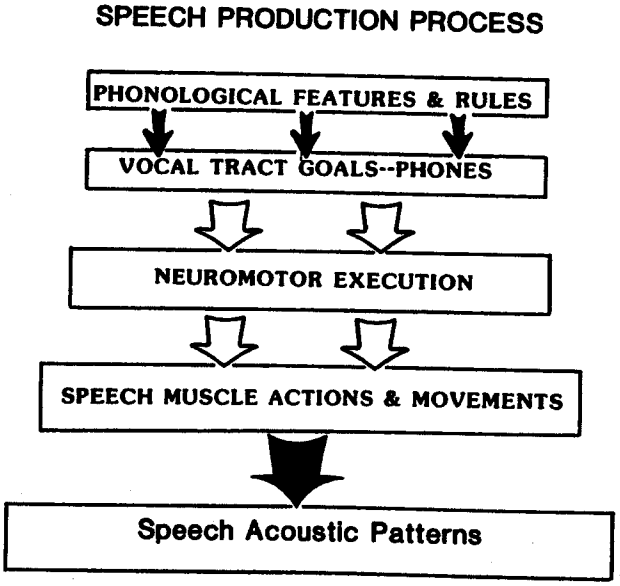
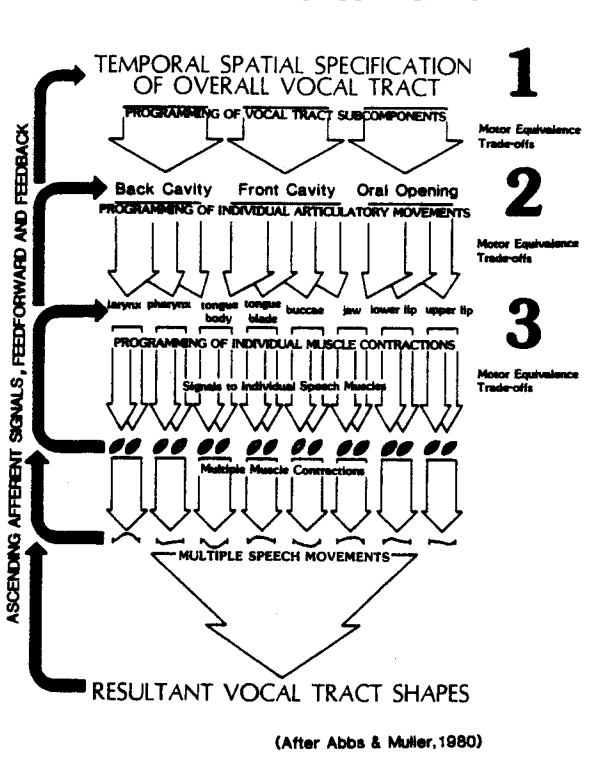


Figure 1. Traditional multilevel representation of the speech production process.

In developing the rationale for the alternate approach to be taken in the present paper, it is useful to document this point more explicitly. Based upon observations in our laboratory and by others, we now know that

speech neuromotor control does not involve specification of individual muscle contractions or individual articulatory movements at the phonological or phonetic level. A more recent conceptualization, shown in Figure 2, incorporates one of the major principles of speech and limb motor control, the phenomenon of motor equivalence (cf. Hebb, 1949; Lacquaniti and Soetching, 1981; Morasso, 1981).



(After Abbs & Muller, 1980)

Figure 2. Representation of the speech motor control process illustrating the phenomenon of motor equivalence.

The operation of motor equivalence affects traditional observations of apraxia of speech because it suggests that there is not a stereotypic or predictable relationship between phonological goals and the details of motor execution. This can be illustrated quite simply. In Figure 3 are shown three productions of the utterance /aba/ that are perceptually, acoustically, and aerodynamically equivalent. However, the movements and muscle contractions are different from one production to another. This illustration shows complementary variation between upper and lower lip gestures to achieve equivalence. In like manner, it has been observed that, via motor equivalence control, individual speech muscle contractions, individual speech movements, and individual vocal tract shapes are implemented in a variable, but complementary fashion to produce perceptually-invariant acoustic signals (cf., Abbs, 1979; Abbs and Cole, 1982; Abbs and Gracco, 1981; Hasagawa, Fletcher and McCutcheon, 1978; Ladefoged, DeClerk, Lindau and Papcun, 1972; Lindau, Jacobson and Ladefoged, 1972).

The specific implications of these recent observations for measuring normal and disordered speech production systems suggest that 1) productions judged equivalent by auditory-perceptual or acoustic analyses are likely to be very different in terms of underlying patterns of muscle activity, movement or specific vocal tract configurations, 2) single measure analyses are insensitive to these motor equivalence control variations, and 3) the degree

of single measure insensitivity is perhaps greatest in disordered speakers who invoke idiosyncratic compensatory adjustments to offset their nervous system abnormalities.

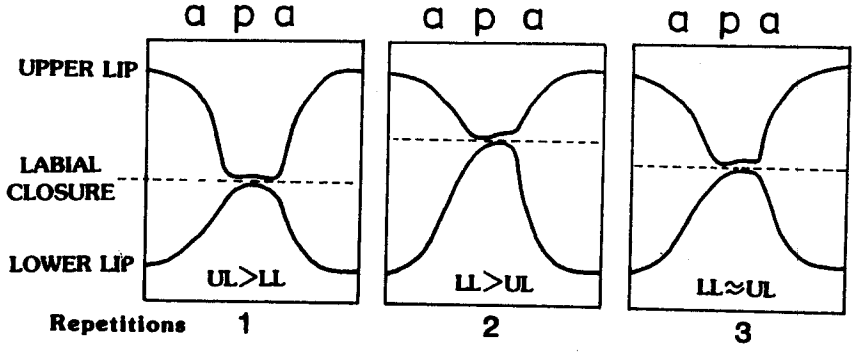


Figure 3. Illustration of speech movement (and presumed muscle contraction) variations for three utterances that are acoustically, aerodynamically and perceptually equivalent.

Current concepts of speech neuromotor programming and control suggest that we must make multiple, simultaneous observations in order to specify the dysfunctions underlying this disorder. The need to observe both movements and muscle activity is particularly important given the classical definition of apraxia of speech, which refers to impairments at these levels of system output (Darley, Aronson, and Brown, 1975). For these reasons, the present investigation was conducted.

METHODS

Subjects in this study were three apraxic and three age-matched normal adult males. Subjects were to be considered apraxic speakers if their spontaneous speech was characterized by the following classical patterns 1) effortful, trial and error groping articulatory behavior, 2) dysprosody throughout segmentally fluent as well as dysfluent productions, 3) evidence of frequent sound substitutions, 4) inconsistency in the presence and nature of errors on repeated productions of the same utterance, 5) difficulty initiating utterances, and 6) imitative speech as good as or better than spontaneous speech. The identification of apraxic subjects was confirmed by three experienced aphasiologists. In addition, subject selection criteria included information obtained from extensive diagnostic testing, including speech, language, neurological, orofacial, audiological and orosensory evaluations. These multiple tests showed that our three apraxic subjects were not dysarthric, and signs of aphasia were minimal or absent (Table 1).

Eight sentences heavily loaded with bilabial and labio-dental sounds were the experimental utterances. Subjects were asked to repeat each utterance a total of ten times. A headmounted movement transduction system (Barlow, Cole and Abbs, in press) was used to monitor inferior-superior movements of the upper lip, lower lip and jaw in the midsaggital plane (Figure 4). This transduction device is lightweight and stable. It provides precise online observations without head restraint and allows for simultaneous observation of speech muscle activity. Bipolar hooked-wire

Table 1

Comprehensive Profile of Subject Characteristics

Measure	Groups					
	Apraxic (N = 3)			Normal (N = 3)		
	A_1	A_2	A_3	N_1	N_2	N_3
Age (Years)	59.11	75.6	56.0	58.6	69.0	55.0
Etiology	Surgical	Vascular	Vascular	--	--	--
Duration (Months)	16	29	64	--	--	--
Judged Severity	Mild	Moderate	Moderate-Severe	--	--	--
PICA Scores						
Overall	11.83	13.08	13.36	14.32	14.2	13.77
Gestural	13.71	14.625	14.4	14.9	14.8	14.79
Verbal	13.95	12.8	12.35	14.425	14.825	14.05
Graphic	7.9	11.2	12.65	13.63	12.98	12.23
RTT Score						
Overall	12.69	14.34	13.56	14.38	14.48	14.89
Oral Sensory Threshold Means						
Touch (.1 mg) [1]						
Upper Lip	2.12	2.7	2.52	2.39	2.36	2.36
Tongue Tip	2.41	2.57	2.28	2.12	2.12	1.65
Tongue Blade	2.36	3.22	3.71	3.69	2.83	2.44
Two-point (mm)						
Upper Lip	1.65	1.68	1.45	1.8	1.5	1.21
Tongue Tip	1.05	1.25	1.375	1.75	0.875	1.25
Tongue Blade	2.5	1.5	1.875	2.25	1.125	1.38
Hearing						
Pure Tone Average						
Left	18	8	13	17	18.3	8.3
Right	13	6.6	11.6	17	8.3	5
Speech Discrimination						
Left	DNT [2]	94%	78%	90%	80%	DNT [2]
Right	DNT	94%	78%	90%	96%	DNT

Table 1 (continued)

Measure	Groups					
	Apraxic (N = 3)			Normal (N = 3)		
	A_1	A_2	A_3	N_1	N_2	N_3
Neurological and Other Impressions	1. physiologic tremor 2. flattening of left nasolabial fold 3. mild aphasia	1. moderate oral nonverbal apraxia	1. mild oral nonverbal apraxia	--	--	--

[1] Refer to Semmes et al. (p. 61, 1960) for conversions from Log_{10} Force (.1mg) to Force (gm) and Diameter (mm).

[2] DNT = Did Not Test. It was not possible to complete speech discrimination testing, however, pure tone thresholds and clinical judgement of speech discrimination were compatible with those of the other subjects.

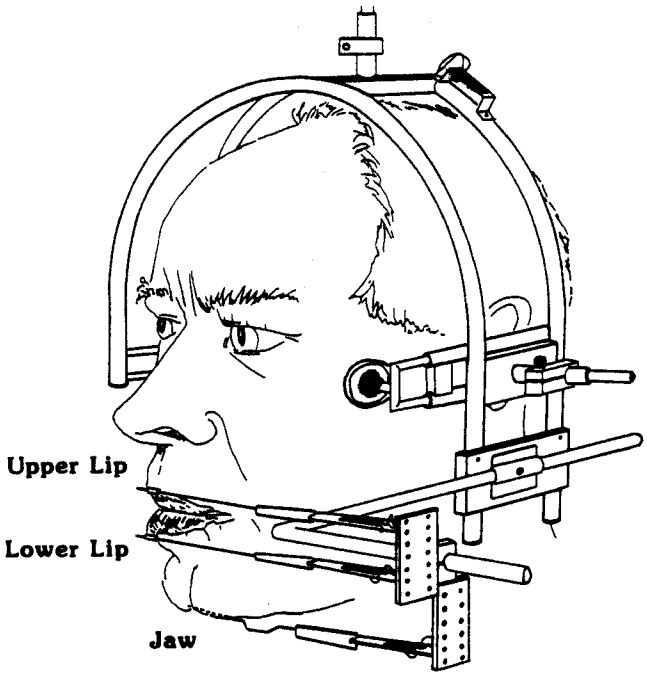


Figure 4. A head-mounted labial-mandibular movement transduction system. (Adapted from Barlow, Cole, and Abbs, in press.)

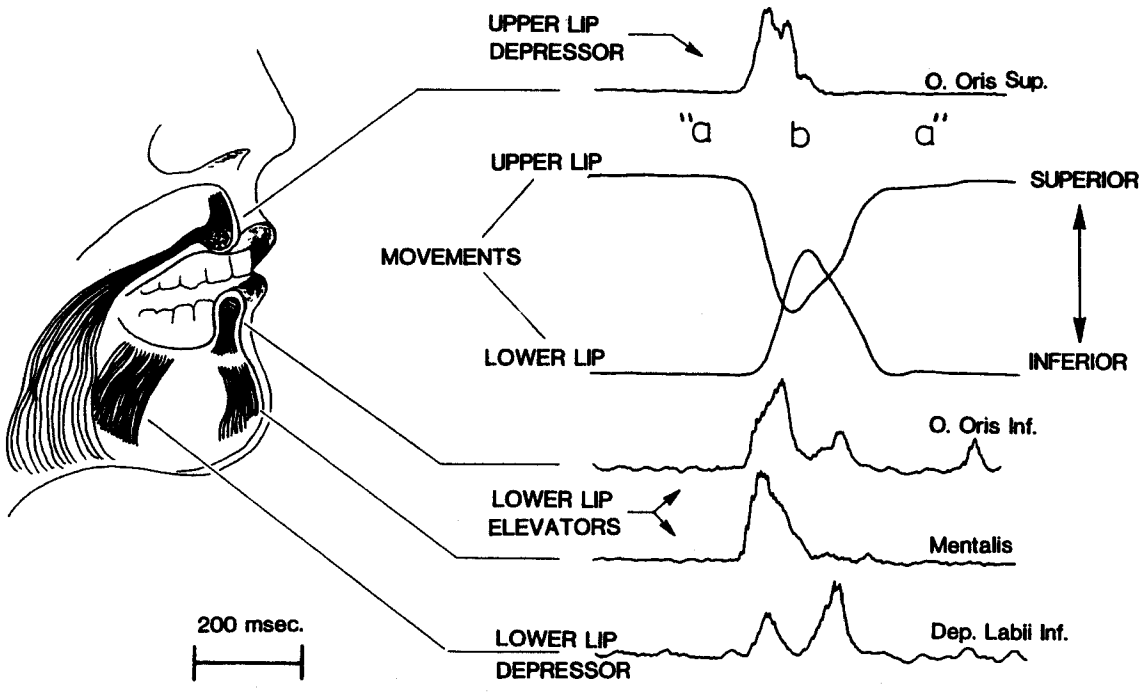


Figure 5. A sample of upper lip and lower lip movement (inferior-superior) along with muscle activity patterns from four facial muscles.

electrodes were used to record the electrical activity of four orofacial muscles: orbicularis oris superior (OOS); orbicularis oris inferior (OOI); mentalis; and depressor labii inferior (DLI). OOS, OOI and mentalis act to close the lips, whereas DLI functions to open the lips by pulling the lower lip down (Figure 5). We also had an accelerometer attached over the thyroid cartilage to record the onset of voicing, and a microphone positioned to record the airborne speech signal.

The analysis proceeded in two stages. First, each tape was transcribed and reviewed at least two times to assure accuracy of transcription. All utterances were subjected to auditory-perceptual evaluation by two clinically sophisticated listeners. Errors were categorized and defined as in previous investigations by Johns and Darley (1970), Trost and Canter (1974), and LaPointe and Johns (1975). For the physiological stage of the analysis, we extracted those speech errors involving labio-mandibular-laryngeal productions, which included 25% of all segmental errors. The associated physiological records were then examined in detail to detect possible cause and effect relations between the judged labio-mandibular errors and potential abnormalities in the underlying movement and EMG patterns.

RESULTS

This analysis allowed us to observe the physiological abnormalities associated with judged speech errors, and from these to address some of the issues and theories surrounding apraxia of speech. With this multimeasure, multistage analysis it was possible to minimize the limitations of single-measure observations. However, given the scope of this paper we will present and discuss only a few examples to demonstrate the efficacy of this approach.

Before discussing characteristics of apraxic movement and muscle activity, we would like to review a typical normal pattern. Figure 6 shows a representative example of a normal subject's production of "Buy Bobby a poppy." From top to bottom the signals are (1) accelerometer, (2) upper lip movement, (3) lower lip plus jaw movement, (4) jaw movement alone, (5) EMG orbicularis oris superior, (6) EMG orbicularis oris inferior, (7) EMG depressor labii inferior, and (8) EMG mentalis. As indicated by the arrows on the accelerometer signal, there is voice onset after the initial /b/ release and appropriate voicing shutdown during the two voiceless bilabials in the final word "poppy." The upper lip, lower lip and jaw move smoothly and synergistically toward oral closing for the four bilabial stops and toward oral opening for the five vowel sounds. This kind of smooth temporal-spatial coordination between the upper lip, lower lip and jaw is seen in all normal subjects.

The upper lip trace shows an indentation which is the result of the upper lip being pushed slightly upward by the lower lip. This indentation serves as an indication that labial contact and closure were achieved. Concomitant muscle activity demonstrates appropriately discrete EMG bursts in OOS, OOI and mentalis for bilabial closure. Conversely, there is total or near total shutdown in these three muscles during labial opening, at which time the antagonist muscle, DLI, is discretely active. This reciprocal on-off pattern, labelled on the record, demonstrates normal temporal coordination in these multiple speech muscles.

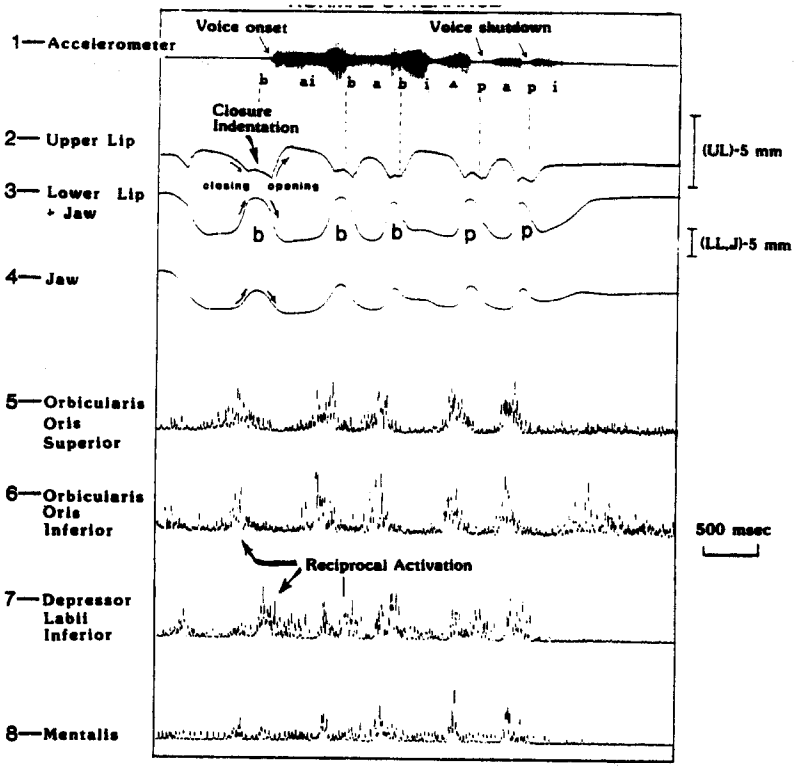


Figure 6. Muscle activity and labial-mandibular movement signals obtained from a normal subject for one of the test utterances.

The apraxic subjects manifested a number of movement and muscle control abnormalities in comparison to the normal subjects. These manifestations included: (1) antagonistic muscle co-contraction, (2) continuous, undifferentiated EMG activity, (3) muscle activity shutdown, (4) movement without voice, (5) movement dyscoordination, (6) addition and (7) groping, each of which was objectively defined. Such patterns are not seen in normal subjects. This pattern of abnormalities also was distinct from patterns seen in Parkinson's, flaccid, ataxic or spastic dysarthric subjects (Netsell and Abbs, 1977; Hunker et al., 1982: Barlow and Abbs, 1979; Abbs et al., in press).

Figure 7 shows a production of the utterance "Buy Bobby a poppy" by one of the apraxic subjects. Although this utterance is one of the more fluent apraxic utterances, several physiological abnormalities are apparent. The EMG signals at the bottom of the record show that the depressor muscle is co-contracting abnormally with the orbicularis oris inferior and mentalis muscles, particularly for the first three bilabial stops. There is an abnormal shutdown of all four muscles in the middle of the utterance, during which time the movement effectively ceases. The movements also show subtle control disturbances as well, particularly for the second and third bilabial stops. First, the degree of labial closure, as reflected in the amount of upper lip indentation, appears reduced for the first two closures. Secondly, and more importantly, the third attempt at bilabial closure is

incomplete--there is no indentation of the upper lip. It is apparent that the amount of jaw elevation was progressively reduced from the first bilabial stop to the third. One might suggest that this apraxic subject began to lose control during the third bilabial stop due to antagonist co-contraction and the reduction in lower lip movement, and stopped the utterance completely. These patterns of antagonist co-contraction were observed in 35% of all apraxic speech utterances and often co-occurred with muscle shutdown, which occurred in 19% of the apraxic speech utterances. Finally, in this apraxic utterance, inspection of the accelerometer signal suggests that this subject is devoicing voiced stops. Although this example might be judged, via auditory-perceptual analysis, to be segmentally normal, no bilabial stops are produced normally. Rather, these patterns reflect a problem of temporal distortion in all the muscle actions along with parallel problems in spatial coordination of multiple movements.

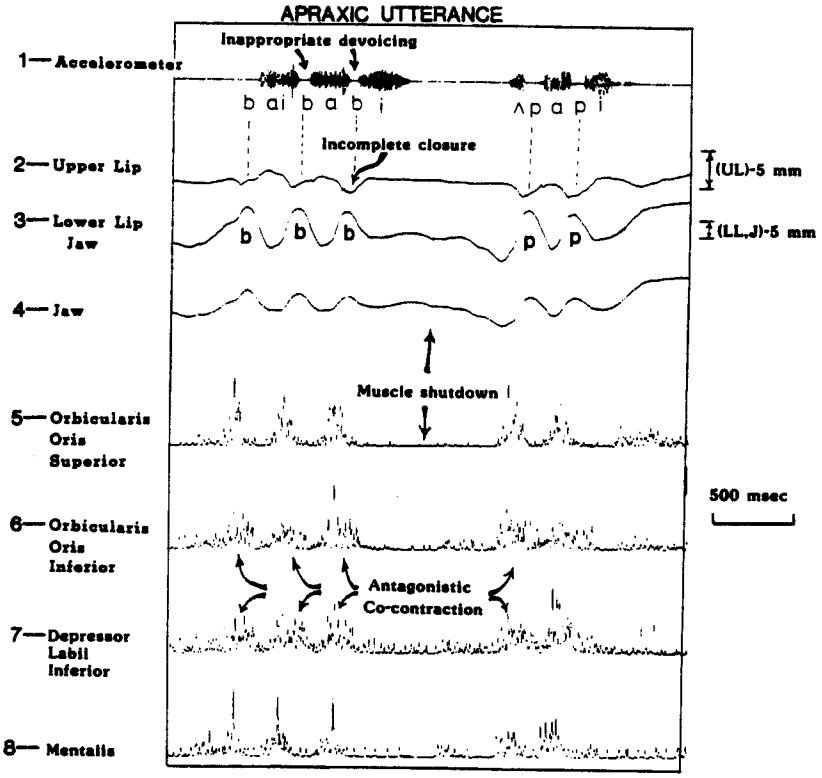


Figure 7. Muscle activity and labial-mandibular movement signals obtained from one of the apraxic subjects for one of the test utterances.

Figure 8 shows movement and muscle activity from another apraxic subject during one of his more dysfluent productions. Again, a number of physiological abnormalities are apparent. The production by this apraxic subject shows undifferentiated activity in both the mentalis and orbicularis oris superior muscles. Discrete bursts, as are seen in normal subjects, are absent. There is also a degree of antagonistic co-contraction between the lower lip depressor muscle (DLI) and the lower lip elevators (OOI and MTL). There is an extended period of groping that precedes the production of the

first bilabial stop in "build." There is a substantial degree of dysco-ordination between the upper lip, lower lip, and jaw. In contrast to normal upper lip, lower lip, and jaw synergy, these movements by an apraxic subject appear to be controlled almost independently. The upper lip moves toward closure five times. By contrast, the lower lip moves four times toward the same closure while the jaw elevates two or three times, in a slow, graded fashion. This kind and degree of dyscoordination is not seen in ataxic subjects, nor in any other dysarthric group (Netsell, Daniel and Celesia, 1975; Barlow and Abbs, 1979; Abbs, Barlow and Cole, 1978; Netsell and Abbs, 1977; Hunker, Abbs and Barlow, 1982; Abbs, Hunker and Barlow, in press). This subject apparently selected the proper movements, but was unable to accomplish the multi-structure, temporal-spatial programming necessary to produce the bilabial closure. From these kinds of patterns, seen in 31% of the apraxic utterances, it would appear that there is a substantial degree of dyscoordination associated with apraxia of speech, much of which would not be discernible with only auditory-perceptual or acoustic analyses.

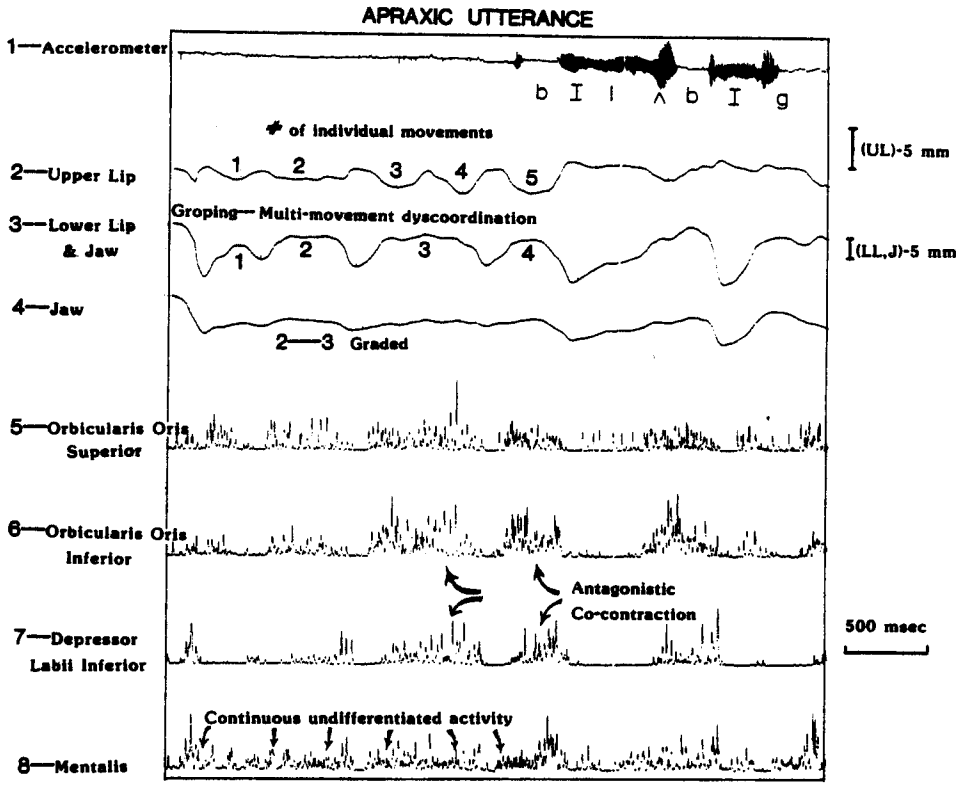


Figure 8. Muscle activity and labial-mandibular movement signals obtained from one of the apraxic subjects for the first portion of the utterance "Build a big building".

CONCLUSIONS

These data, and 238 other utterances from our three subjects, suggest certain new perspectives on apraxia of speech and support our rationale for using this method. While full explication of these observations is outside

the scope of this paper, we would like to share with you some of the conclusions we feel are warranted.

First, it appears necessary to redefine apraxia of speech to reflect the fact that there is a substantial degree of dyscoordination between speech muscles and speech movements. We define coordination as the control over multiple motor outputs to insure their contribution to common temporal-spatial goals. The dyscoordination seen in apraxic speakers is different in degree and kind from that seen in our parallel observations of ataxic dysarthrics (Abbs et al., 1978; Abbs et al., in press).

Second, these data strikingly illustrate the point we made at the beginning of the paper, namely, if one is dealing with a disorder where the impairment is potentially one of movement and muscle control, it may be impossible to discern the underlying neuromuscular pathophysiology without direct observations of those physiological variables. For example, many aberrations are silent, while others are perceived as normal, perhaps because of compensatory behaviors that temporarily overcome the neuromuscular abnormality.

Finally, we would like to make a comment about the nature of the segmental errors that we observed. Traditionally there has been disagreement as to whether or not these errors represent a so-called phonological selection problem or a motor programming problem. Ninety-five percent of all substitution and addition errors were produced with one or more accompanying neuromuscular abnormalities of the forms described here. In other words, these were not fluent substitutions, as might be expected if apraxia of speech were a phonological selection problem; rather, we suspect that these segmental errors were due to neuromotor execution and programming aberrations.

We feel that this approach offers important advantages over single-measure analyses. The benefit of focusing on the physiology of the disorder at multiple levels is twofold. Such a focus (1) reduces the number of questionable inferences or potential errors in interpretation that may occur with more global levels of observation and (2) allows one to obtain a characterization of the nature of apraxia of speech with measures that more directly reflect nervous system function and dysfunction. This approach, if applied carefully, should lead to a more refined understanding of a disorder that has been subject to controversy and ambiguity.

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