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REPRODUCIBILITY AND VARIABILITY OF SPEECH MUSCLE ACTIVITY IN ATHETOID DYSARTHRIA OF CEREBRAL PALSY

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Athetoid dysarthria is thought to result from involuntary movements which are variable and irregular in nature. In this study, electromyographic (EMG) activity recorded from six speech muscles was quantified during repetitions of a test sentence by normal and athetoid adult subjects. In the athetoid subjects the articulation of the test sentence was disrupted intermittently by involuntary activity which usually occurred in the time intervals between the syllables in the test sentence, rather than during articulation of the syllables themselves. The EMG activity associated with each syllable in the test sentence was partitioned into reproducible and variable components. The ratio of the reproducible component to the variable component—the signal-to-noise ratio—did not differ significantly between the two subject groups. In the athetoid subjects, however, the reproducible component of the EMG activity was grossly abnormal. We concluded that this abnormal voluntary activity, rather than variable involuntary activity, was the primary cause of athetoid dysarthria.

In both the speech literature and the neurological literature it is agreed that athetosis is characterized by abnormal involuntary movements. These involuntary "athetoid" movements are described as variable, irregular, and even random. This is consistent with neuropathological accounts of involuntary movements in general as "release phenomena" due to loss of normal inhibitory activity (Boszormenyi, 1970; Brain, 1977; Bucy, 1942; Cooper, Upton & Amin, 1982; Denny-Brown, 1962; Hagbarth, 1974; Hassler, 1980; Herz, 1944; Jung & Hassler, 1960; Martin, 1968; Spiegel, 1980; Stanley-Jones, 1956). The involuntary movements of athetosis are exaggerated by, and perhaps only occur with, attempts at voluntary activity (Brain, 1977; Clement & Twitchell, 1959; Denny-Brown, 1962; Foley, 1983; Hallett & Alvarez, 1983; Holt, 1965; Koven & Lamm, 1954; Lance & McLeod, 1981; Paine & Oppe, 1966; Spiegel & Baird, 1968; Twitchell, 1961; Wilson, 1925; Yahr, 1972). They are said to be superimposed upon, to distort, or even to replace voluntary movement (Duvoisin, 1972; Koven & Lamm, 1954; Levitt, 1982; Wilson, 1925; Yahr, 1972). Indeed, some authors think these athetoid movements may be virtually the only manifestations of motor activity in patients in whom athetosis has existed since infancy (Denny-Brown, 1962; Hankinson, 1973; Spiegel & Baird, 1968), as is the case in cerebral palsy. Athetoid dysarthria is thought by most authors to result from such involuntary movements (Brain, 1977; Darley, Aronson, & Brown, 1975; Ingram & Barn, 1961; Netsell, 1975; Paine & Oppe, 1966; Rosenbek & LaPointe, 1978; Spiegel & Baird, 1968).

Recently, we have had reason to question some of these views of athetosis. In particular, the notion that dysarthria in athetoid cerebral palsy is due to the involuntary movements of athetosis was not supported when the patterns of EMG activity recorded from lip, tongue, and jaw muscles of normal and cerebral-palsied adult subjects were examined (Neilson & O'Dwyer, 1981). That study, however, like most of the literature on athetosis, was limited by qualitative, as opposed to quantitative, data observations. The purpose of the present study was the quantification of these earlier observations and their evaluation in the light of existing views of athetosis in cerebral palsy. Accordingly, a more detailed account of some of these existing views is presented.

Descriptions of the abnormal movements of athetosis are not always in complete agreement. However, the consensus of opinion drawn from all sources cited here is that athetoid movements are relatively slow and sustained and are sinuous and writhing in nature. The slow sustained movements of athetosis are frequently contrasted with the continuous flow of rapid, unsustained, discrete and jerky movements of chorea (Brain, 1977; Denny-Brown, 1962; Herz, 1944; Lance & McLeod, 1981; Spiegel & Baird, 1968; Yahr, 1972). However, the speed of movement may not be a reliable factor. Rigidity, or spasticity, which commonly accompanies athetosis in cerebral palsy (Carpenter, 1950; Cooper, 1969; Low, 1972; Molnar, 1973; Poirier, Filion, Langelier, & Larochelle, 1975) may slow the movements (Denny-Brown, 1968; Yahr, 1972), but rapid movements may occur as well (Bucy, 1942; Calne & Eisler, 1979; Denny-Brown, 1968; Duvoisin, 1972; Spiegel & Baird, 1968), perhaps due to an absence of hypertonicity (Denny-Brown, 1968; Herz, 1944; Spiegel & Baird, 1968; Yahr, 1972). The lack of pattern in individual patients' movements (Denny-Brown, 1968) or the coexistence of choreiform movements (Brain, 1977; Calne & Eisler, 1979; Lance & McLeod, 1981; Paine & Oppe, 1966) has resulted in the use of hybrid labels such as choreoathetosis. When hypertonicity is present, the movements are usually described as athetoid (Martin, 1968), but some authors emphasize that muscle tone may fluctuate between *hypo-* and *hyper-*tonicity (Bobath, 1966; Brain, 1977; Carpenter, 1950; Kent & Netsell, 1978; Narabayashi, Nagahata, Nagao, & Shimazu, 1965; Spiegel & Baird, 1968). Indeed, a state of fluctuating muscle tone has been suggested as the basis of

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the involuntary movements (Bobath, 1966; Narabayashi et al., 1965; Netsell, 1975; Perlstein & Shere, 1946).

With regard to both chorea and athetosis, Wilson (1925) stated that "a typical case is easy of recognition, though to define the symptom is not equally simple" (p. 215). This problem of definition remains. Where such definition is attempted in the literature the movements of athetosis are usually described as being one or more of the following: variable, irregular, purposeless, bizarre, unpatterned, uncontrolled, nonrepetitive, ever-changing, inconstant, adventitious, and even random. The variability has been said to apply to the amplitude, speed, timing, sequence, and location of the movements (Bucy, 1942; Koven & Lamm, 1954; Marshall, 1968; Richardson, 1969). As noted by Narabayashi et al. (1965), the clinical definition has been based primarily on simple visual description. Clearly, there is a need for objective documentation of the amplitude, velocity, and frequency characteristics of athetoid movements.

Early attempts at objective analysis of athetosis emphasized simultaneous, irregular, and poorly correlated activity of agonist and antagonist muscles and a lack of reciprocal inhibition (Herz, 1944; Hoefer & Putnam, 1940; Lindsley, 1936; Wilson, 1925). Recent studies also have reported cocontraction of opposing and inappropriate muscles (Hallett & Alvarez, 1983; Milner-Brown & Penn, 1979; O'Dwyer, Neilson, Guitar, Quinn, & Andrews, 1983). Visual tracking tests have been employed at this laboratory to differentiate voluntary and involuntary activity in athetosis. Through this technique, a quantitative description of the voluntary control of arm movement in athetoid patients has been provided (Neilson, 1974a), and three separate components of involuntary activity have been documented (Neilson, 1974b). One of these components consisted of an "action tremor" which varied in frequency over a wide range (1.5–4 Hz) in individual patients. The presence of tremor in athetosis was previously reported in several studies (Andrews, Burke, & Lance, 1973; Hoefer & Putnam, 1940; Lindsley, 1936).

In reviewing the literature on the communicative handicap in athetoid cerebral palsy, Kent and Netsell (1978) pointed out that much of it involves comparing athetoids with other cerebral-palsied groups, especially spastics. Although some inferences concerning the physiological characteristics of athetoid dysarthria have been made from perceptual analyses and phonetic transcriptions of athetoid speech (e.g., Platt, Andrews, Young, & Quinn, 1980), only a few attempts at direct measurement have been made in this area. These have included recordings of aerodynamic variables (Hardy, 1961, 1964; Netsell, 1969b), cinefluorographic analyses of speech movements (Hardy, 1961, 1967; Kent & Netsell, 1978; Kent, Netsell, & Bauer, 1975; Netsell, 1969a), and analyses of EMG (Neilson & O'Dwyer, 1981) or combined EMG and movement variables (Netsell, 1975, 1978). The articulatory abnormalities reported in these studies were large ranges of jaw movement, inappropriate tongue positioning, difficulty in achieving velopharyngeal closure, and prolongation of transitions from one articulatory position to another (Hardy, 1961, 1967; Kent & Netsell, 1978; Kent et al., 1975; Netsell, 1969b).

Although one of the primary characteristics reported of athetoid movements is their variability, this has not been studied systematically. Yet the consistency of motor output in repeated utterances of the same speech material provides an excellent opportunity to study this characteristic. If dysarthria results from involuntary movements in athetoid speakers, then their motor output should show evidence of such movements. Since such movements are variable, irregular, or even random, the motor output for repetitions of the same speech material should not be reproducible. Rather, it should show excessive variability compared to that of normal speakers. Some limited data are available on this in the speech literature.

Kent and Netsell (1978) reported the variability of the motion paths of two radiopaque tongue markers between two recitations of the same sentence in an adult athetoid subject. Netsell (1978) noted the variability of lip displacement and velocity between repetitions of the same syllable in athetoid subjects, compared to the remarkable consistency of normal subjects. Netsell (1975) also observed that articulator coordination problems in four athetoid subjects appeared to be random in replications of the same sentence material. Instability of velar position, with intermittency of velopharyngeal closure, also has been reported (Kent & Netsell, 1978), and Hardy (1961) concluded that the isolated instances of velopharyngeal closure observed in an athetoid boy were random and unpredictable.

On the other hand, Hardy (1964) reported that the rest tidal breathing patterns, averaged over 3 min, of cerebral-palsied children (both spastic and athetoid) showed no evidence of the excessive variability that would be expected from the uncoordinated and irregular breathing patterns that these children are said to show. In our previously cited study (Neilson & O'Dwyer, 1981), the patterns of EMG activity recorded simultaneously in many muscles during multiple replications of a test sentence by athetoid cerebral-palsied subjects demonstrated a degree of reproducibility which appeared comparable to that in normal subjects. Moreover, an acoustic analysis of the associated speech waveforms also indicated reproducibility of the formant trajectories (van Doorn, 1982). Thus, the few attempts at direct physiological assessment in this area have produced apparently conflicting results with respect to this important characteristic of athetosis. In view of the potential significance of this issue, we have extended our previous qualitative study and have now quantified the patterns of EMG activity observed in normal and athetoid subjects during multiple replications of a test sentence. This has enabled statistical tests to be made of the hypothesis that the reproducibility and variability of the EMG activity do not differ in the two subject groups. This quantification is now described.

METHOD

The data for this study were collected as part of a more general procedure concerned with the pathophysiology

of dysarthria in cerebral palsy, which has been described previously (Neilson & O'Dwyer, 1981; O'Dwyer et al., 1983). Copper bipolar hooked-wire electrodes with 1-mm deinsulated tips were employed to record EMGs simultaneously from multiple lip, tongue, and jaw muscles. The following six muscles were selected for the present analysis: orbicularis oris superioris (OOS), depressor labii inferioris (DLI), anterior genioglossus (GG), geniohyoideus (GH), internal (medial) pterygoideus (IP), and anterior belly of digastricus (ABD). Surface electrodes were used to record from OOS in two normal subjects. Procedures employed for insertion and verification of the electrode placements have been described in detail elsewhere (O'Dwyer, Quinn, Guitar, Andrews, & Neilson, 1981).

Subjects

The subjects were five normal fluent speakers, aged 20–30 years, and five cerebral-palsied dysarthric speakers, aged 19–34 years. All were volunteers who gave informed consent to the experimental procedures. None of the cerebral-palsied subjects were mentally retarded, and they had no difficulty in understanding speech. However, all were severely disabled. On detailed neurological examination they were judged to be predominantly athetoid, although additional milder upper motor neuron signs were also present. Three subjects had slight weakness of finger abductors and extensors and of dorsiflexors of the ankles, without increased tendon jerks. The two other subjects had hyperreflexia without any demonstrable weakness. Sensation was intact based on clinical examination in all subjects. Two of the subjects had an intermittent torticollis to the right. A third had difficulty with vertical deviation of the eyes above the horizontal plane. Facial grimacing was present in all five subjects. Their speech was grossly athetotic. Speech intelligibility was not quantified, but these subjects could not be understood by those unfamiliar with their speech, so that they required a point board for communication.

Stimuli

The test sentence "Do all the old rogues abjure weird ladies" was recited 50 times by each subject. The first 20 recitations only are analyzed here. This sentence has a broad transcription in general Australian accent of / du ɔl ðɪ ɒld rougz æbdʒuə wɪəd leɪdiz / (van Doorn, 1982). The sentence was composed to require large ranges of movement of the lips, tongue, and jaw for its production (van Doorn, 1982). Moreover, abnormalities of timing and coordination in athetoid subjects that might possibly be avoided in the articulation of isolated words would be evident in the articulation of a sentence (Kent & Netsell, 1978). Each recitation was produced by each subject on cue from the experimenter, with a pause after every five recitations. The subjects were instructed to attempt to maintain the same rhythm, stress, intonation, and loudness as in a "reference" recitation which was prerecorded for each subject and was replayed before the first recitation and again after every five recitations.

Instrumentation

EMG signals from the hooked-wire electrodes were amplified in Dynamic Electronics type 3160 EMG preamplifiers (bandwidth 80 Hz–2.5 kHz), displayed on Dynamic Electronics type 3120 oscilloscopes, and recorded on a Philips Ana-Log 14 FM tape recorder (tape speed 38.1 cm/s, bandwidth 0–5 kHz). The speech signal, transduced with a shielded high-quality microphone mounted 40 mm from the subjects' lips, was also recorded on the tape recorder via a specially modified audio amplifier.

Digital Storage of Data

The EMG signals were played back from the tape recorder via a Neomedix NT123 active filter (bandwidth 80 Hz–2.5 kHz; 50-Hz notch filter) and monitored on oscilloscopes. The EMG and speech signals were full-wave rectified and low-pass filtered through two first-order filters connected in cascade (time constant $T1 = T2 = .02$ s; corner frequency, 8 Hz) in an EAI-180 analog computer. Because the same low-frequency filter characteristics were applied to both signals, no phase discrepancy was introduced between the acoustic and EMG signals through this signal conditioning. The resulting rectified and filtered signals are referred to as IEMG and speech intensity signals, respectively. The IEMG and speech intensity signals were displayed on oscilloscopes, sampled synchronously at 100/s by a 12-bit A-D converter, and stored in digital form in PDP 11/40 computer files. The IEMG signal associated with a sustained maximum contraction of each muscle was also sampled at 100/s and stored in computer files. Maximum contractions of each muscle were produced both before and after the 50 recitations of the test sentence, so that any changes in the EMG signals due to movement of the electrodes were detected (for details see O'Dwyer et al., 1981, 1983). None of the six muscles in the present analysis were contaminated in this way. A scaling factor was computed from the maximum for each muscle, and the IEMG signals were calibrated and expressed as a percentage of maximum contraction. For the measurement to be employed throughout this study, one unit of IEMG was defined as 1% of maximum voluntary contraction. Using DEC LAB-11 software, the speech intensity waveform and any of the IEMG waveforms associated with each recitation of the test sentence could be retrieved from computer files and displayed on a VR-17 graphics display. A cursor on the display provided a numerical readout of the X and Y values of any data point selected.

Syllable Trigger Points

The FM tape recorder was played at normal or reduced speed while the investigators watched the speech intensity waveform displayed on a Tektronix 5103N storage oscilloscope and listened to the audio signal. In this way each of the 10 syllables in each recitation of the test sentence was associated with a peak in the speech intensity waveform, as can be seen in Figure 1a. The speech intensity waveform was differentiated using a 5-point empirical method DEC LAB-11 subroutine, and both the speech intensity waveform and its differential were displayed on the VR-17 screen. The point of maximum onset velocity of the intensity peak associated with each syllable was identified by a peak in the differentiated waveform, as illustrated by an × in Figure 1b. This point did not correspond to the onset of the acoustic waveform for the syllable but was determined by the voicing intensity of the syllable. This point was chosen to provide a consistent measure of the location in time of each syllable and is referred to as the *trigger point* of the syllable. The 10 trigger points for the speech intensity waveform in Figure 1a are each indicated by an ×. The locations of the trigger points of all of the syllables in each of the 20

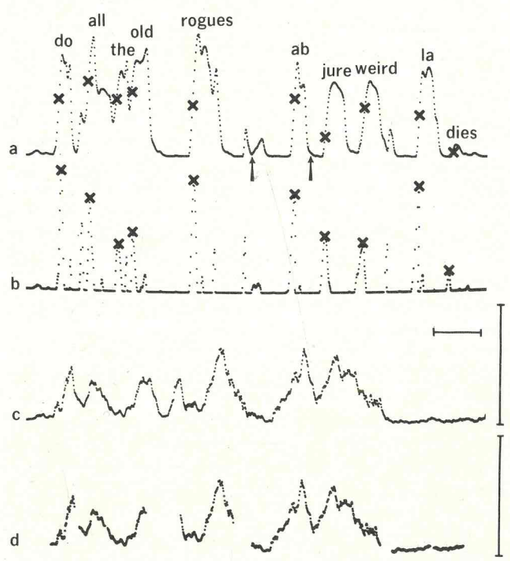

FIGURE 1. (a) The speech intensity trace for one recitation of the test sentence by an athetoid subject. (b) The positive differential of the intensity trace. The ×s mark the point of maximum onset velocity of the intensity trace for each syllable, thus identifying the "trigger points" for each syllable. The arrows in (a) mark the boundaries for the syllable *ab*. (c) The complete IEMG trace for OOS during this recitation of the sentence. (d) The trace in (c) after activity between some syllables has been removed, according to the procedure described in the text. The 10 IEMG syllable waveforms are represented in (d), and only this IEMG activity was analyzed further. The calibrations are 1 s and 0–100% of maximum voluntary contraction.

recitations of the test sentence were identified for each subject and recorded.

Timing of Syllables

A measure of the time between syllable trigger points was obtained by counting data points (data sampled at 100/s). Nine intervals were measured between the 10 syllables in each recitation of the sentence. The average value and the standard deviation of each of these nine intervals across the 20 recitations were computed for each subject. A relative measure of the variability of each time interval across the 20 recitations was obtained by expressing the standard deviation as a coefficient of variation; that is, standard deviation divided by the mean (Yamane, 1973). Such a relative measure was employed because of the tendency for variability of phonetic segment duration to vary with mean duration of segments (Kent, Netsell, & Abbs, 1979). In addition, the minimum value of each of the nine intervals over the 20 recitations was obtained for each subject.

Formation of IEMG Waveform Ensembles

As can be seen in the results, analysis of the time intervals between syllables showed that the rate of syllable production was variable in both subject groups—particularly so in the athetoid group. This variability precluded averaging of IEMG waveforms across the whole sentence with respect to a single trigger point, because this would produce misalignment or smearing of IEMG activity removed in time from the trigger point. Therefore, the IEMG waveforms associated with each syllable were averaged with respect to the trigger point for each syllable. A computer program was written to determine the IEMG waveform associated with each syllable. The program performed the following procedure.

The beginning of the IEMG waveform was taken as the point which was .6 of the minimum interval between the trigger point of the syllable and the trigger point of the preceding syllable. The end of the waveform was taken as the point .6 of the minimum interval between the trigger point of the syllable and the trigger point of the following syllable. The start and end points for the syllable *ab* are indicated by the arrows in Figure 1a. For the first syllable, *do*, for which there was no preceding syllable, .6 of the minimum distance to the adjacent syllable, *all*, was used to determine both the beginning and end points of the IEMG waveform. An analogous procedure was used for the last syllable, *dies*. This standardized procedure could be employed for all subjects despite the intersubject variability in rate of syllable production. Thus, IEMG waveforms of equal duration were obtained for the 20 repetitions of any given syllable by any given subject, the actual durations being determined in the above procedure by the minimum intersyllable distances occurring in

each subject. The IEMG waveforms ranged in duration from a minimum of 50 ms (in a normal subject) to a maximum of 1510 ms (in an athetoid subject). The means and standard deviations of the waveform durations for the normal and athetoid subjects were 321 +/− 140 ms and 725 +/− 286 ms, respectively.

Figure 1c shows the complete activity recorded from OOS during one recitation of the test sentence by an athetoid subject. The IEMG waveforms that were determined for the 10 syllables according to the above segmentation procedure are shown in Figure 1d. Only the IEMG activity in Figure 1d was included for further analysis. In this instance, muscle activity was excluded from the analysis in the intersyllable intervals *do-all*, *all-the*, *old-rogues*, *rogues-ab*, *weird-la*, and *la-dies*, as evidenced by the discontinuities in the waveform. At the same time, muscle activity was included twice in the analysis (that is, at the end of the waveform for one syllable and again at the beginning of the waveform for the next syllable) in the intersyllable intervals *the-old*, *ab-jure*, and *jure-weird*, where no discontinuities in the waveform in Figure 1d are apparent. The overlap of the muscle activity in these latter syllables is not shown here, in order not to disrupt the temporal alignment of Figure 1c and 1d.

The choice of .6 as the proportion of the minimum interval was a compromise between the exclusion and inclusion of muscle activity between syllables. Muscle activity between the syllables was excluded from the analysis when the intersyllable interval exceeded the minimum interval by 20%. The important point here is that the muscle activity which was excluded occurred in prolonged intervals between syllables and not during syllables. The IEMG waveform which was determined for each syllable through this segmentation procedure consisted of muscle activity both preceding and during each syllable, but not always following each syllable. The fact that muscle activity precedes or accompanies, rather than lags, the movements for a syllable was therefore accounted for through this procedure. Moreover, the segmentation tended to overestimate the duration of the IEMG waveform associated with each syllable; that is, IEMG activity associated with movements between syllables was included. Since, as already noted, the results showed that the time intervals between syllables were more variable in the athetoid subjects than in the normal subjects, this overestimation of the duration of the waveform would tend to differentially overestimate the variability of the IEMG activity associated with each syllable in the athetoid subjects. Nevertheless, this was considered preferable to an underestimation of the duration of the IEMG waveform, which might not have included the full range of IEMG change for each syllable. Moreover. in terms of testing the null hypothesis that the two groups do not differ in variability, this represented a conservative error.

An ensemble of IEMG waveforms was formed for each subject-muscle-syllable combination, giving a total of 300 ensembles for each group (5 subjects × 6 muscles × 10 syllables). Each ensemble contained 20 waveforms corresponding to the 20 recitations of each syllable, and the waveforms were aligned relative to the trigger points in the acoustic signal.

Waveform Analysis

Signal-to-noise ratio is a measure commonly employed in communications to assess quality of waveforms. In the present study, the ratio of the reproducible (i.e., signal) component to the variable (i.e., noise) component of the IEMG waveforms in each ensemble was computed. Such signal-to-noise ratios were necessary because, as seen below, the reproducible component and the variable component of the waveforms varied together. Therefore, assessment of one without the other was inappropriate. These signal-to-noise ratios were then compared between the normal and athetoid subject groups.

Each of the 600 ensembles was analyzed as follows. The 20 waveforms in each ensemble were averaged to produce an average waveform for the ensemble. Visual inspection of the waveforms on the VR-17 graphics display indicated that the individual waveforms in an ensemble were homogeneous in respect of the average pattern, in the sense that each individual waveform comprised the same pattern combined with a different background. This was confirmed in the linear regression analysis outlined below—in which the individual waveforms were correlated with the average waveform—by the consistently high correlation coefficients obtained. The average waveform therefore represented the reproducible component of the waveforms in the ensemble, that is, the *signal*. The power of the signal was computed by calculating the mean square value of the average waveform. The variable component of each waveform in the ensemble—that is, the *noise*—was obtained by subtracting the average waveform from each of the individual waveforms in the ensemble. Thus, the noise represented the deviation of each waveform about the average waveform. The power of the noise was computed for each waveform by calculating the mean square value of the noise. The average-noise-power for the ensemble was obtained by averaging the mean square values of the noise across the 20 waveforms in the ensemble. The signal-to-noise ratio was determined for each waveform as the ratio of signal-power to noise-power. The average signal-to-noise ratio for the ensemble was obtained by averaging the signal-to-noise ratios across the 20 waveforms in the ensemble.

A linear regression analysis was performed between each waveform in the ensemble and the average waveform. This enabled each waveform to be separated into a component that was correlated with the average waveform and a component not correlated with the average waveform. The correlated component from this analysis may have had different DC-level and different peak-to-peak amplitude from the average waveform—as detected by changes in the intercept and slope of the regression line, respectively—but always had the same waveshape as the average waveform. The uncorrelated component from this analysis was due to differences in waveshape

from the average waveform and was reflected in the scatter of the data points about the regression line, as detected by the residual variance of the regression analysis. From this analysis the following measures were made: contraction level (i.e., DC-level) of each waveform, peak-to-peak amplitude of the correlated component of each waveform, and residual variance of each waveform. The average value, standard deviation, and variance of each of these measures across the 20 waveforms in the ensemble were computed.

Thus, in addition to the average-noise-power, which measured the average variability in each ensemble, three component variability measures were obtained: (a) DC-noise-power measured by the variance of the DC-levels across the 20 waveforms, (b) amplitude-noise-power measured by the variance of the peak-to-peak amplitudes across the 20 waveforms, and (c) waveshape-noise-power measured by the average residual variance across the 20 waveforms. Similarly, in addition to the average signal-to-noise ratio for each ensemble—the signal-to-DC-noise ratio, signal-to-amplitude-noise ratio and signal-to-waveshape-noise ratio—were computed for each ensemble.

RESULTS

Timing of Syllables

The average times between syllable trigger points and their variability, as measured by coefficients of variation, were compared statistically by means of factorial analysis of variance with one group factor (A) and one repeated measures factor (B) (Winer, 1971, p. 261). In each analysis Factor A had two levels representing the normal and athetoid subject groups, whereas Factor B had nine levels representing the nine time intervals between the 10 syllables in the test sentence. Profiles of the average times between syllable trigger points and of their variability are presented for normal and athetoid subjects in Figure 2(a and b). Times between syllable trigger points were significantly longer for athetoid subjects than for normal subjects [A effect: $F(1, 8) = 73.81$, $p < .001$]. There was a significant interaction between subjects and time intervals [AB effect: $F(8, 64) = 9.09$, $p < .001$], indicating that the pattern of times between syllable trigger points across the sentence was different in the two subject groups. The times between syllable trigger points were also significantly more variable in athetoid subjects than in normal subjects [A effect: $F(1, 8) = 71.49$, $p < .001$].

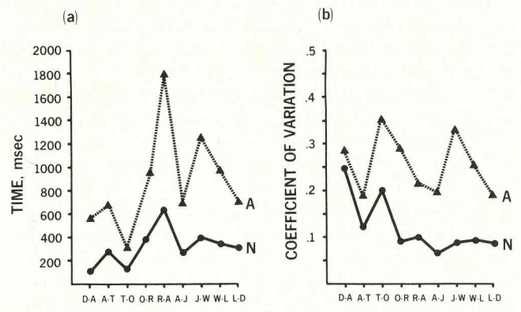

FIGURE 2. The average durations, in ms, of the time intervals between syllable trigger points (a) and their coefficients of variation (b) in the normal (N) and athetoid (A) subject groups. The nine intervals between the 10 syllables in the test sentence are identified by D-A for *do-all*, A-T for *all-the*, and so on.

Noise

A multiple regression analysis of average-noise onto DC-noise, amplitude-noise, and waveshape-noise was performed in order to determine how well the average-noise of each ensemble was accounted for by a combination of the three component noise measures. This analysis is summarized by the following multiple regression equation,

$$N = .82\,DCN + .23\,AN + .67\,WN$$

where N = average-noise, DCN = DC-noise, AN = amplitude-noise, and WN = waveshape-noise. The multiple correlation coefficient from this analysis was .98. This indicates that the average-noise was almost completely accounted for by a linear combination of the three component noise measures. An analysis of an ensemble having average levels of each of these four noise measures showed that typically, 42% of the average-noise was attributable to DC-noise, 35% to amplitude-noise, and only 23% to waveshape-noise.

Signal

Signal-power across the 600 ensembles was compared statistically by means of factorial analysis of variance with one group factor (A) and two repeated measures factors (B and C) (Winer, 1971, p. 546). In each analysis Factor A had two levels representing the normal and athetoid subject groups, Factor B had six levels representing the six muscles tested, and Factor C had 10 levels representing the 10 syllables in the test sentence. Signal-power was significantly greater in athetoid subjects than in normal subjects [A effect: $F(1, 8) = 9.94$, $p < .05$], and it varied significantly with muscles [B effect: $F(5, 40) = 3.32$, $p < .05$]. There were also significant interactions between subjects and muscles [AB effect: $F(5, 40) = 2.73$, $p < .05$] and between subjects and syllables [AC effect: $F(9, 72) = 2.05$, $p < .05$], indicating that the variation of signal-power with both muscles and syllables was different in the two subject groups.

Signal-to-Noise Ratios

Scatter plots of signal-power versus each of the four noise measures for the 600 ensembles are presented in Figure 3. There was considerable overlap in the ranges of

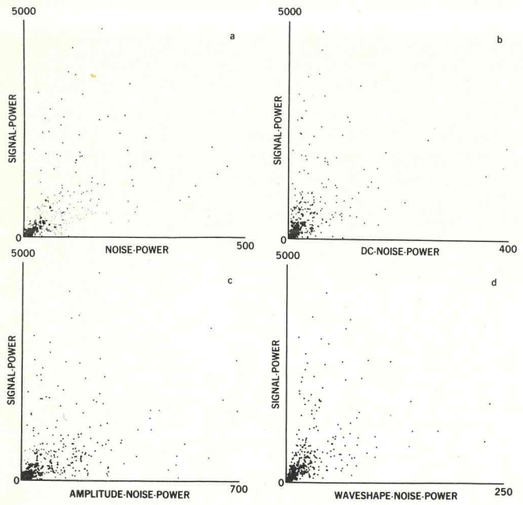

FIGURE 3. Scatter plots of signal-power against average-noise-power (a), DC-noise-power (b), amplitude-noise-power (c) and waveshape-noise-power (d). Each plot contains 600 data points, 60 for each subject. Each data point represents the average value for one ensemble of 20 IEMG syllable waveforms. Power is in square units, where 1 unit = 1% of maximum voluntary activity.

signal-power in the two subject groups; for example, of the 300 waveform ensembles in each group, 282 in the normal subjects had a signal-power less than 500 square units (1 unit = 1% of maximal voluntary contraction), whereas 187 in the athetoid subjects had a signal-power in this range. The coefficients of correlation obtained between signal-power and noise-power measures in parts a, b, c, and d of Figure 3 were $r = .60$, $r = .49$, $r = .52$, and $r = .56$, respectively. Each of the scatter plots had a "fan-shaped" distribution of points, indicating that an increase in signal was accompanied not only by an increase in noise, but also by an increase in the scatter of the noise.

Amplitude distribution histograms for each of the four signal-to-noise ratios were calculated on the data from the 300 ensembles in each group. The distributions were all positively skewed. Each of the signal-to-noise ratios was expressed in decibel units by multiplying the log transform of each ratio by 10. The grand mean signal-to-noise ratios, prior to log transformation, for the normal and athetoid subject groups were 19:1 and 16:1, respectively. Histograms showing the amplitude distribution of the average signal-to-noise ratios, following log transformation, for the 300 ensembles in the normal and athetoid subject groups are presented in Figure 4. The log transformation removed the positive skewness from the signal-to-noise ratio measures, causing the distributions to approach normality, and ensured homogeneity of variance across cells in the factorial analysis of variance that was employed to compare the signal-to-noise ratios across the 600 ensembles (Winer, 1971, p. 397).

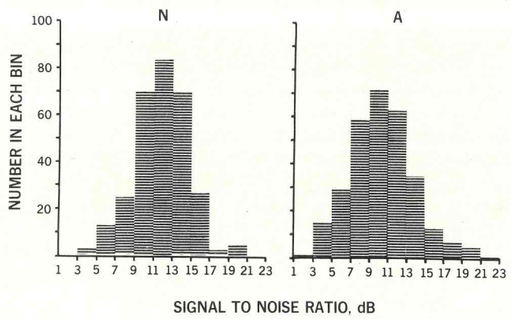

FIGURE 4. Amplitude distribution histograms of the average signal-to-noise ratios in dB (i.e., after log transformation) for the 300 ensembles of IEMG waveforms in the normal (N) and athetoid (A) subject groups.

The factors in the analyses of variance for the signal-to-noise ratios, signal-to-DC-noise ratios, signal-to-amplitude-noise ratios, and signal-to-waveshape-noise ratios were the same as for the analysis of signal-power. There was no statistically significant main effect difference between the two subject groups in any of the four signal-to-noise ratios [A effect: $F(1, 8) <= 2.78$, $p => .13$ for the four analyses]. Particularly important here was the analysis of variance of the signal-to-waveshape-noise ratios [A effect: $F(1, 8) = .43$, $p = .53$], because according to the alternative hypothesis, the IEMG waveshape should have been especially variable due to the superposition of irregular involuntary activity. The similarity of the signal-to-waveshape-noise ratios in the two subject groups is borne out by the profiles of the mean values of these ratios versus syllables for the six muscles presented in Figure 5. There was no significant interaction between subjects and muscles in any of the four signal-to-noise ratios, so that the variation of these ratios across muscles was similar in the two subject groups [AB effect: $F(5, 40) <= 1.97$, $p => .10$ for the four analyses]. There was, however, a significant interaction between subjects and syllables in the four signal-to-noise ratios, indicating that the variation of these ratios with syllables was different in the two subject groups [AC effect: $F(9, 72) => 2.26$, $p < .05$ for the four analyses].

These findings indicate that the ratio of the reproducible (signal) and variable (noise) components of the patterns of IEMG activity is comparable in the normal and athetoid subjects. This is borne out in Figure 6, in which the 10 IEMG waveforms from OOS for a typical replication of the test sentence have been concatenated and are contrasted with the concatenated average waveforms from OOS for each syllable. The similarity of the waveforms in the individual recitations to the average waveforms demonstrates the degree of reproducibility of the IEMG activity in both the normal and the athetoid subjects. The dissimilarities of the normal and athetoid waveforms are impressive evidence of the muscle control abnormalities of athetosis.

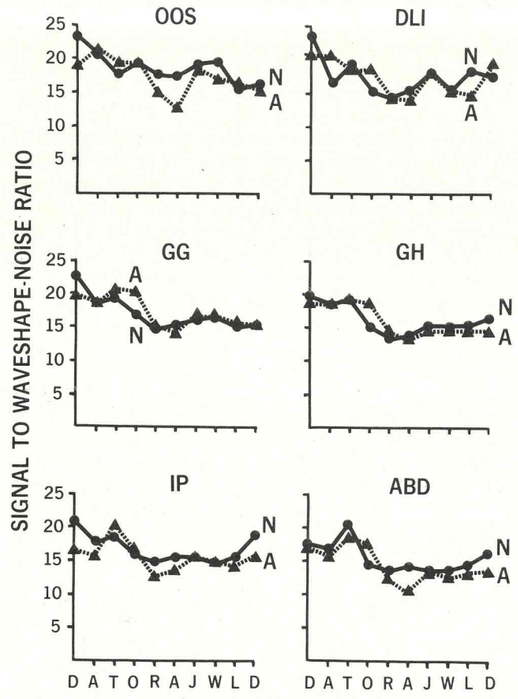

FIGURE 5. The average signal-to-waveshape-noise ratios, in dB, for the IEMG waveforms for each syllable are shown for each of the six muscles in the normal (N) and athetoid (A) subject groups. Abbreviations for muscles as in text and for syllables as in Figure 2.

DISCUSSION

The results of this study show that the syllables in the test sentence were produced both more slowly and at a more variable rate by the athetoid subjects than by the normal subjects. The IEMG waveforms associated with individual syllables in the sentence had reproducible and variable parts which varied together in power (mean square value). Both the DC-level and the variance of the reproducible part of the IEMG waveforms were significantly greater in the athetoid subjects than in the normal subjects. The ratio of the reproducible part to the variable part did not differ significantly between the two subject groups, indicating that relative to the level of the signal, the IEMG waveforms associated with each syllable were no more variable in athetoid dysarthric speakers than in normal fluent speakers. Thus, our initial hypothesis was confirmed. The fact that the athetoid subjects showed reproducibility comparable to that of normal subjects in the IEMG waveforms associated with individual syllables in the test sentence does not support the idea that

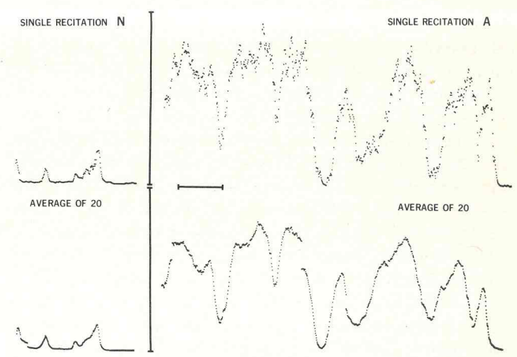

FIGURE 6. IEMG waveforms recorded from OOS during a single recitation of the test sentence (top trace) compared to the average waveforms for this muscle (bottom trace) in a normal (N) and an athetoid (A) subject. The 10 IEMG waveforms for the individual syllables have been concatenated to form each trace. The calibrations are 1 s and 0–100% of maximum voluntary contraction.

athetoid dysarthria is due to irregular and variable involuntary movements.

The fan-shaped distribution of points in the scatter plots of signal against noise (Figure 3) suggests the presence of an interaction between signal and other factors which influences the level of noise; that is, the noise level was influenced by factors other than signal, but these influences themselves were potentiated by an increase in signal. This complex relationship between signal and noise indicated that a comparison of IEMG noise measures which did not control for varying levels of signal would be inappropriate. Signal-to-noise ratios provided measures of the variability of IEMG waveforms in each ensemble which took the level of the signal into account and therefore could be used for comparisons across ensembles having different levels of signal.

The groups in this study consisted of only five subjects. Therefore, the risk of a type II statistical error—that is, failing to find a difference between the groups where one really exists—should be borne in mind. The cerebral-palsied subjects, however, all had severe athetoid dysarthria, to the extent that their speech was unintelligible. Hence, the variable involuntary activity which has been thought to underlie their dysarthria should have been particularly obvious in these subjects. Indeed, if the existing view of athetoid dysarthria were valid, the difference between the normal and athetoid subjects should have been so obvious as to be detectable without the need for statistical analysis. The signal-to-noise ratio and signal-to-waveshape-noise ratio data in Figures 4 and 5, respectively, show clearly that this was not the case. Since such a large effect size was predicted by the alternative hypothesis, the power of the present statistical test may be considered adequate despite the small number of subjects. In addition, the analysis of variance of the times between syllable trigger points (cf. Figure 2a)

showed a large A effect—$F(1, 8) = 73.81$, $p < .001$—yet this test had the same statistical power as the analyses of variance of the signal-to-noise ratios.

All four signal-to-noise ratios varied differently with syllables in the two subject groups, as indicated by the significant interaction between subjects and syllables (AC effect) in these data. However, the differences in the shape of the profiles of signal-to-noise ratios versus syllables were small, as Figure 5 bears out. Moreover, to the extent that the signal-to-noise ratios were not constant across all IEMG waveforms, some variation in the ratios would be expected on the basis of differences between the waveforms in factors such as duration, signal power, and waveshape complexity—factors which varied differently with syllables in the two subject groups.

Analysis of the time intervals between syllable trigger points showed they were considerably more variable in the athetoid subjects than in the normal subjects (Figure 2b), bearing in mind that the measure of variability (coefficient of variation) was a relative one which took the mean intervals between trigger points into account. The time between syllable trigger points, however, included the time interval to the next syllable as well as the production time of the syllables themselves. Although it may be concluded from this that the athetoid subjects had a more variable rate of syllable production than the normal subjects, data on the production time of the syllables themselves are lacking. In the study by van Doorn (1982) cited earlier, an acoustic analysis of the speech waveforms from the first five of the sentences examined here was carried out and the duration of each of the 10 syllables was estimated, employing a combination of the formant trajectories, intensity contours, and the acoustic waveform to determine the syllable boundaries. A factorial analysis of variance of the coefficients of variation of these syllable durations, calculated for the present study, showed no significant difference between the normal and athetoid subjects [A effect: $F(1, 8) = 3.28$, $p = .11$]. This is consistent with the finding of no significant difference between the two groups in the signal-to-noise ratios of the IEMG waveforms for each syllable. The variable rate of syllable production appears, therefore, to be predominantly a consequence of variability in the time between syllables, rather than variability in the production time of the syllables themselves.

Athetoid speakers experienced various involuntary responses such as facial grimacing, spasms, blocking, tongue thrusting, and gagging. These responses usually occurred between syllables, however, and following them the subject continued with the next syllable, producing the same abnormal but reproducible pattern of muscle activity as if the involuntary response had not occurred. Only occasionally were the syllables themselves disrupted by involuntary responses, and in such instances the subject repeated the disrupted syllable, again producing the same abnormal but reproducible pattern of IEMG activity as if the involuntary response had not occurred. Moreover, replications of the test sentence were obtained from each athetoid subject which were not interrupted by such involuntary responses, and these remained dysarthric and unintelligible. Thus, the involuntary responses which interrupted athetoid speech occurred intermittently and irregularly, but usually between syllables.

This involuntary activity accounts for the variability in the timing of the total motor output of the athetoid subjects and is consistent with descriptions of involuntary athetoid movements as being variable and irregular. The notion that athetoid dysarthria is due to variable involuntary activity may have resulted from observation of such variable activity occurring between syllables. In the present study, however, since the IEMG waveforms were averaged with respect to the trigger point for each syllable, variable activity occurring between syllables was removed from the analysis, as illustrated by the differences between Figure 1c and 1d. It seems likely that such variable activity occurring between syllables will affect intelligibility adversely by disrupting prosody (Rosenbek & LaPointe, 1978). Articulation of the syllables themselves, however, is largely unaffected by such activity, and the speech of these subjects remains unintelligible in its absence. Therefore, this variable involuntary activity occurring between syllables cannot be the primary cause of athetoid dysarthria.

The possibility remains that although the relative variability of the IEMG waveforms associated with each syllable was not significantly greater in athetoid speakers than in normal speakers, the resulting articulator movements and vocal tract shapes may have been more variable in athetoid subjects. Folkins (1981) observed that the variability in EMG activity in jaw closing muscles for repetitions of the same syllable suggested that normal speakers may have the flexibility to use different combinations of muscle activity to achieve similar jaw closing movements. Abbs (1982) reported compensatory tradeoffs in the levels of activity in synergistic lip closing muscles contributing to the same lip movement. In the absence of the appropriate measures, it is not possible to assess the relationship of IEMG variability to variability of articulator movement and vocal tract shape in the two subject groups in the present study. It must be recognized, however, that whatever variability in articulator movements is present in the athetoid subjects, it almost certainly is variability about a pattern of movements that itself is abnormal, owing to the gross abnormality of the reproducible component of their muscle activity (Figure 6). The abnormal patterns of articulatory movement documented in athetoid subjects by Kent and Netsell (1978) support this view. It does not seem reasonable, therefore, to account for athetoid dysarthria on the basis of such variability.

The reproducibility of the signal component of the lip, tongue, and jaw muscle activity in the athetoid subjects strongly suggests that for each syllable in the test sentence, a similar abnormal pattern of motor commands was produced centrally on each replication of the test sentence by these subjects. The only alternative interpretation to this appears to be that these abnormal but reproducible signals consist, at least partially, of involuntary activity which has been triggered reproducibly on each attempted replication of the test sentence.

The notion of consistently triggered involuntary movement is at variance with the notion of athetoid movement as being irregular and variable. Furthermore, as detailed in a previous report on these same adult athetoid subjects (Neilson & O'Dwyer, 1981), no primitive reflexes or pathological reactions could be elicited in these subjects by tickling, stroking, rubbing, or tapping of the head, neck, cheeks, lips, gums, tongue, palate, ears, or nostrils. No movements of the head, rooting, sucking, biting, pouting, movements of the lips, jaw deviations, mouth opening, or tongue movements were observed in response to such stimulation. Thus, if consistently triggered involuntary movement occurred during speech, but no involuntary movement could be evoked by direct stimulation when the subject was at rest, it would appear that the mechanism for such self-generated involuntary movement would have to be activated centrally in association with attempts to speak. Inappropriate central activation of such a reflex mechanism, however, is equivalent to the selection of an inappropriate motor program (see Evarts, 1982, p. 24), and the resultant movement would be more correctly termed *inappropriate voluntary movement*, rather than involuntary movement. The only tenable interpretation of the reproducible signals, therefore, appears to be that for each syllable in the test sentence a similar abnormal pattern of motor commands is produced centrally on each replication of the test sentence by the athetoid subjects.

It follows from this interpretation that in order to elucidate the pathophysiological mechanisms of athetoid dysarthria, we ought to direct our attention to the characteristics of the abnormal patterns of muscle contraction evident in the reproducible parts of the IEMG waveforms, rather than to the variable muscle contractions associated with involuntary movements. The excessive power of these reproducible signals, for example, is one clear abnormality demonstrated in the present study (cf. Figure 6), in agreement with the excessive activity we observed previously in upper airway muscles during nonspeech tasks in cerebral-palsied subjects (O'Dwyer et al., 1983). Excessive activity was also observed by Hallett and Alvarez (1983) in upper arm muscles during elbow movements in athetoid subjects. Other abnormalities in these signals are apparent in Figure 6 and are to be the subject of a subsequent report.

Such emphasis on abnormalities of voluntary movement is also evident in the study by Hallett and Alvarez (1983), and Foley (1983) maintained that voluntary movements are incorrectly programmed in athetosis. Yet such attention to motor programming is at variance with much of the literature on cerebral palsy in general, which focuses attention on rigidospasticity, spasm, and involuntary movement as the primary cause of the motor disorder. With regard specifically to dysarthria in cerebral palsy, the existing hypotheses concerning the pathophysiological mechanisms involved were described by Foley (1983) as "cherished theories that have held sway too long" (p. 293). The findings of our previous experimental investigations, as with the present study, do not support these existing hypotheses (Neilson, Andrews, Guitar, & Quinn, 1979; Neilson & O'Dwyer, 1981; O'Dwyer et al., 1983). The available experimental results, therefore, point to a fundamental abnormality of voluntary motor programming in cerebral palsy.

This represents a new and very different approach to cerebral palsy, the significance of which can only be fully appreciated when it is seen in the context of recent findings on both the neuroanatomical/neurophysiological substrates of voluntary movement control and the neuropathology of cerebral palsy. In this context a definitive account is not yet possible, since it must draw on information and conceptualizations from the current forefront of neuroscience. Consequently, some of what follows must necessarily be speculative. Nevertheless, we feel it is imperative to offer some account of this new perspective on cerebral palsy, because it provides a focus for further much needed physiological research in this neglected area.

PATHOPHYSIOLOGY OF CEREBRAL PALSY

The advent of computed tomographic (CT) and ultrasound brain scanning, together with advances in neonatal intensive care, has shed new light on the neuropathology of cerebral palsy in recent years. Concern for the neurodevelopmental outcome of infants, especially premature ones, at risk for or surviving brain damage has focused attention on the mechanisms of perinatal hypoxic-ischemic encephalopathy. The relative contributions of hemorrhage, ischemia, and hypoxia to perinatal brain damage remain contested (Fenichel, 1983; Pape & Wigglesworth, 1979). They depend on gestational age and are multifactorial in origin (De Reuck & Vander Eecken, 1983; Horbar, Pasnick, McAuliffe, & Lucey, 1983; Norman, 1979; Pape & Wigglesworth, 1979; Volpe, 1981). In preterm infants, periventricular-intraventricular hemorrhage and periventricular leukomalacia are the most frequent varieties of cerebral disorder (De Reuck & Vander Eecken, 1983; Kulakowski & Larroche, 1980; Pape & Wigglesworth, 1979; Volpe, 1981). Periventricular-intraventricular hemorrhage is also of major importance in term infants (Ludwig, Brand, & Brockerhoff, 1980), along with ischemic lesions affecting the periventricular area (Pape & Wigglesworth, 1979).

Although the linking of clinical syndromes to patterns of pathological lesion within the neonatal period is far from complete, nevertheless, some of the above lesions have already been linked with cerebral palsy (Armstrong & Norman, 1974; Norman, 1979; Pape & Wigglesworth, 1979). Moreover, periventricular-intraventricular lesions have been identified on CT (Williamson, Desmond, Wilson, Andrew, & Garcia-Prats, 1982) and ultrasound (Palmer, Dubowitz, Levene, & Dubowitz, 1982; Stewart et al., 1983) brain scans in preterm infants who subsequently developed cerebral palsy. Furthermore, these lesions have been identified on CT scans in diagnosed cerebral-

27 502–517 December 1984

palsied children (Cohen & Duffner, 1981; Koch, Braillier, Eng, & Binder, 1980; Kulakowski & Larroche, 1980).

As noted by Fenichel (1983), "the most common site of abnormality in the brains of infants, especially premature ones, who die in the first three months post partum is in the deep white matter surrounding the lateral ventricles" (p. 262). This area represents the boundary zones between the ventriculofugal and ventriculopetal arteries within the brain (Armstrong & Norman, 1974; De Reuck, 1971; Takashima & Tanaka, 1978). The vulnerability of this area has been noted previously (Gilles & Murphy, 1969; Kulakowski & Larroche, 1980; Leech & Alvord, 1974) and is supported by animal models (Conner, Lorenzo, Welch, & Dorval, 1983; Fenichel, 1983; Young, Hernandez, & Yagel, 1982). In discussing the neuropathology of spastic diplegia, Pape and Wigglesworth (1979, p. 105) indicated that the sites typically involved in periventricular leukomalacia include the area of white matter through which the long descending tracts from the motor cortex pass. They pointed out that the leg distribution, being closer to the ventricles, is thus most likely to be damaged. In this respect, however, it may be noted that at the level of the internal capsule, the ascending fibers from the ventral tier thalamic nuclei to the peri-Rolandic cortex lie medial to the leg distribution, closer to the ventricles (Carpenter, 1976), and therefore are even more likely to be damaged. The emerging functional significance of these thalamic nuclei in motor control is now elucidated.

Projections of the nuclei ventralis anterior (VA) and ventralis lateralis (VL) of the thalamus upon the frontal cortex in the primate have been investigated with retrograde labeling (Jackson, Anden, & Dahlstrom, 1975; Kievit & Kuypers, 1977). These studies indicate that VA does not project to the motor cortex (area 4) but upon more anterior portions of the frontal lobe, whereas influences from the ventralis lateralis pars oralis (VLo) are directed largely upon area 6 of the sensorimotor cortex. DeLong and Georgopoulos (1981) concluded that it seems unlikely that basal ganglia outputs influence the primary motor cortex directly because they do not terminate in the nucleus ventralis posterior lateralis pars oralis (VPLo) of the thalamus, which provides the major input to the motor cortex.

Based on a review of the neuroanatomical connections of the basal ganglia (DeLong & Georgopoulos, 1981), it has been argued that a topographically organized loop concerned with motor control passes from the sensorimotor cortex to the putamen, to the ventral two thirds of the globus pallidus, then to VLo in the thalamus and back to area 6 of the cortex (DeLong, Georgopoulos, & Crutcher, 1983). The somatotopic motor representation present in this loop suggests that there exist segregated pathways through the basal ganglia for the control of movement of different body parts (DeLong et al., 1983). Neurophysiological evidence for the predominant motor function of this loop comes from the relationship of the discharge of basal ganglia neurons with specific aspects of limb movements in monkeys (DeLong, 1971, 1973; DeLong & Georgopoulos, 1979, 1981; DeLong & Strick, 1974). The discharge of basal ganglia neurons was found to be related predominantly to the direction, amplitude, and force of arm movement rather than to the activity of individual muscles. Based on this evidence, it was suggested that the basal ganglia play a role in the integration of information from cortical areas and in the determination of specific parameters of movement (DeLong et al., 1983).

Consistent with formulations by both Brooks (1979) and Arbib (1981), Evarts (1981) suggested that initiation of movement might involve a connection between area 6 and the motor cortex either directly via corticocortical pathways and/or through the cerebellum via cerebrocerebellar thalamocortical pathways. There are well-developed projections from area 6 via pontine nuclei to the dentate nucleus and to the lateral parts of the cerebellar hemispheres (see Brooks & Thach, 1981), and the dentate projects heavily to VPLo nucleus of the thalamus which in turn projects to the motor cortex (Tracey, Asanuma, Jones, & Porter, 1980). Evidence that cerebellothalamocortical pathways are involved in initiating centrally programmed movements has been provided by Evarts and Tanji (1974), Thach (1975), and Strick (1978), who showed that discharges of dentate neurons associated with centrally programmed movements in the monkey occur as early as 10 ms ahead of similar activity in the motor cortex.

Additional evidence for involvement of the cerebellum in central programming of movement is provided by studies on the effects of cooling of cerebellar nuclei, which show a change in the long-latency (50–100 ms), voluntary component of motor cortex activity in response to limb displacement (Vilis, Hore, Meyer-Lohmann, & Brooks, 1976) and an increase in simple visual reaction times by as much as 100 ms (Meyer-Lohmann, Hore, & Brooks, 1977). Furthermore, Sasaki and Gemba (1983) showed that the characteristic surface-negative, depth-positive field potentials in the motor cortex preceding visually initiated hand movements in the monkey are completely eliminated following extirpation of the cerebellar hemisphere ipsilateral to the moving hand. They concluded that fast and skillful reaction time movements are initiated by impulses impinging upon the motor cortex through the neocerebellum and superficial thalamocortical projections.

Thus, as reviewed by Evarts (1981) and by DeLong and Georgopoulos (1981), there is now considerable evidence indicating that subcortical loops through the basal ganglia and cerebellum are involved in initiating centrally programmed movements. Consequently, it seems justified to suppose that lesions of periventricular white matter in the perinatal brain would interfere with central processes involved in movement programming. The question can now be asked, What kind of motor programming deficit could result from damage to white matter within these loops?

A number of authors (see Bowman, 1971; Neilson, Neilson, & O'Dwyer, 1984; Sperry, 1967) have suggested that movement might be planned at a cortical level in terms of the desired perceptual consequences of the movement and then transformed in subcortical motor structures into properly timed, graded, and coordinated patterns of muscle contraction. The need for such transformations has also been postulated in the field of robotics (Brady & Hollerbach, 1982), where it has been shown that the desired movement of a robot arm must be transformed from an objective level, such as the desired trajectory of the end-point of the arm in three-dimensional space, to a joint level, representing the required angular changes of each degree of freedom of movement, and finally to an actuator level, representing command signals to the motor control systems.

Since the dynamic relationships between motor commands and their perceptual consequences are easily altered, for example, by changes in mechanical loads or changes in the external system being controlled, it follows that in order to control movement accurately the CNS must adaptively adjust its transformations of desired perceptual effects into motor commands in order to compensate for such changes. According to our formulation of a Sensory-Motor (S-M) Model theory of adaptive movement control (Neilson et al., 1984), transformations of neural signals in subcortical loops through the basal ganglia and cerebellum represent transformations of desired perceptual effects into appropriate inputs to the motor cortex to achieve them. Changes in relationships between motor commands and their perceptual consequences are detected in association cortex by a continuous monitoring of sensory and motor feedback signals. Indeed, it is suggested that one of the main functions of the association cortex is to maintain an accurate "internal model" of the external world by continuously computing, storing, and updating the reciprocal relationships between each and every sensory and motor feedback signal.

Bowman (1982) has also noted the reciprocal nature of the sensory-to-motor and motor-to-sensory relationships occurring at a cortical level and suggested that they are an essential substrate for speech development. The association cortex uses these reciprocal relationships to compute an appropriate descending modulation of information processing in the basal ganglia and cerebellum. Thus, the transformations of desired perceptual effects into motor commands in the subcortical loops through the basal ganglia and cerebellum are adaptively controlled by sensory and motor feedback signals via the association cortex. The existence of such adaptive compensation is adequately demonstrated in the case of speech control by the ability of speakers to employ highly flexible motor programs to achieve a highly consistent acoustic product (see Bowman, 1971; Lashley, 1968; Netsell, 1982; Sperry, 1967).

Based on the results of regional cerebral blood flow studies in normal human subjects performing voluntary movements (Roland, Larsen, Lassen, & Skinhoj, 1980), we propose that the premotor cortex in the lateral part of area 6 normally plays a role in computing internal models of the reciprocal relationships between motor commands and their perceptual consequences and in adaptively modulating information processing in the basal ganglia and cerebellum. Regional cerebral blood flow studies show increased bilateral activity in area 6 when subjects plan and execute voluntary movements of the hand. When subjects only plan the movement without actually performing it, however, only the supplementary motor area in the mesial part of area 6 shows increased activity, whereas the premotor cortex in the lateral part of area 6 shows no increase in activity (Roland et al., 1980). This is consistent with the notion that the premotor cortex in the lateral part of area 6 monitors feedback from ongoing movement. The anatomical connections of area 6 are also consistent with this suggestion.

Area 6 receives highly processed sensory information from the neighboring prefrontal cortex, which functions as a high-level association field (Fuster, 1981), from visual area 19 (Pandya & Kuypers, 1969), from the supplementary sensory cortex of area 5 (Jones & Powell, 1969), and from a prominent somatotopically organized input from somatosensory areas I and II (Bowker, Murray, & Coulter, 1979; Jones & Powell, 1969, 1970). Area 6 also receives afferent connections from motor cortex area 4 and from VPLo, VA, and VL nuclei of the thalamus (Bowker et al., 1979; Wiesendanger, 1981). The output neurons of area 6 project heavily on tegmental regions of the brainstem (see Kuypers, 1981) and can influence information processing in the basal ganglia and cerebellum (Allen & Tsukahara, 1974; Dhanarajan, Ruegg, & Wiesendanger, 1977; Oka & Jinnai, 1978; Wiesendanger, Wiesendanger, & Ruegg, 1979). Thus, the premotor cortex is appropriately connected to integrate high-level sensory and motor feedback signals and to modulate the transformations of neural signals through the basal ganglia and cerebellum.

Damage to periventricular white matter commonly observed in cerebral palsy (Cohen & Duffner, 1981; Koch et al., 1980; Kulakowski & Larroche, 1980; Pape & Wigglesworth, 1979) could disrupt pathways in these subcortical circuits. In particular, lesions of the cerebellothalamocortical fibers to area 6 would, according to the above hypothesis, produce fundamental problems in motor programming. Damage to these cerebellothalamocortical fibers would prevent the premotor cortex from receiving copies of the neural input to the motor cortex from the cerebellum. Consequently, the premotor cortex would be unable to establish accurate internal models of relationships between motor commands and their perceptual consequences. Moreover, since the lesions in cerebral palsy occur before, during, or shortly after birth, the CNS would be deprived of an essential sensorimotor integration process during the normal period for development of motor skills. This would manifest itself during motor programming as an inability to transform desired perceptual effects into the appropriate motor commands to achieve them. Although a cerebral-palsied person would appreciate precisely the desired perceptual consequences of a movement, he/she would be unable to transform this appreciation into an appropriate input to the motor cortex to achieve them.

A further implication from the above formulation may be noted in respect of athetoid movement. The hypothesis incorporates the notion of feedback of motor com-

mands (efference copy in the terminology employed by Von Holst & Mittelstaedt, 1950) as a fundamental postulate. Disruption of such internal feedback of motor commands is seen as the core problem in cerebral palsy. An increase in efference copy discharge has been suggested by Matthews (1982) to account for the fact that muscle fatigue increases the apparent heaviness of a given weight (McCloskey, Ebeling, & Goodwin, 1974). The sensation of increased heaviness is attributed to the fact that the motor drive to the fatigued muscle must be increased to produce the same contractile force and hence there is an enhancement of associated efference copy discharge. In the event of a reduction in the efference copy discharge associated with a movement, however, something akin to a reduction in the sensation of heaviness or in the sense of effort may occur. Moreover, if a reduction in efference copy discharge were to occur due to disruption of the neural pathways mediating feedback of motor commands, then the perceptual feedback from the movement could not be correlated with the motor commands that produced it. Hence, the movement would be interpreted as arising from unknown sources—in other words, as involuntary. In athetoid cerebral palsy, therefore, disruption of internal feedback of motor commands might lead not only to the generation of inappropriate movement, but also to such movement being experienced as involuntary by the athetoid individual.

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