Pathophysiology of dysarthria in cerebral palsy

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SUMMARY Electromyograms were recorded with hooked-wire electrodes from sixteen lip, tongue and jaw muscles in six normal and seven cerebral palsied adult subjects during a variety of speech and non-speech tasks. The recorded patterns of muscle activity fail to support a number of theories concerning the pathophysiology of dysarthria in cerebral palsy. There was no indication of weakness in individual articulator muscles. There was no evidence of uncontrolled sustained background activity or of abnormal tonic stretch reflex responses in lip or tongue muscles. Primitive or pathological reflexes could not be elicited by orofacial stimulation. No imbalance between positive and negative oral responses was observed. The view that random involuntary movement disrupts essentially normal voluntary control in athetosis was not supported. Each cerebral palsied subject displayed an idiosyncratic pattern of abnormal muscle activity which was reproduced across repetitions of the same phrase, indicating a consistent defect in motor programming.

There has been little experimental verification of existing theories concerning the pathophysiology of dysarthria in cerebral palsy. The present study provides electromyographic (EMG) data in the light of which these theories can be examined.

Dysarthria in cerebral palsy has been attributed to impaired control of respiration,¹⁻⁹ laryngeal muscles,^{4 8 10 11} pharyngeal muscles,^{5 12} opening and closing of the velopharyngeal port^{2 4 10 11 13-17} and muscles of articulation.^{10 17-21} In short, malfunction has been reported in every aspect of the speech mechanism. It would seem that the basic cause of the speech defect in cerebral palsy is not located in any one of these systems in particular, but is an underlying neuromuscular control problem central to them all.

The pathophysiology of dysarthria in cerebral palsy is uncertain but a number of theories are current.²² These can be summarised as follows: inefficient valving of the air stream caused by a generalised paresis of speech musculature;²⁻⁴ abnormalities of tone due to sustained background activity¹⁵ or spasticity of speech muscles;^{5 11 12 23} primitive or pathological reflexes interfering with articulatory control;^{19 24} imbalance between positive and negative oral responses caused by cortical lesions;^{5 25 26} and

disruption of the voluntary control of speech muscles by random involuntary activity of the type associated with athetosis.^{6 12 15}

EMG studies of the speech musculature in cerebral palsy are practically non-existent.^{15 17 27} In the present study EMG activity was recorded simultaneously from sixteen lip, tongue and jaw muscles during both speech and non-speech tasks in normal and cerebral palsied adult subjects. Certain features of the muscle activity were found consistently in all of the cerebral palsied subjects. The purpose of the present report is to describe these consistent features with reference to the five theories of dysarthria in cerebral palsy mentioned above.

Methods

Subjects

The study involved seven young adult cerebral palsied subjects, aged 19-34 years, and six young adult normal subjects, aged 20-30 years. All cerebral palsied subjects were severely disabled in all four limbs. Five were predominantly athetoid and two were predominantly spastic. There was no loss of sensation in any of the subjects. A normal to brisk jaw jerk was present in all subjects and pouting was present in two. Facial grimacing was present in all five of the athetoid subjects and a typical clasp-knife spasticity was present in the lower limbs of both spastic subjects. All had dysarthric speech to the extent that either they could just be understood by those familiar with their speech, while remaining unintelligible to others, or they were completely unintelligible and

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required a point board for communication. None of the cerebral palsied subjects was mentally retarded. They all had normal hearing as revealed by audiometric testing and they had no difficulty in understanding speech.

Procedure

The method of inserting the electrodes has been described elsewhere²⁸ and will be mentioned only briefly here. The recordings were obtained from copper bipolar hooked-wire electrodes with 1 mm non-insulated tips. The EMG signals were amplified in Devices 3160 EMG preamplifiers, displayed on oscilloscopes and recorded on a Philips Ana-Log 14 FM tape recorder running at a tape speed of 15 inch/s (38.1 cm/s) (frequency response DC-5 kHz). The subjects' voice signals were recorded on a high quality audio channel added to the FM tape recorder.

Electrodes were inserted into the following muscles: levator labii superioris (LLS), zygomaticus major (ZYG), buccinator (BUCC), risorius (RIS), orbicularis oris superioris (OOS), orbicularis oris inferioris (OOI), depressor anguli oris (DAO), depressor labii inferioris (DLI), mentalis (MENT), anterior genioglossus (GG), geniohyoideus (GH), mylohyoideus (MH), anterior belly of the digastricus (ABD), internal (medial) pterygoid (IP), tongue intrinsic (INT) and styloglossus (SG). After the electrodes were inserted into the muscles the subjects performed a sequence of non-speech gestures for which they had been trained in front of a mirror before the experiment started. These gestures were selected to activate specific "target" muscles and have been described elsewhere.²⁸ In addition, the subjects were trained to produce calibration gestures of maximal contraction of the target muscles; that is, they were asked to carry out each gesture again but to intensify the contraction to a maximum degree regardless of activity in neighbouring muscles. This procedure was designed to produce the largest possible EMG potentials for any given muscle. Maximum EMG activity recorded in each muscle was designated as 100% and activity recorded subsequently was expressed as a percentage of this maximum level.

After the patterns of EMG activity had been recorded during the gestures the subjects next repeated a number of speech samples which they had practised prior to the experiment and which were written on cards and displayed to them during the experiment. Fifteen isolated words and four test phrases were each repeated either three or five times and the test sentence "Do all the old rogues abjure weird ladies" was repeated fifty times by each subject. The calibration gestures were then repeated and recorded again so that any changes in the EMG signal due to movement of the electrodes could be detected. The few muscles for which movement of the electrodes was suspected were dropped from the study. Following the recording of the speech samples and the second calibration procedure, an investigation of tonic stretch reflexes in lip, tongue and jaw muscles, which has been described elsewhere,²⁹ was carried out. On completion of this investigation all of the calibration gestures were recorded again.

An attempt was made to elicit primitive and pathological reflexes in the cerebral palsied subjects, using techniques similar to those described by Mysak,³⁴ Sheppard³⁰ and Love, Hagerman and Taimi.³¹ Tickling,

stroking, rubbing and tapping were applied to the subjects' head, neck, cheeks, lips, gums, tongue and palate. The lobes of the ears were rubbed and the nostrils were tickled. Light stroking with the finger was applied to the angle of the mouth in an attempt to produce lowering of the corresponding half of the lower lip and to the lips and cheeks in an attempt to elicit a rooting response (that is, turning the head in response to stimulation of the cheek).

For purposes of quantification and visual inspection, the recorded EMG signals were played back from the tape recorder at a reduced speed of $3\frac{1}{2}$ inch/s 9.4 cm/s to Grass 5P3 EMG preamplifiers switched to "integrator" mode, where the signals were full-wave rectified and low-pass filtered to yield integrated EMG (IEMG) signals. The time constant of the IEMG filter was modified to 0.08 s, but with the 4:1 reduction in tape speed this was equivalent to a time constant of 0.02 s. The IEMG signals were recorded on a four-channel Grass 5D polygraph (paper speed 5 or 10 mm/s). The high frequency filter of the driver amplifier was set at 3.0 Hz which, with the reduced tape speed, gave an effective filter setting of 12.0 Hz. These time constant and filter settings were chosen to provide the best compromise between smoothing the rectified EMG signal and reproducing rapid changes in muscle activity. Tracings of the unprocessed EMG signals were obtained by replaying the FM tape recorder at the reduced speed of $3\frac{1}{2}$ inch/s (9.5 cm/s) to a Siemens-Elema Mingograf (bandwidth 5.0 kHz).

Results

Maximal activity of the target muscles was produced during the calibration gestures, although in both subject groups there was often maximal or near maximal activity in other muscles as well. Moreover, these maximum contractions were reproduced by each subject during the course of the experiment, as described above and the contortions of the face accompanying them were characteristic of forceful contractions. Although the voltage levels of the EMG varied between electrodes, probably because of variation of spacing between electrode tips within the muscles, no differences between the groups were detected in either the magnitude or the quality of the EMG interference patterns recorded during maximal activity. The EMG recordings showed the large amplitude spikes and strong interference patterns expected during maximal contractions.

Activity in articulator muscles during speech in cerebral palsied subjects was similar, or more often greater in amplitude than in the normal controls (figs 1, 2 and 3). Peak levels of IEMG activity were usually in the range 30-100% of maximum during speech in cerebral palsied subjects compared with peak levels of 5-50% in normal subjects. A muscle producing a normal level of activity during one test phrase or word sometimes showed an excessive level of activity in a different test phrase or word. However, there was no evidence of a pathological imbalance in levels of activity between muscles involved

Fig 1 IEMG recordings of a lip closing (OOI) and a lip opening (DLI) muscle from a normal (N) and a predominantly spastic cerebral palsied (CP) subject during two repetitions of the test phrase "I live in Emu Plains". The 0% and 50% marks represent the level of IEMG activity relative to the maximal activity recorded during calibration gestures.

Fig 2 IEMG recordings of a number of facial muscles from a predominantly athetoid cerebral palsied subject during two repetitions of the test phrase "That's an eagle a month". The figure illustrates how spasms were clearly detectable in both speech and IEMG recordings. The 0% and 100% marks represent the level of IEMG activity relative to the maximal level recorded during calibration gestures. (a) Test phrase disrupted by a spasm. (b) Repetition of the same test phrase in the absence of a spasm, where the pattern of IEMG activity was still abnormal and the speech dysarthric.

Fig 3 IEMG recordings of twelve lip, tongue and jaw muscles from a predominantly athetoid cerebral palsied subject during two successive repetitions of the test sentence "Do all the old rogues abjure weird ladies". The figure illustrates how the abnormal pattern of IEMG activity was reproduced during repetitions of the same utterances by the same subject. 0% and 100% marks as in fig 2.

in positive oral responses (lip closing) and muscles involved in negative oral responses (lip opening) (fig 1).

There was no evidence of uncontrolled sustained background activity in any of the muscles studied. Subjects were able to relax all muscles when they were not speaking and could modulate the level of activity in all muscles during speech. In fact, the

IEMG activity often fell to zero or near zero levels during speech (figs 1, 2 and 3).

No primitive or pathological reflex responses were observed in response to tickling, stroking, rubbing or tapping 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. A sudden loud noise, such as produced by a handclap, often produced a startled response. Although most of the cerebral palsied subjects experienced spontaneous spasms involving the speech musculature during the course of the experiment, such spasms were not produced in response to stimulation of the type mentioned above.

Spasms and other involuntary movements which occurred spontaneously, such as gagging, tongue thrusting and coughing, were clearly detectable in both the speech and IEMG recordings, since speech was halted transiently and a different pattern of muscle activity was generated during the involuntary movement (fig 2a). Many recordings of each test utterance which were not interrupted in this way were obtained from each subject (fig 2b). During these utterances the speech remained dysarthric and largely unintelligible, and the patterns of IEMG activity were abnormal (figs 1, 2b and 3).

For any one test utterance each cerebral palsied subject produced an idiosyncratic pattern of abnormal muscle activity. There was, nevertheless, an unexpected degree of reproducibility in such abnormal patterns, for repetitions of the same speech material by the same subject (figs 1 and 3). Except for occasional disruptions such as in fig 2a, the abnormal pattern of IEMG activity in each cerebral palsied subject was reproduced for the fifty repetitions of the test sentence "Do all the old rogues abjure weird ladies" (fig 3).

Discussion

As IEMG increases with force of contraction^{32 33} and the amplitudes of the IEMG recorded during speech in the cerebral palsied subjects were the same or higher than in the normal controls, there was no evidence that dysarthria in these subjects was caused by weakness of articulator muscles. As judged from EMG recordings, the cerebral palsied subjects produced a maximum contraction in each articulator muscle on request. Thus, there was no evidence of weakness in individual lip, tongue or jaw muscles. However, this does not preclude the possibility that inappropriate contractions of antagonist muscles might produce a "functional weakness" by reducing the effectiveness of the agonist muscles.

Abnormalities of tone of the speech musculature have been suggested as contributing to dysarthria.^{15 23} Spasticity is characterised by abnormalities of tonic stretch reflexes.³⁴ Since, in a previous study at this laboratory,²⁹ it was demonstrated that tonic stretch reflex responses are not present in lip or tongue muscles of either normal or cerebral palsied subjects, dysarthric speech in cerebral palsy cannot

be caused by spasticity in these muscles. If there is an abnormal reflex stiffness of lip and tongue muscles in cerebral palsy, it must be produced by reflexes other than tonic stretch reflexes. For example, the second component of the perioral reflex response^{35 36} has been shown to have an abnormal amplitude in athetosis and not to habituate as it does in normal subjects.¹⁵ However, the contribution of the perioral reflex to mechanical stiffness of the lips has yet to be elucidated.

Netsell¹⁵ suggested that tone is manifest in speech muscles as a degree of background contraction upon which speech movements are superimposed. This is comparable with Leanderson's³⁷ observation that background activity is increased and sustained in some facial muscles in Parkinson's disease. In the present study, however, there was no evidence of sustained background activity of this kind. It seems reasonable to conclude that dysarthria in these adult cerebral palsied subjects is not caused by rigidity due to sustained background activity in lip, tongue or jaw muscles.

It has been argued that dysarthria in cerebral palsy is caused by a variety of released or retained primitive or pathological reflex responses.^{19 24 30 38} For this reason, desensitisation of the orofacial region is considered important in speech therapy.¹⁹ However, such responses have been shown to decrease with age and to be absent in many adults with cerebral palsy.³¹ Moreover, when such responses are present they cannot be elicited consistently and there is no systematic relationship between the number of abnormal reflexes present and the level of articulatory proficiency.³¹ In the present study, primitive or pathological reflex responses could not be elicited in adult subjects by direct orofacial stimulation, despite the presence of severe dysarthria. It is apparent, therefore, that primitive or pathological reflex responses are not the primary cause of dysarthria in adults with cerebral palsy.

The suggestion that dysarthria in cerebral palsy is caused by an imbalance between positive and negative oral responses^{5 26} was not supported in this study, in that no imbalance in levels of activity was detected between lip opening and lip closing muscles during speech.

Although spasms, gagging, tongue thrusts and coughing interrupted speech, as illustrated in fig 2a, these cannot constitute the principal cause of dysarthria, since their occurrence was intermittent and irregular while speech remained dysarthric throughout. In the absence of such responses the pattern of abnormal IEMG activity was reproducible in repetitions of the same test utterance by the same subject (fig 3). This reproducibility was unexpected since five of the seven cerebral palsied subjects were classi-

fied on neurological examination as athetoid dysarthrics. The involuntary movements of athetosis have not been described as reproducible; on the contrary, they have been considered to lack fixed amplitude, rhythmicity or direction³⁹ and have been described as extremely variable and irregular.^{40 41} The theory that athetoid dysarthria results primarily from disruption of voluntary articulator control by superimposed random involuntary movements^{6 12 15} is not supported by our findings. However, this does not exclude the possibility that speech might be disrupted by consistently triggered involuntary movements.

The timing and sequence of muscle activity was abnormal in each of the cerebral palsied subjects and discrete use of individual muscles was impaired. Timing is probably a major factor contributing to lack of intelligibility in dysarthric speech, as was concluded previously.^{42 43} However, the reproducibility of patterns of abnormal muscle activity suggests that individual cerebral palsied speakers should make consistent speech errors. This is supported by the observation that many cerebral palsied speakers can be understood by those familiar with their speech patterns while remaining unintelligible to others. The fact that listeners appear to be able to learn a "translation code" suggests the feasibility of developing speech analysis and synthesis devices^{44 45} to improve intelligibility of speech in cerebral palsy.

The question remains, what is the primary cause of dysarthria in cerebral palsy? The reproducibility of patterns of abnormal IEMG activity across repetitions of the same speech material by the same subject suggests that the same inappropriate motor commands must be formulated centrally each time the speaker attempts to produce the same utterance. Dysarthria in cerebral palsy might result, therefore, from the distortion of motor commands by transmission through damaged descending pathways, or because appropriate motor commands are not correctly formulated in the first place. In the latter case the speech defect can be viewed as a motor learning deficit, as suggested by Kent and Netsell¹⁷ in the case of athetosis. If motor commands are generated on the basis of previously learned correlations between motor events and their sensory consequences, as argued by many movement control theorists,⁴⁶⁻⁵³ then impairment of sensory-motor integration processes involved in establishing such correlations should disrupt the ability to formulate appropriate motor commands. Periventricular lesions of the type commonly found in cerebral palsy^{54 55} could disrupt the flow of neural activity from the basal ganglia and lateral zones of the cerebellum to the motor and premotor cortex,⁵⁶ and thereby impair sensory-motor integration in the

premotor cortex. Since these lesions affect the immature nervous system, the cerebral palsied infant may be deprived of normal sensory-motor learning mechanisms during development of speech. The residual or compensatory learning processes available could result in the idiosyncratic abnormal patterns of muscle activity seen in this study.

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