

Oral Dyspraxia in Inherited Speech and Language Impairment and Acquired Dysphasia

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Half of the members of the KE family suffer from an inherited verbal dyspraxia. The affected members of the family have a lasting impairment in phonology and syntax. They were given various tests of oral praxis to investigate whether their deficit extends to nonverbal movements. Performance was compared to adult patients with acquired nonfluent dysphasia, those with comparable right-hemisphere lesions, and age-matched controls. Affected family members and patients with nonfluent dysphasia were impaired overall at performing oral movements, particularly combinations of movements. It is concluded that affected members of the KE family resemble patients with acquired dysphasia in having difficulties with oral praxis and that speech and language problems of affected family members arise from a lower level disorder. © 2000 Academic Press

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INTRODUCTION

Of 30 members of the KE family in three generations, 15 are affected with a speech-and-language deficit that has been characterized as a verbal dyspraxia (Hurst et al., 1990; Vargha-Khadem et al., 1995, 1998). Each affected family member has one affected parent, and the disorder occurs in

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both sexes in approximately equal numbers. Pembrey (1992) has suggested an autosomal dominant mode of inheritance, with full penetrance. Recently, a linkage study has mapped the defective gene (SPCH 1) to a small region on chromosome 7q31 (Fisher et al., 1998). Finally, neuroimaging studies have revealed both structural and functional abnormalities in several cortical and subcortical motor-related areas (Vargha-Khadem et al., 1998).

A linguistic account of the speech-and-language disorder affecting the KE family has been provided by Gopnik and colleagues (Gopnik, 1990; Gopnik & Crago, 1991). They have suggested that the affected family members fail to use or comprehend regular grammatical morphemes in the normal manner due to an underlying inability to master a selective part of the grammar, namely morphological features. They term the disorder "feature blindness" and suggest that this disorder is a selective one. Pinker (1994), among others, has suggested further that this selective disability in the use of syntactic forms, resulting from a genetic abnormality, is evidence for a specific biological basis for grammatical ability.

An alternative account of the disorder in affected family members emphasizes instead their dyspraxia, in particular their impairment in speech articulation (Vargha-Khadem et al., 1995). Dewey et al. (1988) studied a group of children with impaired articulation and found that those with a deficit in verbal sequenced motion rate (SMR) were also impaired on the performance of sequenced oral and limb movements. Other children with impaired articulation but with no deficit in SMR had no problems with sequenced motor tasks. The suggestion was made that children with an impairment in speech articulation can be divided into two groups: those with a higher level praxis disorder, encompassing all modalities, verbal and nonverbal oral movements as well as limb movements, and those with a higher level phonological disorder, affecting speech planning but not other motor performance. The first aim of the present study was to examine this issue in the affected family members by determining whether their articulatory deficit extends to the performance of nonverbal oral movements.

A second aim of this study was to compare the affected family members to adult patients with speech impairments due to unilateral hemispheric damage in order to determine how closely the impairments in the two groups resemble each other. Pinker (1994) has claimed that the pattern of linguistic deficits found in affected family members does not resemble adult patients with aphasia. Most studies, however, show that adult patients with Broca's or nonfluent aphasia have some degree of oral apraxia. Thus, Mateer and Kimura (1977; Mateer 1978) and Kimura and Watson (1989) found that nonfluent aphasics were impaired on oral movements and rapid sequencing of syllables. There could be two different reasons for these impairments. First, the mechanisms for speech and the control of the mouth may be intrinsically linked (Kimura, 1993). Alternatively, these mechanisms may be sepa-

rate, but located in neighboring regions. Mohr et al. (1978) have shown that small lesions that are mainly restricted to Broca's area (Brodmann area 44) do not result in a lasting disturbance of language, but can cause loss of control of oral movements, whether in speech or nonspeech contexts. Tognala and Vignolo (1980) identified the frontal operculum and anterior insula as critical for oral apraxia, and, more recently, Dronkers (1996) has suggested that there is an area in the insula that is specialized for the motor planning of speech. By studying subjects with inherited as well as acquired speech-and-language disorders, one can test whether there is a necessary association between the control of speech and oral movements.

In choosing which oral movements to test, we thought it important to contrast simple and complex movements. Clinical tests of oral apraxia tend to use complex movements involving several different muscle groups (e.g., sticking out the tongue, which involves first opening the mouth and then sticking out the tongue, or humming, which involves closing the mouth and phonation). The movements are generally elicited to command or, sometimes, to imitation, but not usually to both. Square-Storer et al. (1989) investigated the performance of aphasic and normal control subjects on simple movements, and they found significant impairment of performance by aphasic subjects equally in all modes of elicitation.

In examining apraxia of speech, Miller (1989) noted that patients do not necessarily have problems with force, speed, and range of movements. Rather, targets are overshoot and there is an impairment in temporospatial programming. We therefore included sequences of oral movements and measured the overall speed of the movements in the test battery. Although, for the subjects with acquired dysphasia, a facial hemiparesis may well slow movement, the affected family members show no such gross evidence of neuromuscular weakness.

In addition, Blumstein (1990) suggests that some aspects of speech timing are impaired in acquired nonfluent aphasia. Voice-onset time in stop consonants was significantly impaired (Blumstein et al., 1980), as was the timing of the release of the velum in nasal consonants (Ryalls, 1986), and the onset of voicing relative to fricative noise was incorrectly timed (Harmes et al., 1984). The problem appears to be with simultaneous movement of two oral structures. We therefore also examined simultaneous oral movements in the affected family members and the adult patients.

Borod et al. (1988) studied the effect of an emotional context on the performance of oral movements by adult left- or right-hemisphere-damaged patients. The same movements were found to be better performed when an emotional context was present than when it was not. It was suggested that this is due to the differential hemispheric representation of material with emotional content, but it is possible that the presence of a context per se makes movements easier for individuals with dysphasia. To examine this

issue, we included both emotional and nonemotional contexts so as to separate the effect of a context from that of emotion.

Thus, in the test battery of oral movements, we included movements which require the subject to use various parts of the oral musculature singly and in combination, in sequence and simultaneously, and with and without an emotional and nonemotional context. In studying members of the KE family, we sought to determine the selectivity of the impairment caused by the defective gene. In comparing their performance to that of normal, age-matched controls and to that of adult patients with unilateral hemispheric lesions, we aimed to see if impairments in subjects with an inherited speech-and-language disorder did or did not resemble those in subjects with acquired disorders.

METHODS

Subjects

Twelve affected members of the KE family were studied (7 female, 5 male). Their mean age was 27.08 years ($SD = 21.21$). Their mean Performance IQ (PIQ), measured on the WAIS-R (Wechsler, 1981), WISC-R (Wechsler, 1976), or WISC-III UK (Wechsler, 1992), was 80.7 ($SD = 14.49$). Their mean length of utterance (MLU) as measured on the Bus Story Test (Renfrew, 1977) was 7.11 ($SD = 0.76$). Not all affected family members were tested on the Bus Story, and, of those tested, most were adults. However, this test provides a measure of MLU comparable to that provided by the Cookie Jar story (Goodglass & Kaplan, 1982), which was given to subjects with acquired dysphasia due to left hemisphere damage, and hence the Bus Story data are included for purposes of comparison.

Performance of affected family members was compared to that of 59 age-matched normal controls with a mean age of 25.8 years ($SD = 20.8$). These control subjects included school-age children attending one of four different schools in three varied areas of Oxford, adult volunteers from the Department of Experimental Psychology's Subject Panel, and adults recruited through an employment agency. The mean PIQ of the control subjects was 104.9 ($SD = 9.79$), significantly higher than that of the affected members.

The patients with acquired lesions consisted of 9 adults with lesions of the left hemisphere (Group LH, mean age 57.44, $SD = 18.27$), and 10 with lesions on the right (Group RH, mean age 63.00, $SD = 7.52$). All patients were right-handed, and all were tested 2 months or more after their lesions, which were unilateral cerebrovascular accidents, except for one case with a gunshot wound. The patients were selected on the basis of having left- or right-sided hemiplegia and, in the case of the LH group, of having aphasia. This was primarily nonfluent dysphasia accompanied by some expressive speech difficulties. At the time of testing, all of the patients also had some oral movement difficulties, manifested either as "literal paraphasia" (phoneme substitutions; Goodglass & Kaplan, 1982) or as dyspraxic errors made on the rapid oral movements subtest of the Boston Diagnostic Examination of Aphasia (BDEA) (Goodglass & Kaplan, 1982). The MLU, as calculated from description of the Cookie Theft picture from the BDEA, was 4.58 ($SD = 2.42$). Patients with right-hemisphere damage were not aphasic, as assessed on the basis of the Frenchay Aphasia Screening Test (FAST) (Enderby, & Crow, 1996). Typical lesions in the adult patients are shown in Fig. 1. The diagrams are based on drawings of the lesions on standard sections taken from Damasio and Damasio (1989). Lesion diagrams and descriptions for all the patients are available in Alcock (1995).

The performance of these patients with lesions acquired in adulthood was compared to that of 26 age-matched normal controls (mean age 61.96, $SD = 10.28$). The control subjects were

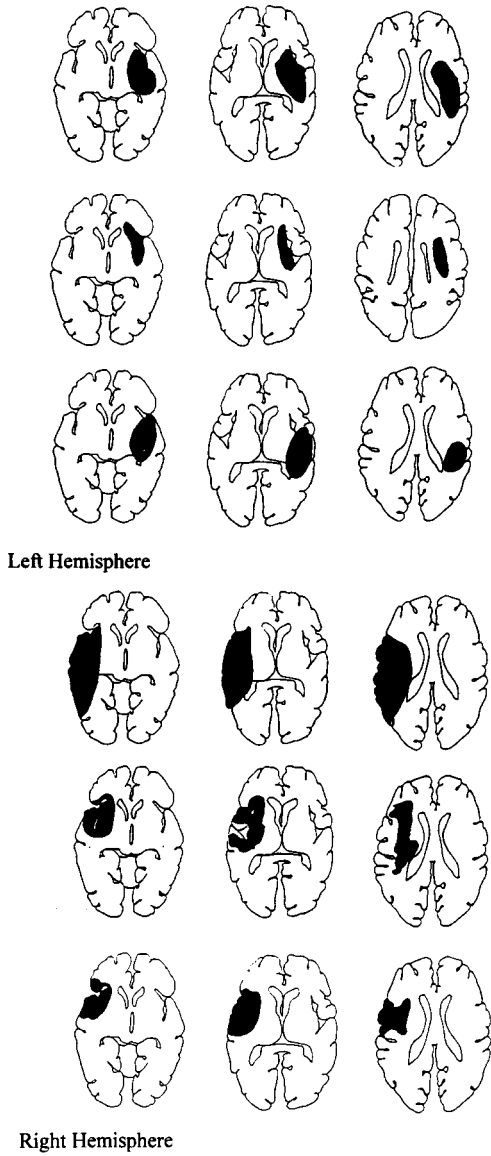


FIG. 1. Lesions of six adult patients.

TABLE 1
Screening Data from Affected Family Members and Controls

Subject	Sex	Age	Years of education completed	MLU on bus story	Score on animal naming task
AF1	F	13			
AF2	F	14			18
AF3	M	14		6.2	23
AF4	M	11			15
AF5	F	11			25
AF6	F	10			16
AF7	F	41	11	8.6	33
AF8	M	21	11	7.1	21
AF9	F	48	11	7.3	26
AF10	F	78	9	6.6	14
AF11	M	44	11	7.2	14
AF12	M	20	12	6.8	21
Mean		27.08	10.83	7.11	20.54
<i>SD</i>		21.21	1.27	0.76	5.56
Controls, mean		18.2716	11.35		
Controls, <i>SD</i>		11.3941	1.07		

members of the Department of Experimental Psychology's Subject Panel, members of the support staff of the Department, spouses of patients in the unilateral brain lesion group, subjects recruited by a local employment agency, and unaffected members of the KE family.

Data on screening tests for all patients together with summary data for control subjects are shown in Tables 1 and 2.

Procedures

The subjects were seated facing the experimenter. All movements made by the subject as well as the instructions of the experimenter were recorded on video for later scoring and analysis.

Subjects were required to perform five types of movements, first performing the movements to verbal instructions and then after the instruction to imitate the tester. The movements were as follows.

Eight single, simple movements involving the use of only one set of muscles (for example, "make an 'ah' sound," involving only the vocal chords) and nine single, complex movements involving the use of more than one set of muscles (for example, "stick out your tongue," involving both jaw muscles and tongue muscles).

Eleven sets of simultaneous movements, each set consisting of three single movements carried out at the same time (for example, "open your mouth wide, stick out your tongue and make an 'ah' sound"); 11 sets of sequential movements, each set consisting of three single movements carried out one after the other (for example, "first open your mouth wide, then close your lips tightly together, then make an 'ah' sound"); and finally, 11 repeated single movements (for example, "put your front teeth together again and again").

In addition, all of the single, simple and single, complex movements were also made to

TABLE 2
Screening Data from Left and Right Unilateral Lesion Patients and Controls

Subject	Sex	Age	Time since onset (months)	Years of education	FAST score (C + E)	MLU on cookie jar story	Score on animal naming task
LH1	M	53	18	11		2.75	0
LH3	M	60	486	12		7.86	13
LH4	F	79	7	9		4.67	10
LH5	F	36	22-26	10		6.00	12
LH7	M	60	15-26	10		1.50	6
LH9	M	72	8-10	9			6
LH10	F	30	98	11		5.60	7
LH11	M	82	64-65	13		6.35	6
LH16	M	43	4	11		7.70	12
Mean		57.22	81.11			5.30	8.00
<i>SD</i>		18.48	154.97			2.25	4.15
RH2	F	61	14	9	16		
RH3	F	75	14-23	10	17		
RH4	M	62	16-27	13	20		
RH5	M	50	12-24	10	18		
RH8	F	75	10	11	19		
RH9	F	64	5	10	18		
RH10	M	59	10	11	19		
RH11	F	60	3	10	16		
RH12	F	63	15		18		
Mean		63.22	12.78	10.5	17.71		
<i>SD</i>		7.8	6.22	1.20	1.50		
Controls, mean		63.04		12.28			
Controls, <i>SD</i>		9.34		2.53			

command with a context. Contexts could be either emotional (for example, "stick out your tongue as if you were making a rude face") or nonemotional (for example, "stick out your tongue as if you were showing it to the doctor"). The order of presentation of emotional and nonemotional contexts was varied in a pseudorandom order. The full list of movements is given in Alcock (1995).

The subjects were requested to carry out each movement after the experimenter had either described it verbally or presented it for imitation. Movements of a given type were tested first to command, then (for the single movements) to command with context, and last to imitation. The different types of movement were tested in the order listed above.

Scoring

The videotaped movements were scored a system of error notation based on that of Square-Storer et al. (1989). The scoring system requires comparing each movement against a large number of possible errors, including perseveration, repetition, augmentation, and omission. Although error types were not analyzed statistically, the focus on error types ensured accurate

scoring: the movements of 10% of the subjects were scored independently by two raters, and interrater agreement was found to be 85%.

RESULTS

Affected Family Members

A MANOVA was carried out with Group (affected family members and controls), Movement Type (simple, complex, simultaneous, and sequential), and Presentation (to command and to imitation) as the factors. All main effects were found to be significant, indicating that (a) the affected family members performed more poorly than the controls (Group: $F = 63.40$, $df = 1$, $p < .001$), (b) the more complex the movement, the poorer the performance (Movement Type: $F = 12.21$, $df = 3$, $p < .001$), and (c) movements made to command were poorer than those made to imitation (Instruction: $F = 82.18$, $df = 1$, $p < .001$). These results are illustrated in Figs. 2–4. In addition, the interaction between Group and Movement Type was significant ($F = 8.17$, $df = 3$, $p < .001$), indicating that although the affected family members were impaired overall, they showed little or no impairment on the single, simple movements. This is supported by post hoc comparisons showing that they were not significantly worse than controls on single, simple movements made either to command ($t = 1.98$, $df = 66$, $p > .05$) or to imitation ($t = 2.00$, $df = 12.38$, $p > .05$). Finally, the interaction between Movement Type and Presentation was significant ($F = 3.38$, $df = 3$, $p < .01$), demonstrating that imitation aided performance of the combined movements (simultaneous and sequential) more than that of the others.

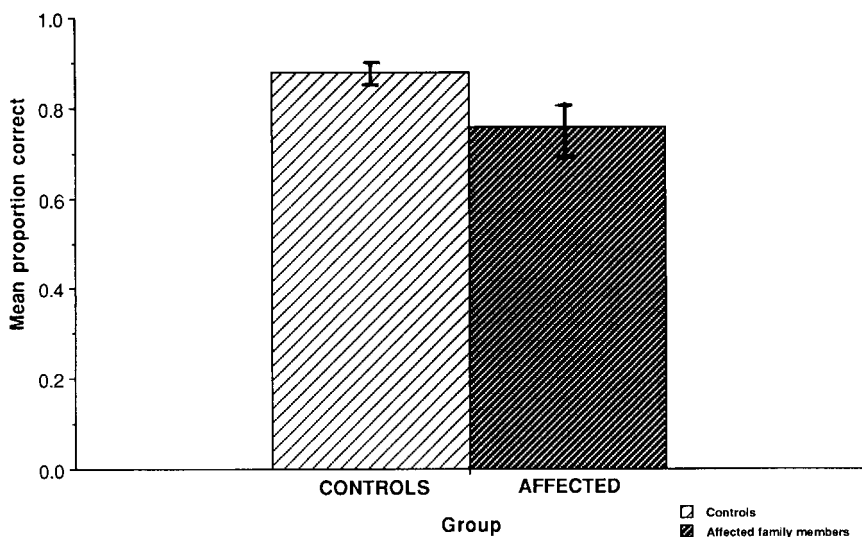


FIG. 2. Overall proportion correct for affected family members and normal controls.

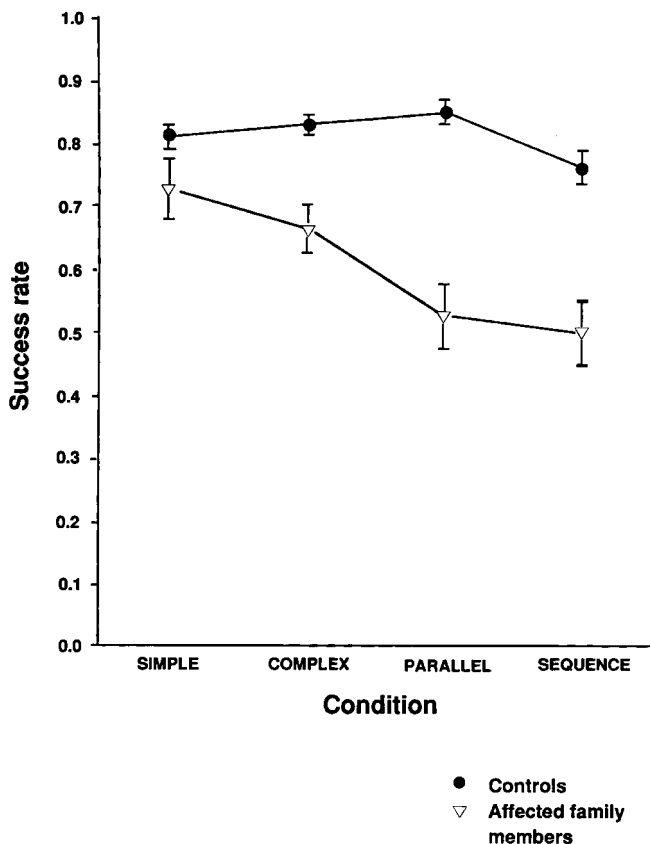


FIG. 3. Movements performed to command. Proportion correct in different conditions for affected family members and normal controls.

A MANOVA was also carried out to determine whether a context aided performance of the single movements (simple and complex). There were main effects of Group ($F = 17.32$, $df = 1$, $p < .001$) and of Context ($F = 7.71$, $df = 1$, $p = .01$), but there were no interactions. Thus, context aided performance, but not more so for the affected family members than for the controls.

Finally, MANOVAs were performed to determine whether the groups differed on the repeated movements in terms of either errors or speed. For errors, there was a main effect of Group ($F = 5.04$, $df = 1$, $p < .01$), indicating that the affected family members were again worse than the controls; and for speed, there was a main effect of Instruction ($F = 5.80$, $df = 1$, $p < .01$), movements performed to imitation being faster than those performed to command.

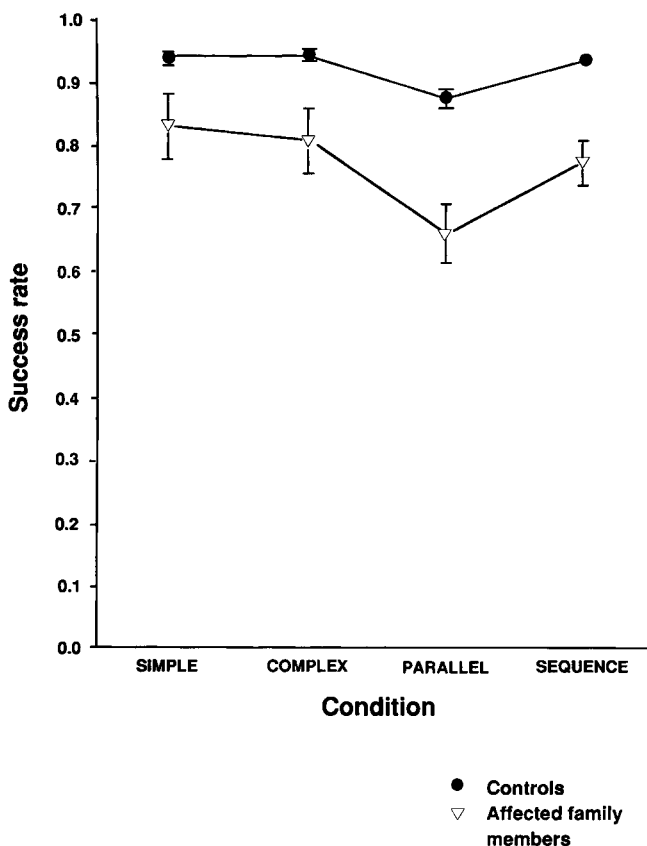


FIG. 4. Movements performed to imitation. Proportion correct in different conditions for affected family members and normal controls.

Patients with Acquired Unilateral Lesions

A MANOVA was carried out with Groups (LH, RH, and controls), Movement Type, and Presentation as the factors. Again, all main effects were significant: Group ($F = 13.39$, $df = 37$, $p < .001$), Movement Type ($F = 21.83$, $df = 3$, $p < .001$), and Presentation ($F = 60.90$, $df = 1$, $p < .001$). In addition, interactions were found between Group and Movement Type ($F = 5.33$, $df = 6$, $p < .001$), the patients performing differentially worse than the controls on the combined as compared with the single movements, and between Movement Type and Presentation ($F = 17.80$, $df = 3$, $p < .001$), with imitation aiding performance of the combined more than that of the single movements. The results are illustrated in Fig. 5. (Of the nine patients in the LH group with acquired dysphasia, four were so impaired that they were unable even to attempt either the simultaneous or sequential move-

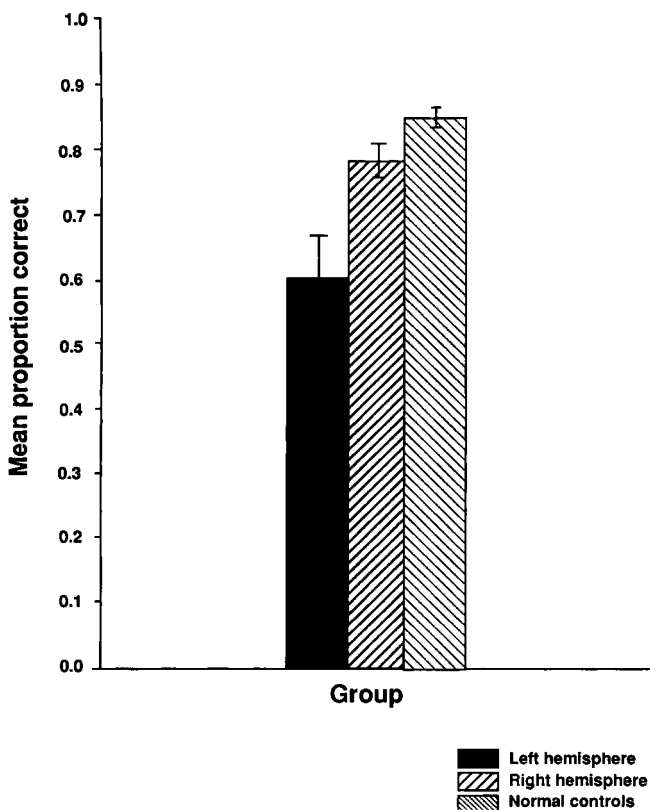


FIG. 5. Overall proportion correct for patients with LH damage, patients with RH damage, and normal controls.

ments. The data for the five remaining subjects are presented in Figs. 6 and 7, which show performance to command and to imitation, respectively.)

A separate MANOVA comparing just the two patient groups revealed that although the effect of Group was not significant, there were significant interactions between Group and Movement Type ($F = 4.63$, $df = 3$, $p = .021$) and between Group, Movement Type, and Presentation ($F = 4.61$, $df = 3$, $p = .021$). Thus, whereas the two groups did not differ overall, the performance of the LH group deteriorated more than that of the RH group as the movements became progressively more difficult. Separate MANOVAs between each patient group and the normal controls led to the same conclusion; that is, there was a significant interaction between Group and Movement Type when the controls were compared with group LH ($F = 17.97$, $df = 3$, $p < .001$), but not when they were compared with group RH.

A MANOVA carried out on all three groups to determine whether the presence of a context affected performance of the single movements (simple

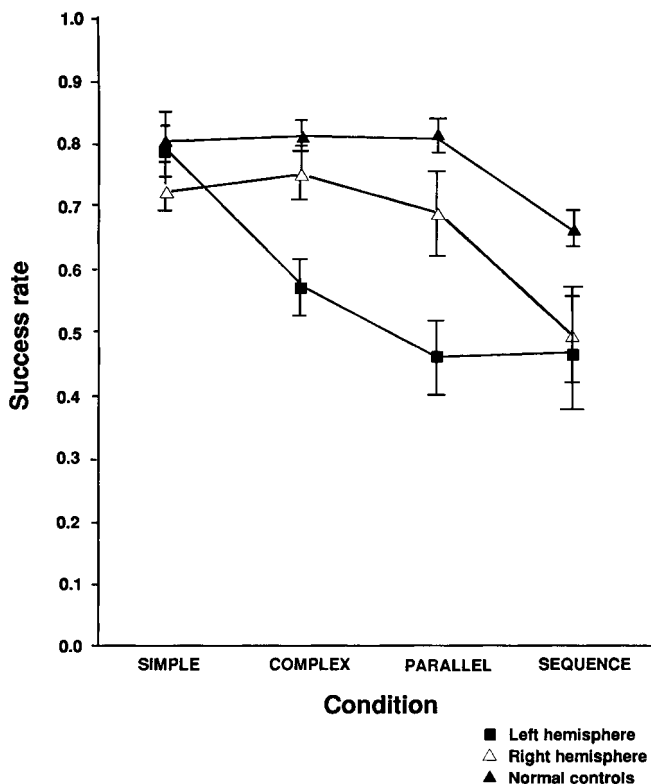


FIG. 6. Movements performed to command. Proportion correct in different conditions for patients with LH damage, patients with RH damage, and normal controls.

and complex) revealed a Group effect ($F = 17.13$, $df = 2$, $p < .001$). There were also interactions between Group and Complexity (simple versus complex) ($F = 6.37$, $df = 2$, $p = .01$); between Complexity and Context ($F = 21.15$, $df = 1$, $p < .001$); and between Group, Complexity, and Context ($F = 5.42$, $df = 2$, $p = .01$).

A MANOVA on errors for the repeated movements revealed only a main effect of Group ($F = 8.40$, $df = 2$, $p < .001$). For speed, there was also a significant main effect of Group ($F = 4.73$, $df = 2$, $p = .015$) as well as for Instruction, but there was no interaction.

DISCUSSION

Relationship between Oral and Verbal Dyspraxia

The affected family members performed more poorly than age-matched controls on the test of nonverbal oral movements. Thus, the affected family members make dyspraxic errors not just in speech (Hurst et al., 1990;

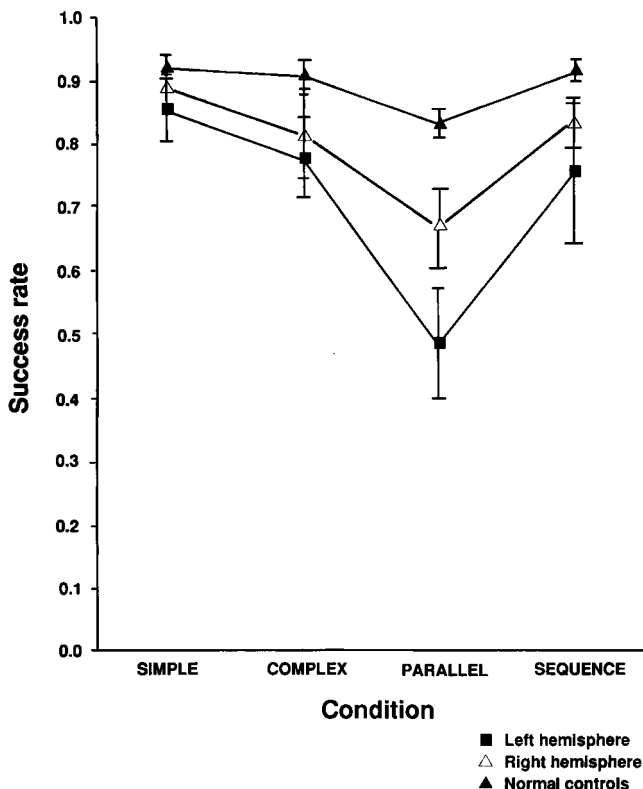


FIG. 7. Movements performed to imitation. Proportion correct in different conditions for patients with LH damage, patients with RH damage, and normal controls.

Vargha-Khadem et al., 1995) but in oral movements generally. The one condition in which they were not significantly impaired was in performing single oral movements involving only one group of muscles. On the single oral movements involving more than one group of muscles, however, as well as on the combinations of movements, both simultaneous and sequential, their performance was markedly impaired.

A similar pattern of performance was observed in the adult patients with left-hemisphere lesions and dysphasia. Like the affected family members, they were able to perform the simple movements normally, though in their case only to imitation, whereas they became increasingly impaired as the movements became progressively more difficult. The patients with right hemisphere damage also performed more poorly than the controls, perhaps as a result of residual orofacial hemiparesis. However, unlike the other impaired groups, their impairment did not increase with an increase in the difficulty of the movements.

As in the present study, Square-Storer et al. (1989) had found that oro-

motor performance of subjects with left-hemisphere damage gradually deteriorated as the number of movements being performed increased. Similarly, Blumstein (1990) had shown that patients with nonfluent dysphasia have a greater difficulty with simultaneous combinations than with simple movements of the articulators. On the other hand, Kimura and colleagues (Ma-teer & Kimura, 1977; Kimura, 1993) reported that such patients are impaired as well in the performance of single oral movements. However, in the latter experiments, the single movements included protruding the tongue, blowing, and whistling, all of which require the use of more than one set of muscles. In the current study, such movements would have been classified as complex movements, involving more than one group of muscles.

The implication of the present findings for the relationship between oral and verbal dyspraxia in the case of the affected family members is the same as one that can be drawn for patients with acquired dysphasia or, indeed, for other cases of developmental verbal dyspraxia. Stackhouse (1992) has described the earliest manifestation of verbal dyspraxia in children as delayed and disordered speech combined with an inability to perform nonverbal oral movements, particularly in sequences. The onset of the production of speech sounds does not normally occur until an infant reaches the stage where oromotor control is sufficiently developed (Whitaker et al., 1981). Thus, children who find it difficult to perform complex oral movements of all types should also find it difficult to learn and perform the complex movements involved in speech production. In short, the present findings suggest that a central aspect of the disorder in the affected members of the KE family is a difficulty in coordinating the oral musculature for anything but the simplest movements and that this difficulty precludes the fine oromotor control needed for articulate speech.

Relationship between Dyspraxia and Linguistic Impairments

It could be argued that the affected family members and patients with left-hemisphere lesions were impaired in performing movements to command because they had difficulty understanding the verbal instructions due to their verbal dyspraxia and dysphasia, respectively. However, both groups were also impaired in performing all but the single, simple movements even to imitation, and imitation aided neither group more than the controls in performing the complex and combined movements.

The present results thus lend further support to the view that the impairment in the affected members of the KE family is not selective to linguistics and, in particular, is restricted neither to the production of morphological markers nor to grammar more generally (Gopnik, 1990; Gopnik & Crago, 1991). Vargha-Khadem et al. (1995) have pointed to other nonlinguistic impairments in the affected family members, such as their poor repetition of nonwords. Also, besides the nonverbal oromotor impairments described

here, Alcock and colleagues (1994; Alcock 1995) found that they had difficulty in reproducing rhythms tapped by the experimenter, whether the temporal pattern involved a series of different movements or the same movement.

A question remains, however, as to what the relationship is, if any, between the dyspraxic and linguistic impairments in the KE family. One possibility is that the linguistic impairments are an indirect consequence of the same neuropathology that is the direct cause of the dyspraxia. Leonard (1989) has suggested that subjects who have difficulty producing phonologically accurate speech may economize their grammatical production in order to maximize comprehension, compensating for the difficulty they have in producing morphemes of low phonetic substance. Bates et al. (1991) likewise have suggested that patients with dysphasia use those aspects of their language that maximally enable communication, in preference to aspects that do not add substantially to the information being transmitted. The whole linguistic system in such cases can be seen to be under stress, with the possible consequence being a marked simplification and reduction of linguistic output.

Alternatively, the dyspraxic and linguistic impairments may result from the abnormal development of neighboring and perhaps related brain regions. Dronkers (1996) analyzed the lesions of 25 patients with a disorder in the motor planning of articulation and found that the area of lesion overlap lies in a portion of the insula just anterior to the central sulcus, not far from Broca's area in the frontal operculum. Recently, Vargha-Khadem et al. (1998) compared affected family members and normal controls in a PET activation study that involved repeating heard words. The results did not show any abnormality in the insula, but they did reveal abnormal activity in many other motor-related areas of the left hemisphere. Thus, compared to the activation in controls, the affected family members showed underactivity in the face area of motor cortex and the supplementary motor area (SMA) and overactivity in Broca's area and dorsally adjacent premotor cortex as well as in the head and tail of the caudate nucleus. In the same investigation, statistical parametric mapping of MRI scans disclosed significant differences between affected and unaffected family members in the amount of gray matter within Broca's area, left anterior insula, and left preSMA, among others, and bilaterally in the head of the caudate nucleus. Finally, a volumetric analysis of the head of caudate nucleus indicated that in the affected family members this structure is abnormally small bilaterally. The caudate nucleus is anatomically linked via the pallidum and ventral thalamus with the premotor cortex (for review see Passingham 1993). Ullmann et al. (1997) have proposed that disruption of circuits linking frontal cortex and basal ganglia may impair not only the learning of motor programs but also the learning of rules, including grammatical rules. Correct sequencing of the oral movements for the production of phonemes and morphemes, of phonemes and morphemes for the production of words, and of words for the production of sentences could all be impaired as a result of a deficit in motor rule learn-

ing. It is thus possible that the affected family members' impairments in speech and language result from the abnormal operation of parallel but related circuits.

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