

Progressive Dysfluency Associated with Right Hemisphere Disease

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A late onset progressive dysfluency following a right hemisphere stroke occurred in a 62-year-old male. Dysfluency was characterized by pronounced word and phrase reiterations, and sound and syllable reiterations to a lesser degree. Spontaneous speech was significantly more dysfluent than oral reading and repetition. Recitation and singing were minimally dysfluent. Reiterations were accompanied by reduced vocal loudness and increased rate of speech. Reiterations occurred in initial, medial, and final sentence position. Secondary symptoms such as facial grimacing were absent. In contrast to acquired cortical stuttering which is characterized by a preponderance of sound and syllable sentence initial repetitions, dysfluency was more closely characteristic of palilalia. The dysfluency occurred as a symptom of a diffuse nonspecific subcortical projection system defect related to massive infarction in the right middle cerebral artery distribution with associated atrophy.

INTRODUCTION

Acquired neurogenic dysfluency takes at least two major forms: "cortical stuttering" and "palilalia." Palilalia has been recognized for many years as a clinical entity (Souques, 1908, in Critchley, 1927). Cortical stuttering has recently gained such recognition. Palilalia is classically associated with bilateral neuropathology, either in the frontal lobe (Valenstein, 1975) or basal ganglia (Boller, Boller, Denes, Timberlake, Zieper, & Albert, 1973). Cortical stuttering in its transient form is typically associated with acute unilateral vascular disease; in its persistent form, with bilateral cortical pathology, either bilateral cerebral vascular accident or head trauma (Helm, Butler, & Canter, 1980; Quinn & Andrews, 1977; Rosenbek, Messert, Collins, & Wertz, 1978). Other entities associated with cortical stuttering include postencephalitic Parkinsons disease (Rosenbek et al., 1978) senile dementia (Quinn & Andrews, 1977), and dialysis dementia (Rosenbek, McNeil, Lemme, Prescott, & Alfrey, 1975). Persistent dys-

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fluency following right cerebral vascular accident in patients with intact language is rare (Rosenbek et al., 1978), and late onset progressive dysfluency following right hemisphere cerebral vascular accident has not been previously reported.

Few studies are available regarding the neuropathologic and clinical aspects of cortical stuttering (Helm, Butler, & Canter, 1980; Rosenbek et al., 1978). Boller and his associates (Boller, Albert, & Denes, 1975; Boller et al., 1973) discussed the neuropathologic features of palilalia; LaPointe and Horner (1981) described the locus and type of verbal reiteration; and Helm (1979) described the management of palilalic speech with a pacing board.

Our case presented a progressive dysfluency with its onset 2 years after a right hemisphere stroke. Partial recovery from visual-agnosic misnaming and visual dyslexia following treatment 1 year postonset has been reported (Horner, 1980). The purpose of this paper is to describe the clinical manifestation of this patient's dysfluency, which shares characteristics of both cortical stuttering and palilalia. The differential characteristics of these clinical syndromes, both in terms of motor speech and neuropathologic aspects are explored.

CASE HISTORY

Medical Background

A 62-year-old right-handed white male who experienced a sudden onset of complete left hemiparesis in July of 1978 was studied. Lethargy gradually progressed to a semicomatose state over the next few days and the patient was transferred to another hospital where angiography showed right internal carotid artery occlusion. Tentorial herniation was present secondary to cerebral edema. He was placed on a high dose of steroids and swelling resolved over several days. By the third week he was alert and responding but with profound left hemiplegia. The subsequent month he was transferred to a hospital near his home. He developed pneumonia secondary to aspiration which required tracheostomy. Improved pulmonary hygiene with care to avoid aspiration allowed for resolution of his pneumonia and the tracheostomy was closed. Physical examination revealed a complete left hemiplegia including the face and generalized hypesthesia to light touch over the left lower extremity. He was discharged to a nursing home where he continued physical therapy. Twelve months following cortical infarct little functional return had occurred on his left side. He was confined to a wheelchair and performed few activities of daily living. He fed himself only with prompting. At the family's request he was admitted to Duke University Hospital for evaluation of rehabilitation potential.

Past history was significant for hypertension, controlled on medical therapy. He had a history of mild congestive heart failure and was asymp-

tomatic on Lanoxin. The patient had been self-employed as a home builder and had a 12th grade education. Physical examination revealed a grade II/VI systolic ejection murmur heard over the left base with radiation to the left carotid, and bilateral undescended testes. Neurologic examination revealed a left homonymous hemianopsia, a left central facial paresis, left hyperreflexia, and left Babinski sign. There was marked spasticity on the left upper and lower extremity with minimal grip in the left hand and no functional contraction of the left shoulder girdle or thigh muscles. Strength on right side was normal. Sensation was intact. He was oriented and able to verbalize without difficulty, but a mild dysarthria and significant deficits in naming, reading, drawing, and copying were present. Apraxia was evident on dressing, topographical orientation, and constructive tasks. Hearing screening revealed responses to 25-db threshold in the right ear at 500 Hz and 1K and 2K Hz. In the left ear he responded to a 25-db tone at 1K Hz only; responses at other frequency levels were slightly elevated.

Speech/Language and Visual-Constructional Performance

At initial evaluation (1 year poststroke) he presented with normal auditory-verbal language, and depressed visual-constructional abilities. On the *Western Aphasia Battery* (Kertesz, 1979), he achieved an Aphasia Quotient of 94.8 out of 100.0, and a Cortical Quotient of 86.7 out of 100.0. Spontaneous speech was fluent and free of phonologic, semantic, or syntactic errors. Discourse was organized and appropriate. On a picture description task, left-side neglect caused him to omit items on the left, lowering his "content" score slightly. However, object naming, sentence completion, and responsive speech were normal. Comprehension of "yes-no" questions was unimpaired. Comprehension of spoken words representing 10 semantic categories was essentially normal except for errors of recognition of two finger names and two numbers. Repetition of words and sentences was unimpaired. On the *Token Test* (DeRenzi & Faglioni, 1978), he achieved 34 of 36 correct. On the *Boston Naming Test* (Kaplan, Goodglass, & Weintraub, 1978), responses were characterized by a preponderance of visual-associative errors, for example, "snake" for pretzel, "parallel bars used in therapy" for stilts, "seats in a stadium" for pyramid, "necklace" for noose, and "hypodermic needle" for tripod. When the *first* response was used as the basis for scoring, 25 out of 85 items were correct. Responses were minimally influenced by the familiarity of the word.

Reading and writing subtests of the *Western Aphasia Battery* were administered. He was able to read most single letters, though visual confusion occurred on occasion, e.g., f/t, a/d, e/u. Similar errors were made on single-word reading tasks, e.g., nap/map, hip/ship, groom/broom. Word, sentence, and paragraph reading were influenced by visual perceptual

difficulties and neglect of the left side of printed stimuli. In contrast, he recognized orally spelled words and was able to spell words aloud. He achieved 74.0 out of 100.0 on the reading subtest of the *Western Aphasia Battery*. On the *Reading Comprehension Battery for Aphasia* (LaPointe & Horner, 1979), initially he achieved a score of 20 (100 possible points). His reading difficulty was consistent with a diagnosis of "neglect dyslexia" associated with right hemisphere damage as described by Kinsbourne and Warrington (1962).

He was able to write single words and sentences. Letter size and legibility of cursive writing were normal, but writing was disoriented on the page. Visually related substitutions (e.g., u/w, t/i) and letter omissions occurred (e.g., refrig...rator, physic...an). Sentences were grammatically complete. He scored 73.5 (out of 100.0) on the writing subtest of the *Western Aphasia Battery*.

On a Kohs block design task, he was able to reconstruct only one of three block designs. Drawing and copying were characterized by neglect of details and lack of perspective (see Fig. 1). On Raven's *Coloured Progressive Matrices* (Raven, 1962), 18 of 36 were correct. This placed him at the 35th percentile as compared to right brain-damaged individuals, and below the 10th percentile as compared to normal adults. On the calculation subtest, 9 of 12 problems were correct, reflecting inconsistent recognition of arithmetic signs.

A 6-week course of daily therapy was rendered for remediation of visual-agnosic misnamings and visual dyslexic deficits (Horner, 1980). After treatment, reading comprehension had improved from 20 to 61 (100

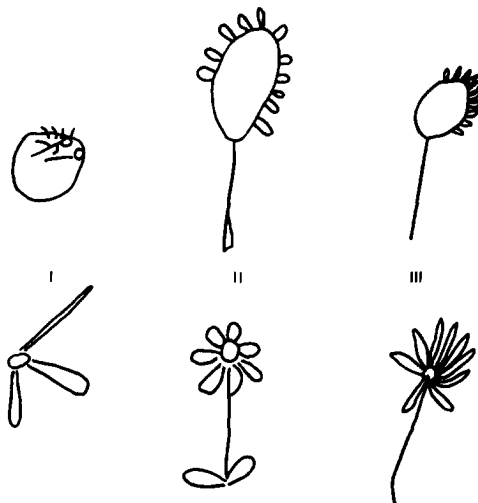


FIG. 1. Drawing from memory (above) and copying (below) (I) before treatment and at (II) 4 months and (III) 18 months after treatment shown from left to right.

possible) on the *Reading Comprehension Battery for Aphasia*, and from 25 to 56 (85 possible) on the *Boston Naming Test*. In addition to speech therapy, the patient underwent occupational and physical therapy and nursing care education. At the time of discharge, he was virtually independent in his activities of daily living but required periodic coaching. Due to a hyperreflexic bladder with bladder outlet obstruction secondary to prostatic hypertrophy he was placed on Banthine therapy. Otherwise his medication was unchanged and he was followed as an outpatient.

Reevaluation

At a clinic visit 2½ years postonset of stroke he complained, "I just talk too fast sometimes." His wife stated that he had begun to "stutter" 3–6 months previously, and it was getting worse. When asked if he repeated himself, he stated, "They can't understand me and I have to repeat it for them." His wife further reported that the patient seemed "depressed . . . unconcerned."

He presented decreased range of articulatory motion with mild speech sound imprecision, a harsh voice quality, and reiteration of sounds, words, and phrases. Secondary symptoms such as facial grimacing and eye blinking were absent. Speech was generally amelodic (reduced stress variation), dysprosodic (atypical phrasing), and rapid, particularly in periods of dysfluency. Overall intelligibility was impaired to a moderate degree. An instrumental evaluation of respiratory, laryngeal, and velopharyngeal function was conducted. The patient performed as follows in comparison to normal: (1) slightly reduced vital capacity, (2) normal subglottic air pressure, (3) moderately elevated mean flow rate indicative of abnormal vocal fold approximation, (4) slightly elevated pitch accompanied by a restricted pitch range, (5) normal velopharyngeal function.

A variety of tasks were presented to elicit speech. He demonstrated dysfluency characterized by sound, syllable, word, and phrase reiteration. He was able to recite rhymes (e.g., Jack and Jill . . . there was an old woman . . . etc.) with minimal instances of reiteration, and was able to sing "Happy Birthday" with fair melody and no reiteration. On reproduction of tapped rhythms, ranging in length from four to six beats, he was able to reproduce the short rhythms, with omissions and dysrhythmia on longer tapped sequences. Spontaneous speech was moderately impaired, while repetition and oral reading were mildly impaired. Though initial sound and syllable repetitions occurred, these were less prominent than word and phrase reiterations. Reiterations occurred in initial, medial, and final sentence position. For example, when asked to recall a memorable experience, the patient said: "Having having a stroke, h-having a stroke, haven't been able to walk, haven't been able to walk, I get get frustrated, b-b-b-been active a-a-all-all, all my life. [Do you remember your stay on the rehabilitation unit?] mmmm . . . stayed over there about 2 months,

I came, to s-see-see you, about everyde-everyde-everyday, t-t-t-took physical therapy, and, speech therapy and, and that's about it, wu-w-w-wor-worked on, re-reading from, left to right, go all the way to the left, I-I had problems go-go-going to my left. [Do you repeat yourself?] What, repeatin' repeatin' myself? I-I-I just, can't seem to get the words out sometimes. They can't understand me, they can't understand me, I have to repe-repeat it, repeat it for 'em.'

On tasks of picture naming, visual perceptual abilities and word retrieval were stable when compared to previous post-treatment status. A circle, square, and clock were drawn symmetrically; a cube and a house lacked perspective and detail; and a flower and a man were marked by omission of details on the left. Performance was slightly improved when copying these same figures, though the cube, house, and man remained asymmetrical and incomplete. Figure 1 shows that drawing from memory had regressed in comparison to his earlier post-treatment status, while copying ability was stable.

At the time of evaluation (2½ years poststroke onset and 1½ years after initial treatment), the patient's speech continued to be free of semantic and phonemic paraphasias, and he responded appropriately to social speech and task instructions. He was oriented, conversant, and appropriate, except for some evidence of denial and a tendency to respond concretely on a task of defining words. As on previous test occasions, writing was legible and grammatically complete with occasional visually related misspellings. Writing was somewhat micrographic in contrast to previous samples.

His motor examination remained unchanged. CT scan demonstrated an extensive nonenhancing low density in the entire distribution of the right middle cerebral artery sparing the right anterior cerebral and posterior cerebral artery distributions. Asymmetrical enlargement of the right lateral ventricle indicated atrophy but no midline shift or abnormal enhancement was present (see Fig. 2). Electroencephalogram showed projected rhythmic frontal delta activity with polymorphic irregular theta in the anterior head regions and mild background slowing of posterior activity. He was begun on Phenytoin 300 mg every evening at another hospital for an unknown reason in the summer prior to reevaluation. No symptoms or signs of toxicity were evident and blood level of Dilantin was 6 µg/ml on two occasions during our evaluation.

ANALYSIS OF DYSFLUENCY

The purpose of the dysfluency analysis was to ascertain the severity and locus of reiteration. Eight types of tasks comprised the speech sample: (1) conversational speech, (2) defining words, (3) formulating sentences, (4) describing pictures, (5) reading sentences, (6) repeating sentences, (7) reciting familiar rhymes, and (8) counting. The tasks were selected

from the *Minnesota Test for Differential Diagnosis of Aphasia* (Schuell, 1965) and the *Boston Diagnostic Aphasia Exam* (Goodglass & Kaplan, 1972). Six types of reiteration were identified and tabulated: (1) sound (e.g., b-b-b-boy), (2) syllable repetition or prolongation (e.g., his-history; re-return), (3) part-word (e.g., retur-return, suppo-supposedly), (4) word (with mono-, bi-, and polysyllabic words counted separately), (5) phrase (e.g., just the name of [the name of] the sign), and (6) sentence (e.g., they can't understand me [they can't understand me]).

The first analysis focused on *words* only. Of 764 words in the sample, 197 words were judged to be reiterative; thus, 25.8% of our patient's speech was dysfluent. Subsequent analyses considered all six types of reiteration. The distribution of dysfluency across tasks and the distribution of dysfluency by reiteration type were considered. In these analyses, any *string of uninterrupted reiterations of a single type* was considered an "episode" of dysfluency. In the following example, the target (intended) utterance of two words was realized as a 12-word utterance. The target was: "(I) can't walk." The actual utterance and corresponding analysis was: "can't can't can't can't walk" (first episode, 3-word reiterations) "can't can't can't walk" (second episode, 2-word reiterations) "can't can't walk" (third episode, 1-word reiteration). The phrase "can't walk" is uttered three times (reiterated twice), and in the context of this target phrase, the word "can't" is uttered nine times (reiterated a total of six times) in three different episodes. Thus, this utterance was described as a 5-episode 2-type dysfluent cluster.

Table 1 shows the distribution of episodes and the distribution of reiterations (all types) across the eight speech tasks. In the 764-word sample, a total of 146 dysfluent episodes and 238 total reiterations (all types) occurred. Dysfluent episodes during conversational speech and the task of defining words (32.9 and 31.5%, respectively) were more frequent than on the remaining tasks. The number of reiterations were also most numerous for these two tasks (29.0 and 34.0%, respectively). Formulating sentences and describing pictures were moderately dysfluent, with oral reading, repeating, reciting, and counting being least affected by reiterations.

Table 2 shows the distribution of reiteration episodes by reiteration type (all tasks combined). Episodes of word reiteration were most frequent (39.7% of reiteration episodes); sound reiterations and phrase reiterations were approximately equal (20.6 and 23.9%, respectively). To clarify these results, two major types of reiteration were differentiated. The first major type (comprised of initial-word sound, syllable, and part-word reiterations) accounted for 35.6% of the reiteration episodes, while the second major type (word, phrase, and sentence reiterations) accounted for 64.4% of the reiteration episodes in the speech sample.

The number of reiterations per episode is another index of the severity



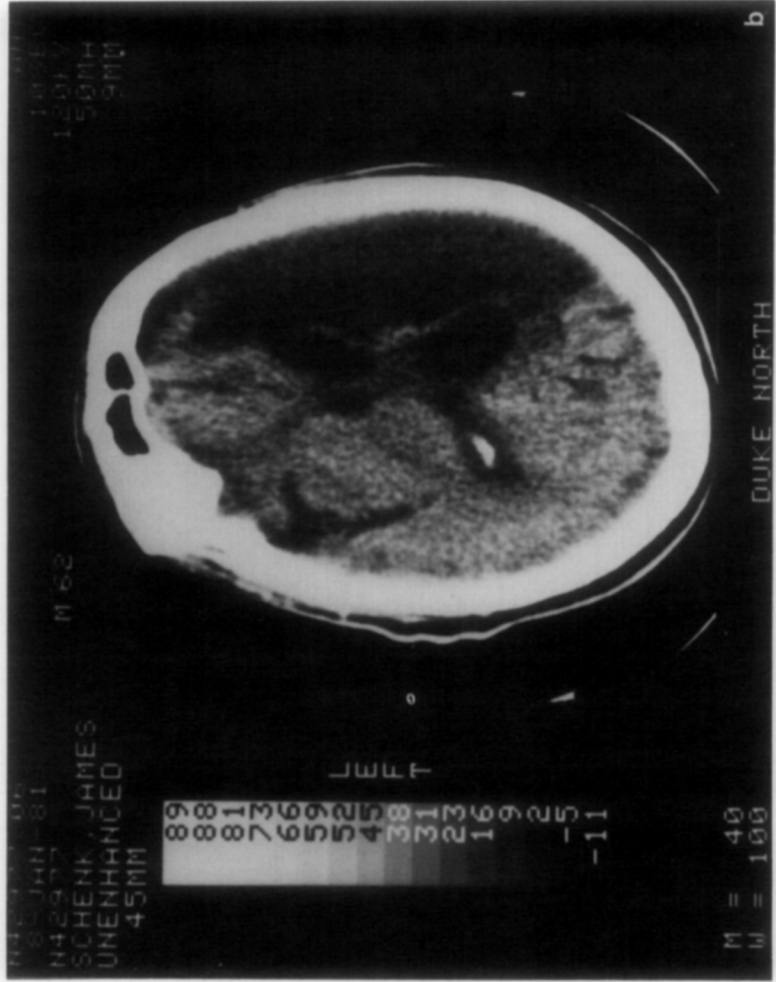


FIG. 2. Unenhanced (a) and enhanced (b) CT scans. There is extensive nonenhancing low density in the entire distribution of the right middle cerebral artery. Associated with this is asymmetrical enlargement of the right lateral ventricle without midline shift.

TABLE 1
DISTRIBUTION OF DYSFLUENCY BY TASK TYPE

Task	No. of words	Distribution of reiteration episodes		Distribution of No. of reiterations	
		No. of episodes	Percentage of total episodes (146)	No. of reiterations	Percentage of total reiterations (238)
Conversational speech	149	47	32.9	69	29.0
Defining words	159	46	31.5	81	34.0
Formulating sentences	63	14	9.6	25	10.5
Describing pictures	127	21	14.4	38	16.0
Reading sentences	36	7	4.1	7	2.9
Repeating sentences	97	9	6.2	16	6.7
Reciting	90	1	0.7	1	0.4
Counting	50	1	0.7	1	0.4

TABLE 2
DISTRIBUTION OF REITERATION EPISODE BY REITERATION TYPE

Reiteration type	No. of episodes of reiteration	Percentage of grand total reiteration episodes (146)
Sound ^a	30	20.55
Syllable ^a	3	2.05
Part word ^a	19	13.01
Word ^b	58	39.73
(Monosyllabic)	(39)	(26.70)
(Bisyllabic)	(14)	(9.60)
(Polysyllabic)	(5)	(3.40)
Phrase ^b	35	23.97
Sentence	1	.68

^a Initial-word position.

^b All positions in sentence.

of dysfluency. In this speech sample, the maximum number of reiterations in an episode was no greater than six. This was partially biased by the type of task. On reading, reciting, and counting tasks, no more than one reiteration occurred in any episode; in the remaining tasks, dysfluencies ranged from one to six reiterations per episode. The average number of reiterations, all tasks combined, was 1.6 reiterations per episode. Again, reading, reciting, and counting tasks yielded the lowest average, and the remaining tasks were not significantly different.

The preceding analyses considered several indices of severity: number of words reiterated, number and distribution of episodes of reiteration, number and distribution of reiterations, and number of reiterations per episode. The locus of reiteration (reiteration type) was also described. None of these measures was completely satisfactory in reflecting the quality and severity of the patient's dysfluent speech.

"Clustering" (co-occurrence) of several types of reiteration in an utterance was a striking feature of the patient's dysfluency pattern. A palilalic patient described by LaPointe and Horner (1981) is similar to our patient in this regard. For this analysis, counting and recitation tasks were excluded because of the minimal occurrences of dysfluency. The remaining sample consisted of 82 utterances. Nineteen (23.3%) of these utterances were free of reiteration; 23 (28.0%) were marked by a single type of reiteration; and 40 utterances (48.4%) were characterized by clusters of different reiteration types. As in preceding analyses, conversational speech and defining words were most likely to be marked by clusters of reiteration types. The impression of "severity" increased as the *number of successive episodes* and the *variety of reiteration types* increased. Examples of reiteration clusters are as follows, with inspiratory breaks designated by comma's: (1) "I-la-lady washing, washing dishes and b-b-b-bo, boy in the cookie jar, boy in the cookie jar." (2) ('different') "it means it means its, o-opposite from, from from, opposite opposite from, from from from from, from from, it means its opposite." (3) "a bird a bird a a a bird bird bird bird, a robin robin is a bird b-b-b-b-b-bird." (4) "retur-return something that you, that you bought, return return something that you bought, return, return retur, return re, re-return."

These samples illustrate reiterative speech in the context of abnormal prosody. Utterances were characterized by brief inspirations not only at linguistic boundaries but also within words and phrases.

DISCUSSION

There are three reports of cortical stuttering following right hemisphere damage (Rosenbek et al., 1978). Patient II reported by Rosenbek et al. (1978) differs from our patient in several critical respects and by contrast highlights why we prefer a diagnosis of palilalia in our patient.

Rosenbek's case was a 53-year-old right-handed male who presented dysfluency 96 hr after a right parietal cerebrovascular accident. Fifty-five percent of his speech was dysfluent. An analysis of locus of reiteration showed that sound and syllable repetitions were the most frequent, monosyllabic word repetitions were minimal, and polysyllabic word and phrase repetitions did not occur. There was no difference between spontaneous and imitative speech performance. After six sessions of speech therapy, he regained fluent speech and remained fluent at 1-month follow up.

Our patient, in contrast, presented dysfluency 2 years post-right hemisphere damage. He was moderately severe, with 25.8% overall dysfluency. The locus of reiteration in our patient was distributed among sound, syllable, word, and phrase reiterations. Whereas there was no difference between spontaneous and imitative speech in Rosenbek's case, our patient showed a definite contrast between spontaneous speech and other tasks. Rosenbek's case suffered a circumscribed right parietal lesion; our case suffered massive right hemisphere damage with atrophy, and a diffuse subcortical deficit as documented by CT scan. Rosenbek's case presented a profile consistent with a diagnosis of cortical stuttering. Although the speech of our patient shared characteristics of both cortical stuttering and palilalia, it was predominantly palilalic. Differential characteristics of cortical stuttering and palilalia are presented in Table 3.

The fact that our patient was fluent until 2 years postonset suggests

TABLE 3
DIFFERENTIAL CHARACTERISTICS: CORTICAL STUTTERING VERSUS PALILALIA

	Cortical stuttering	Palilalia
Locus of lesion	Cortical; unilateral (usually dominant hemisphere) or bilateral	Basal ganglia; bilateral Frontal cortex; bilateral
Etiology and duration	Cerebrovascular accident; transient or persistent Chronic conditions; progressive Postencephalitic Parkinson's Senile dementia Dialysis dementia Trauma with seizures	Idiopathic cerebral calcinosis; progressive
General feature	Initiation difficulty	Perseverative, self-echolalic
Locus of reiteration: Type of speech task	Degree of reiteration not necessarily in direct relation to propositionality of speech; self-formulated speech may be easier than automatic speech (counting, reciting rhymes, etc.)	Reiteration most severe for free, open-ended speech; minimal on repetition, reading aloud, rhymes and songs

TABLE 3 (continued)

	Cortical stuttering	Palilalia
Locus of reiteration: Position in utterance	Beginning of utterance	Beginning, middle, and end of utterance
Locus of reiteration: Syllables	Repetition of syllables is a common symptom	Repetition of syllables is the least common symptom
Locus of reiteration: Words and phrases	Repetition of sounds and syllables more frequent than repetition of words and phrases	Repetition of words and phrases predominates; usually the latter part of the phrase
Locus of reiteration: Grammatical form	No particular relationship between dysfluency and grammatical function of words	Greater occurrence of reiteration on contentives than functors
Loudness	Usually normal	Decrescendo at end of phrases
Rate of speech	Increased rate during reiteration episode	Increased rate during reiteration episodes and increasing rate on successive reiterations
Melody (stress and pitch)	Fluent speech is melodic; "blurring" may occur on initial sound-syllable repetitions	Speech is amelodic; reiteration in ongoing speech is accompanied by decreasing loudness and increasing rate of speech
Adaptation effect	No observable adaptation effect	No significant adaptation effect
Reaction to dysfluency	Speaker may be annoyed but not necessarily anxious about his stuttering; secondary symptomatology (facial grimacing, eye blinking) unlikely	Speaker may be annoyed but not necessarily anxious about his dysfluency; secondary symptomatology absent

Note. Boller et al., 1975, 1973; Brown, 1972; Helm et al., 1980; LaPointe & Horner, 1981; Rosenbek et al., 1975, 1978; Valenstein, 1975.

that his dysfluency was primarily a result of neural degeneration of the subcortical projection system in response to right hemisphere cortical damage. The late appearance of cortical stuttering symptomatology co-incidentally with palilalia suggests that the former was a secondary manifestation of the diffuse subcortical deficit.

The neural mechanism underlying fluent speech is highly complex and not yet fully understood. In our patient, periods of fluent speech were melodic (normal stress variation and normal rate) and periods of dysfluent speech were amelodic (reduced stress variation and increased rate). These

amelodic features, in association with reduced range of articulatory motion are characteristic of hypokinetic dysarthria (Darley, Aronson, & Brown, 1975). However, during periods of severe dysfluency (those described above as clusters of reiteration types), speech was both *amelodic* and *dysprosodic*. Here the term dysprosody refers to the irregular inspiratory pattern resulting in breaks in the flow of speech within words and within phrases, causing concomitant aberrations in stress pattern and phoneme duration. Our patient's dysprosody was reminiscent but not completely consistent with the dysprosody of ataxic dysarthria which is associated with excess and equal stress, prolonged phonemes, prolonged intervals, and slow rate overall (Kent & Netsell, 1975).

Dysprosody was an integral feature of the articulatory dysfluency in our patient; dysprosody occurred in proportion to the degree of verbal reiteration. Automatic speech performance (singing, reciting, counting) was least dysfluent and spontaneous speech (conversation, picture description, etc.) was most dysfluent. This might suggest that linguistic propositionality was the critical factor effecting dysfluency and dysprosody. However, our patient had essentially normal language, and a dysrhythmia was also observed during nonlinguistic tapping of lengthy and complex sequences. It appears that the neural regulation of fluent speech—including articulatory and phonatory-respiratory aspects—was stressed as the length and complexity of the motor act increased. Kent and Rosenbek (1981) described the neural pathways concerned with the prosodic regulation of speech. They suggested that this mechanism includes both ipsilateral and contralateral cortical and subcortical (thalamic and cerebellar) pathways, and the right hemisphere appears to have a privileged role in the control of prosodic speech. Why some cortical lesions result in cortical stuttering and others do not, or why some subcortical lesions result in palilalia and others do not, remains unclear.

Further studies of acute and late onset dysfluency—whether transient, persistent, or progressive, following right and left cortical or subcortical lesions—are needed. Case studies describing the severity, locus, and type of reiteration in the context of specified neuropathologic data should lend further insight into the neural mechanisms underlying fluently articulated speech.

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