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Speech Production, Syntax Comprehension, and Cognitive Deficits in Parkinson's Disease

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Speech samples were obtained that were analyzed for voice onset time (VOT) for 40 nondemented English speaking subjects, 20 with mild and 20 with moderate Parkinson's disease. Syntax comprehension and cognitive tests were administered to these subjects in the same test sessions. VOT disruptions for stop consonants in syllable initial position, similar to those noted for Broca's aphasia, occurred for nine subjects. Longer response times and errors in the comprehension of syntax as measured by the Rhode Island Test of Sentence Comprehension (RITLS) also occurred for these subjects. Anovas indicate that the VOT overlap subjects had significantly higher syntax error rates and longer response times on the RITLS than the VOT nonoverlap subjects—$F(1, 70) = 12.38$, $p < 0.0008$; $F(1, 70) = 7.70$, $p < 0.007$, respectively. The correlation between the number of VOT timing errors and the number of syntax errors was significant. ($r = 0.6473$, $p < 0.01$). VOT overlap subjects also had significantly higher error rates in cognitive tasks involving abstraction and the ability to maintain a mental set. Prefrontal cortex, acting through subcortical basal ganglia pathways, is a component of the neural substrate that regulates human speech production, syntactic ability, and certain aspects of cognition. The deterioration of these subcortical pathways may explain similar phenomena in Broca's aphasia. Results are discussed in relation to "modular" theories.

INTRODUCTION

Human linguistic ability has traditionally been associated with specific regions of the cortex on the basis of clinicopathologic studies of aphasia. Indeed, many linguists continue to claim that human syntactic ability (which they equate with human language) derives from a neocortical "module" which is "encapsulated," i.e., functionally and morphologically

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distinct from other modules that govern other aspects of human behavior (Fodor, 1983, Chomsky, 1986). No homologue to the hypothetical syntax module supposedly exists in any other species. According to one theory (Bickerton, 1990), the cortical syntax module evolved by means of a single genetic change that dramatically modified the human neocortex, creating cortical maps encoding grammatical relations and a "syntactic" wiring diagram.

However, this neurophysiological model is not consistent with many early studies of aphasia-producing lesions which pointed to subcortical involvement in language (Marie, 1926). A number of recent studies show that permanent, "big," Broca's aphasia is the consequence of extensive subcortical damage (Stuss & Benson, 1986). Indeed, subcortical damage that disrupts the connections from Broca's area, leaving it intact, can result in Broca-like speech production deficits (Naeser, Alexander, Helms-Estabrooks, Levine, Laughlin, & Geschwind, 1982; Benson & Geschwind, 1972, pp. 206–207; Stuss & Benson, 1986, p. 161; Alexander, Naeser, & Palumbo, 1987; Metter, Kempler, Jackson, Hanson, Mazziotta, & Phelps, 1989). Metter, Riege, Hanson, Phelps, and Kuhl (1984) found that metabolic activity in the caudate nucleus, which forms part of a pathway to the prefrontal cortex (Parent, 1986), correlated with mean writing and reading scores of the Boston Diagnostic Aphasia Examination (BDAE) and with the speaking and comprehension factor scores of the Porch Index of Communicative Ability (PICA). Moreover, Metter and his associates (1987) note hypometabolism in prefrontal cortex with Broca's aphasia and conclude that the behavioral deficits of Broca's aphasia—general "difficulty in motor sequencing and executing motor speech tasks," as well as "language comprehension abnormalities"—derive from damage to neural pathways to prefrontal cortex. However, lesions are generally not so localized nor is the resolution of present imaging techniques adequate to determine whether damage limited to subcortical basal ganglia pathways to prefrontal cortex can cause speech, language, and cognitive disturbances. Since Parkinson's disease (PD), a neurodegenerative disease is primarily limited to subcortical basal ganglia structures, it can serve as an "experiment-in-nature" to resolve the question of whether the linguistic deficits associated with Broca's aphasia can derive from damage to subcortical pathways to prefrontal cortex.[1]

[1] Although cortical pathology also occurs in patients with PD these cortical changes do not appear to be a consequence of PD since similar changes occur in age-matched controls (Jellinger, 1990). Cortical changes are uncommon in PD unless Alzheimer's occurs as well (Jellinger, 1990), or when Lewy bodies occur in widespread fashion throughout the cortex. In this case the diagnosis is distinct being "diffuse Lewy body disease," a rare disorder, rather than PD (Jellinger, 1990; Xuereb, Tomlinson, Irving, Perry, Blessed, & Perm, 1990).

It is important to note that the hypothesis that we are testing is *not* that prefrontal cortex is the brain's "language organ." The hypothesis that we shall test, and the data that we shall discuss, point to a model in which specialized neural structures operate in different behavioral domains through different neural pathways (Mesulam, 1985). In the case of speech production and syntax, we will be testing the claim that basal ganglia "circuits" between Broca's area and prefrontal cortex regulate the precise motor sequencing necessary for speech, and the application of syntactic "rules" (Lieberman, 1991). The data that we will discuss also address another issue, whether the brain bases of linguistic ability are modules distinct from cognition. Since prefrontal cortex is implicated in cognitive functions involving the abstraction of concepts (Stuss & Benson, 1986), we might expect to find cognitive as well as linguistic deficits occurring in our subjects. This would not be surprising since previous studies have demonstrated cognitive deficits in Parkinson's disease (Cummings & Benson, 1984; Flowers & Robertson, 1985) similar to ones associated with frontal lobe damage (Benson & Geschwind, 1972).

It is also important to note that we do not expect to find that all PD patients will have linguistic deficits. Although Parkinson's disease can potentially affect many of the cortical pathways that involve the basal ganglia (Parent, 1986), the specific circuits involving Broca's region and prefrontal cortex may not be affected, particularly in the early stages of the disease process. We, therefore, will make use of one of the attested speech motor deficits of Broca's aphasia as a "probe" that may indicate whether PD has affected pathways involving Broca's area in a particular subject.

PREVIOUS STUDIES

Speech, language, and cognitive deficits previously have been noted in PD, although the results of different studies conflict. Several studies have described patients' difficulty in performing tasks of lexical and sentence disambiguation, confrontation naming, generative naming, and syntactic judgments (Bayles & Boone, 1982; Bayles & Tomoeda, 1983; Bayles, 1984; Tweedy, Langer, & McDowell, 1982). In contrast, other studies show no consistent difference in vocabulary; PD patients in some situations use fewer words than normal control subjects, but use more words in other circumstances (Pirozzolo, Hansch, Mortimer, Webster, & Kuskowski, 1982). Deficits reflecting syntax appear to be more consistent; differences in the syntactic complexity of the spontaneous speech of PD patients as well as increases in the number and duration of the short

These patients are often demented. We excluded demented patients from our PD subject population.

"hesitation" pauses that occur in the flow of speech were noted by Illes, Metter, Hanson, and Iritani (1988) and Illes (1989). Deficits in the comprehension of syntax coupled with some cognitive loss were noted for moderate PD (Hoehn & Yahr (1967), stage III) subjects by Lieberman et al. (1990). Deficits in the comprehension of syntactically complex sentences were noted by Grossman et al. (1991) for 73% of a sample of 22 PD patients (Hoehn & Yahr stages I and II). Grossman and his associates also found that 63% of these patients had praxis deficits in performing tasks from the BDAE. Independent neurophysiologic data indicate that these deficits, as is the case for Broca's aphasia, may result from damage to subcortical basal ganglia pathways to frontal cortex (Albert, Feldman, & Willis, 1974); positron emission tomography (PET) studies which show prefrontal hyopometabolism in PD demonstrate that BG pathways to prefrontal cortex have been degraded (Metter et al., 1984, 1987).

The speech production measure that we will use as a "probe"—that is, a measure that identifies PD subjects exhibiting the "difficulty in motor sequencing" noted for Broca's aphasia—is voice onset time (VOT). VOT is the time that elapses between the onset of phonation and the burst that occurs on the release of a stop consonant (Lisker & Abramson, 1964). "Voiced" sounds such as [b], [d], and [g] are produced with a short VOT, "unvoiced" [p], [t], and [k] with long VOTs. Acoustic analysis reveals that Broca's aphasics merge VOT for voiced and unvoiced stop consonants (Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980). Moreover, recent data on Broca's aphasia show that these VOT overlap deficits occurred in subjects who sustained damage to both Broca's region and subcortical pathways; CT scan data for these subjects show damage to subcortical areas including putamen, caudate nucleus, and the internal capsule (Baum, Blumstein, Naeser, & Palumbo, 1990).

PROCEDURES

Subjects. Forty out-patients with idiopathic Parkinson's disease without evidence of aphasia were tested at Roger Williams Hospital in Providence, Rhode Island. Twenty were evaluated as having stage III PD according to the Hoehn–Yahr scale (Hoehn & Yahr, 1967); these patients were labeled "moderate." The other 20 were evaluated as having stage I–II PD and were labeled "mild." Subjects in the moderate population ranged in age from 56 to 81 years, with a mean age of 69.1 years. Subjects in the mild population ranged in age from 45 to 72 years, with a mean age of 61.0 years. All subjects were free from dementia.[1] Neither the neurologist (JF), who spent significant amounts of time with them, or their families had noted evidence of cognitive decline. All subjects except 6 moderates and 2 milds were native monolingual speakers of English. The patients for whom this information was available (some were enrolled in a NINCDS protocol that limited access to this information) were either unmedicated or on stable programs of treatment—none showed "on–off" patterns of fluctuating short-term PD symptoms. Tables 1A and 1B show the case profiles for all subjects including the duration of their PD (from time of initial diagnosis), level of education, and the type of medication being taken at the time of testing.

TABLE 1A
CASE PROFILES OF SUBJECTS IN THE MODERATE POPULATION

Subject	Age	Sex	Level of education	First language	Age acquired English	Duration of disease (years)	Medication
1	72	M	12th grade	Armenian	4–5	10	SNMT,BCR
2	79	M	B.A.	English		16	BCR
3	74	F	10th grade	French	6–7	5	SNMT
4	68	M	12th grade	English		14	SNMT
5	64	M	9th grade	English		<1	None
6	67	F	Some college	French	4	10	SNMT,AMTD
7	58	M	9th grade	French	7	4	SNMT
8	81	M	B.A.	English		3	ART
9	75	M	12th grade	English		10	SNMT,AMTD
10	74	M	B.A.	English		Unknown	Unknown
11	73	M	B.A.	English		20	SNMT,BCR,AMTD
12	57	M	M.A.	English		12	SNMT,BCR
13	62	M	12th grade	English		12	SNMT,AMTD
14	67	M	9th grade	English		2	BCR
15	60	M	Ph.D.	English		3	SNMT
16	77	M	8th grade	Yiddish	12	13	SNMT
17	56	F	Some college	English		Unknown	Unknown
18	78	M	11th grade	English		7	SNMT
19	69	M	B.A.	English		7	SNMT,ART
20	71	M	8th grade	Polish	23	13	SNMT,BRC

Note. AMTD, Amantadine; ART, Artane; BCR, Bromocriptine; SNMT, Sinemet.

An additional sample of 10 bilingual subjects was tested using the Rhode Island Test of Language Structure—RITLS (Engen & Engen, 1983). These subjects were all native speakers of Spanish who had acquired English after age 7 years. Their ages ranged from 19 to 31 years, mean age 21 years. The ages at which they had started to acquire English ranged from 7 to 16 years, mean age for start of English 10.5 years. These subjects were all undergraduates enrolled at Brown University.

Psychological tests. The tests that were administered were taken from a battery of tests that are at present being used to assess PD patients in a currently funded NINCDS protocol, DATATOP (Parkinson's study group, 1989). Five tests were given at the start of each session: The *Selective reminding* test assesses short-term memory, i.e., the ability to encode and retrieve new information. The *Odd man out* assesses the ability to learn new problem-solving modes and maintain a cognitive set, then shift sets. The *New dot* is a test of spatial memory without a motoric component. Visual perceptual impairments would be reflected in the scores on this test. *Digit span* has two components. The digits *forward* tests attention to a relatively effortless task while digits *backwards* requires additional concentration. The *Verbal fluency* test is a measure of expressive language. It also measures the individual capacity to initiate a behavioral response in a structured manner.

Syntax test. The RITLS (Engen & Engen, 1983) assesses the extent to which subjects are able to use syntactic properties of sentences, such as word order, markers of the relationships between clauses in complex sentences, and markers of noncanonical order, in the understanding of sentences. Simple sentences like, *The man is old* and *The cat chased the dog*

TABLE 1B
CASE PROFILES OF SUBJECTS IN THE MILD POPULATION

Subject	Age	Sex	Level of education	First language	Age acquired English	Duration of disease (years)	Medication
1	72	M	8th grade	English		1	None
2	71	M	12th grade	English		1	None
3	60	M	M.A.	English		8	ART,SNMT
4	61	F	12th grade	English		Unknown	Unknown
5	67	M	12th grade	English		2	DATATOP
6	51	M	M.A.	English		Unknown	Unknown
7	62	F	11th grade	English		3	SNMT
8	66	M	B.A.	English		2	DATATOP
9	64	F	12th grade	Italian	5	Unknown	Unknown
10	64	F	12th grade	English		3	SNMT
11	62	F	Some college	English		2	SNMT
12	59	M	Some college	English		<1	DATATOP
13	62	F	B.A.	English		8	SNMT,AMTD
14	51	F	Ph.D.	Latvian	13	<1	None
15	59	M	12th grade	English		2	SNMT,AMTD
16	45	M	12th grade	English		3	SNMT,BCR
17	68	F	12th grade	English		2	DATATOP
18	54	M	Some college	English		2	DATATOP
19	64	M	12th grade	English		2	None
20	58	M	Ph.D.	English		<1	DATATOP

Note. AMTD, Amantadine; ART, Artane; BCR, Bromocriptine; SNMT, Sinemet; DATATOP, unknown drug protocol as part of study on Deprenyl.

are presented as well as more complex ones like *The boy is small but strong* and *The dog was chased by the cat.* The test presents a representative sample of the syntactic structures of English. Because syntax is the focus of the test, vocabulary and morphology are tightly controlled. A small number of words judged to be understandable by young children are used repeatedly in the test sentences. This makes it possible to use the test with subjects of different ages. The test includes 50 "simple" and 50 "complex" sentences. It starts with extremely direct simple sentences and moves on to the complex sentences. Sentence length is balanced for the set of the first 50 simple and the set of the last 50 complex sentences. The subjects were shown a set of three elaborated line drawings after each sentence and were asked to announce the number of the picture that they thought best characterized that sentence. For example, for the sentence "The man is watching the girl who is in the water" the choices were (1) a man and a girl on the sand, (2) a man on the sand and a girl in the water, and (3) a man in the water and a girl on the sand. Figure 1 shows these drawings.

Two aspects of performance on the RITLS were of interest: errors (numbers and patterns) and response time. Using a Sony Walkman WMD6 and a Sennheiser ME40 condensor microphone, the entire test was recorded without interruption for each subject. The time intervening between the conclusion of each read sentence and the subject's response was then measured by one listener (ETK) using a hand-held electronic stopwatch. Repeated measurements of the same subject's responses showed that these measurements were consistent to within 1/100 sec. The RITLS presents five exemplars of each of the following sentence types for which one example is noted in Table 2. The responses of each subject were examined to determine whether errors were "clustered" for particular sentence types.

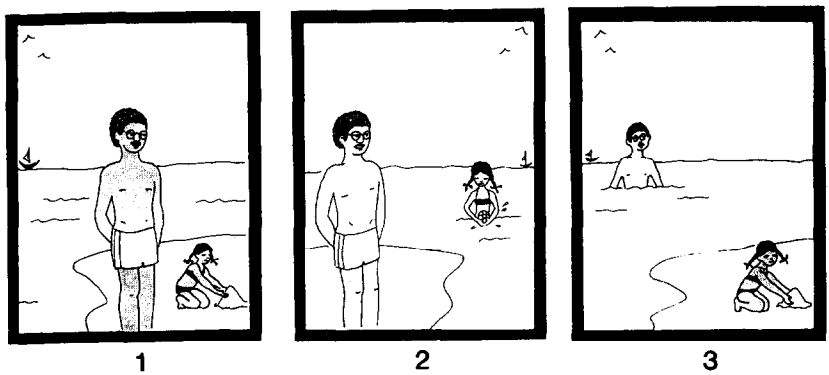

FIG. 1. Drawings corresponding to "complex" RITLS sentence 54, "The man is watching the girl who is in the water."

Speech samples. Following the RITLS, subjects were recorded as they read a set of 29 isolated words that each began with a stop consonant. These utterances were recorded on the same tape casette used for the RITLS. Speech analysis was subsequently performed using the BLISS computer system (Lieberman & Blumstein, 1988) with 12-bit quantization at a sampling rate of 20,000 samples per second using a MICROVAX II computer. The total duration of each syllable was measured as well as vowel duration and the initial stop consonant's VOT.

RESULTS

Voice Onset Time

VOT was measured and plotted for all of the monosyllabic words that each subject read aloud. As noted, one of the speech deficits associated with Broca's aphasia is a loss of control of VOT—the VOTs of the two classes of sounds overlap (Blumstein et al., 1980; Baum et al., 1990). A similar effect occurred for nine of our PD subjects of whom seven were moderates. Figure 2 shows a representative plot of VOT for a mild PD subject who behaves like normal nonafflicted speakers and maintained the VOT distinction for labial [b] versus [p], alveolar [d] versus [t], and velar [g] versus [k] stop consonants. Figure 3 shows VOT for a moderate PD subject who had a VOT "timing deficit" and merged VOT for voiced and unvoiced stops. The VOT overlap for each subject is presented in Tables 3A and 3B.

We derived a quantitative comparative measure of overlap by determining the "optimal VOT boundary" for these speakers using the procedure devised by Miller, Green, and Reeves (1986). Stop consonants having the same place of articulation were grouped for the overlap subjects and the nonoverlap subjects. The percentage of VOT overlap was then calculated by systematically setting and moving the VOT boundary; consonants having VOTs less than the boundary should be tokens of "voiced"

TABLE 2

RITLS—"Simple" sentence types and examples

Pattern 1—*The book fell.*
Pattern 2—*The girl hit the boy.*
Pattern 3—*The boy is happy.*
Pattern 4—*The building is a church.*
Pattern 5—*The boy is in the wagon.*
Imperative—*Open that door!*
Negative—*The boy did not eat the apple.*
Passive, reversible—*The boy was chased by the girl.*
Passive, nonreversible—*The ball was thrown by the boy.*
Dative—*The teacher is giving a book to the girl.*
Expanded—*The boy is picking apples from the front of the house.*

RITLS—"Complex" sentence types and examples

Adverbial clauses
  Main clause first—
    The dog barked because he had no food.
  Subordinate clause first—
    Because it was raining the girl stayed home.
Relative clauses
  Medial—
    The woman who is holding the baby has a hat on.
  Final—
    The man is watching the girl who is in the water.
Conjoined phrases—
    Mother cooked the food and the girl set the table.
Deleted structures—
    The boy ate his lunch but the girl didn't.
Non-initial subject—
    The one who is calling the boy is the girl.
Complements, subject—
    Father's washing the dishes made mother happy.
Complements, object—
    Father wants the dog to go out.

consonants, ones having longer VOTs should be "unvoiced" consonants. We determined the number of "voiced" and "unvoiced" stops that fell into the "wrong" VOT category for a particular VOT boundary. The optimal VOT boundary is that which produces minimal overlap. At the optimal VOT overlap measured by Miller et al. (1986) for non-PD subjects speaking at a normal rate, VOT overlap was 3.6%. Our PD subjects' mean syllable duration (300–500 msec) was similar to those of the normal subjects studied by Miller et al. (1986) speaking at their "normal" rate. Our nonoverlap PD subjects (those who maintained VOT distinctions like that plotted in Fig. 1) likewise had a mean 3.6% VOT overlap. In contrast, the VOT overlap was 18.3% for our nine VOT overlap subjects. The

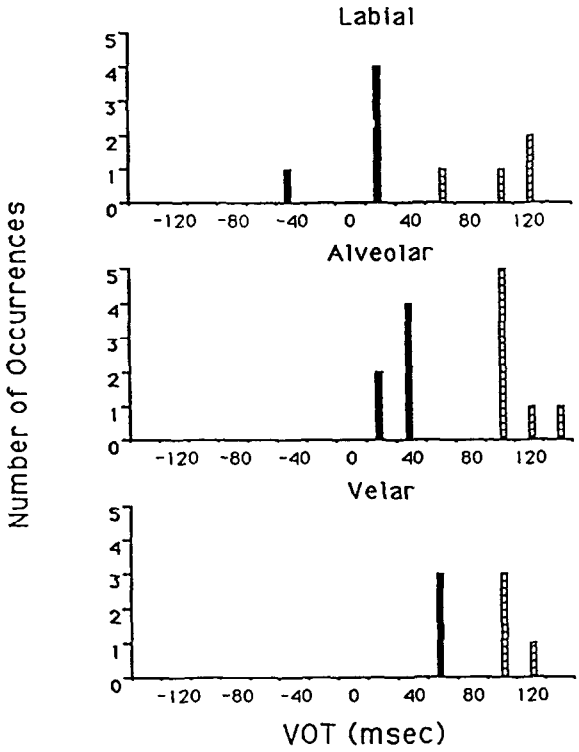

FIG. 2. Voice onset time (VOT) is plotted in milliseconds on the abcissa for a mild PD subject (MF). Note that VOT is distinct for "voiced" ■ and "unvoiced" ⊟ stop consonants.

difference between our VOT timing deficit group and normal subjects is significant ($p < 0.0001$, z test).

Syntax

Tables 3A and 3B also note the number of errors and mean response times for each subject on the RITLS. Moderates generally had higher error rates and longer response times than milds. A two-factor ANOVA analysis was performed in which we compared the errors and response times of mild and moderate PD subjects to the first 50 simple and last 50 complex sentences of the RITLS. The moderate PD subjects had significantly higher syntax error rates and longer response times than the mild PD subjects on the RITLS—$F(1, 76) = 14.15$, $p < 0.0003$ and $F(1, 76) = 30.74$, $p < 0.0001$, respectively. Both moderate and mild PD subjects also had significantly higher error rates and longer response times on the last 50 complex sentences of the RITLS than they had on the first 50 simple sentences—$F(1, 76) = 7.33$, $p < 0.008$; $F(1, 76) = 16.92$, $p <$

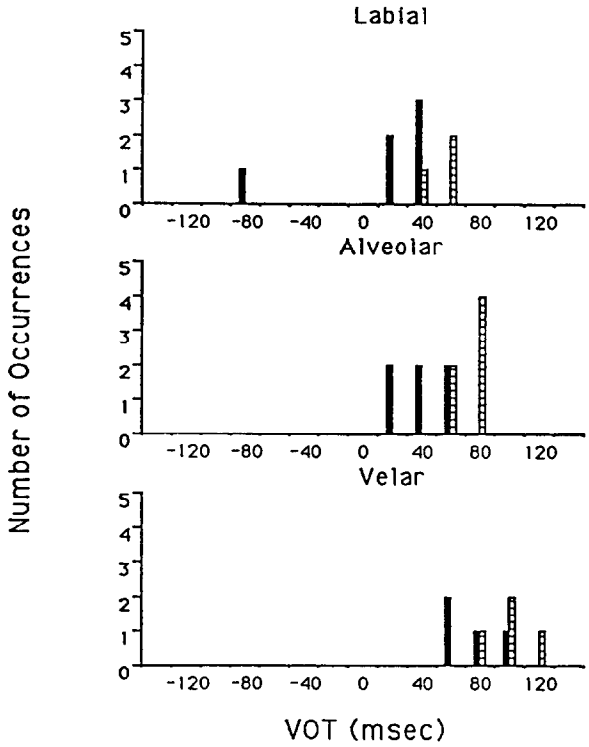

FIG. 3. Voice onset time (VOT) is plotted in milliseconds on the abcissa for a moderate PD subject (NO) who had a VOT "timing deficit" and merged VOT for voiced ■ and unvoiced ⊟ stop stops.

0.0001, respectively. This is consistent with syntactic complexity being the variable tested by the RITLS.

Two two-factor ANOVAS were performed in which we compared the errors and response times of the VOT overlap subjects and VOT non-overlap subjects to the first 50 simple and last 50 complex sentences of the RITLS. The ANOVAs indicate that the VOT overlap subjects had significantly higher syntax error rates and longer response times on the RITLS than the VOT nonoverlap subjects—$F(1, 70) = 12.38, p < 0.0008$; $F(1, 70) = 7.70, p < 0.007$ respectively. Pearson product–moment correlation coefficients were calculated. The correlation between the number of VOT timing errors and the number of syntax errors on the RITLS was significant ($r = 0.6473$, $p < 0.01$). Mean response time (RT) on the RITLS did not correlate with the number of VOT timing errors.

Clustered RITLS Errors and Increased Response Times

The RITLS presents six different sentences that exemplify the same syntactic construction. Therefore, it is possible to determine if a subject

TABLE 3A
RESULTS FOR MODERATE SUBJECTS, RITLS, AND VOT OVERLAP

Subject	Mean response time (sec)			Total number of errors			% VOT overlap
	1–50	51–100	1–100	1–50	51–100	1–100	
1	1.10	1.86	1.49	1	2	3	3.57
2	2.25	3.06	2.65	1	5	6	0
3	2.00	3.98	2.99	1	9	10	0
4	1.19	1.49	1.33	3	6	9	0
5	0.91	1.86	1.39	2	1	3	0
6	2.87	4.18	3.50	6	8	14	3.70
7	2.04	3.35	2.70	2	5	7	0
8	2.89	3.71	3.30	6	8	14	0
9	1.99	2.26	2.12	3	6	9	0
10	2.43	3.30	2.85	1	5	6	0
11	1.90	3.18	2.51	2	1	3	0
12	1.50	2.16	1.83	0	1	1	0
13	3.59	5.13	4.35	2	3	5	3.45
14	3.02	4.15	3.57	7	16	23	6.90
15	1.08	1.90	1.49	0	0	0	0
16	2.65	3.71	3.21	6	16	22	10.35
17	0.93	1.88	1.40	0	0	0	0
18	1.83	2.52	2.17	1	1	2	3.70
19	5.24	6.79	6.02	8	9	17	0
20	2.63	4.31	3.47	10	15	25	6.90
Mean	2.20	3.24	2.72	3.10	5.85	8.95	1.93
SD	1.05	1.32	1.17	2.97	5.16	7.80	3.08

is having difficulty comprehending a particular aspect of syntax when errors or increased response times "cluster" on sentences that involve the same aspect of syntax. Engen and Engen (1983) found that subjects who cluster three or more errors on a similar aspect of syntax exhibit the same behavior when they are retested. Accordingly we looked for clustered errors in which three or more errors occurred on similar sentence types. Moderate VOT overlap PD subjects had clustered errors on three sentence types of the RITLS: Simple expanded, complex adverbial initial subject clause, and complex complement subject clause sentences. One moderate subject (19) for whom VOT data were not available had clustered errors in adverbial initial subject sentences. Mild PD subjects had no clustered errors.

When the moderate PD subjects mean RTs for each of the 100 sentences of the RITLS were compared with those of the mild PD subjects, a series of clusters also was apparent. At a significance level lower than $p < 0.0005$, longer RTs occurred for the following simple sentence types:

TABLE 3B
RESULTS FOR MILD SUBJECTS, RITLS, AND VOT OVERLAP

Subject	Mean response time (sec)			Total number of errors			% VOT overlap
	1–50	51–100	1–100	1–50	51–100	1–100	
1	1.34	2.65	1.99	1	2	3	3.45
2	0.96	1.17	1.06	2	1	2	0
3	1.26	1.37	1.32	0	0	0	0
4	1.06	1.94	1.50	1	2	3	10.35
5	1.33	2.03	1.67	3	5	8	0
6	0.69	1.18	0.93	0	2	2	0
7	0.90	1.44	1.17	3	5	8	*
8	0.87	1.27	1.07	1	2	3	0
9	2.27	3.25	2.75	1	4	5	0
10	0.81	1.10	0.96	2	6	8	0
11	0.74	1.14	0.94	0	3	3	0
12	0.90	1.36	1.13	1	2	3	*
13	1.11	1.36	1.24	0	1	1	0
14	1.21	1.69	1.45	1	0	1	0
15	1.68	2.63	2.17	2	6	8	0
16	1.61	3.65	2.62	1	0	1	0
17	1.19	2.03	1.60	2	1	3	0
18	0.78	1.39	1.08	0	0	0	0
19	1.70	2.70	2.19	2	4	6	*
20	1.42	2.71	2.06	2	1	3	0
Mean	1.19	1.90	1.55	1.25	2.35	3.60	0.81
SD	0.40	0.77	0.57	0.97	2.01	2.68	2.60

* Word list not recorded.

Pattern 1 (present progressive), Pattern 5 ("to be" indicating place), Reversible Passive, as well as the following complex sentence types: final relative clauses, conjunction, deleted sentences, and non-initial subject. The correlation between mean response time and total number of errors on the RITLS proved significant for moderate PD subjects ($r = 0.6195$, $p < 0.005$), but not for mild PD subjects ($r = 0.1599$, $p < 0.05$).

Bilingual Normal Controls

The 10 bilingual controls who acquired English after age 7 years made a total of 11 errors. One subject made three unclustered errors, three subjects each made two errors, three subjects each made one error, while three subjects were error-free. These error rates are similar to those of monolingual age-matched normal controls (Engen & Engen, 1983). There was no correlation between the number of errors and the age at which the subject started to learn English.

Datatop Tests

Moderate PD subjects made significantly more errors than mild PD subjects on all trials of the Odd man out test and scored significantly worse on LTR, Delayed recall, Relayed recognition, and Digit span backward. Moderates recalled significantly more items in short-term recall than they could recall in long-term recall compared with mild PD subjects. The responses of moderate PD subjects were not significantly different from those of mild PD subjects on the New dot, Verbal fluency, and Digit span forwards tests.

DISCUSSION

Syntax Comprehension

The data indicate that PD disrupts aspects of the ability to comprehend syntax and that the extent of the disruption is a function of both the stage of PD and the complexity of the sentence. Vocabulary was balanced for sentences that had both simple and complex syntax which rules out hearing deficits. Sentence length may, in itself, have contributed to the clustered errors and longer RTs of the simple expanded sentences and conjunctions. However, the higher error rates and longer response times observed for certain sentences cannot simply be a consequence of general cognitive processes such as reduced attention span. If that were the case we should expect to find a decrement for all sentences, perhaps correlated with sentence length. Except for the increased RT for conjunction and clustered errors for simple expanded sentences the decrements cannot be ascribed to sentence length. Shorter sentences that were more complex syntactically had more clustered errors and longer RTs than longer syntactically simpler sentences. Moreover, the error rates and RTs for the 50 complex sentences were, as a group, significantly greater than those for the 50 simple sentences; these two sets of 50 sentences were balanced for overall length.

It is possible that the clustered errors for simple expanded sentences may derive from an impairment in processing "long-distance" syntactic dependencies similar to that noted by Baum (1989) for Broca's aphasics. Some PD subjects and Broca's aphasics may have difficulty keeping track of syntactic relationships when dependencies must be indexed over a longer word span. However, the higher error rates for the other complex sentences (adverbial initial subject and complement subject) cannot be ascribed to longer word spans intervening between syntactic dependencies. The longer response times for certain sentence types likewise indicate that they are in some sense more difficult to process. The longer RTs again cannot simply be ascribed to sentence length since they occurred for Pattern 1 (present progressive, Pattern 5, and the reversible passive; as well as complex final relative clauses, deleted, and non-initial subject sentences. Although response time and error rate are correlated for the

moderate PD subjects, the pattern falling out from response time and error rate form a disjoint set; no aspect of syntax has both a longer response time and a higher error rate. This might reflect a speed–accuracy tradeoff. Alternately it might indicate that certain sentences are too complex syntactically for some PD impaired subjects to comprehend at all. In short, syntactic complexity appears to have been a factor contributing to RITLS sentence comprehension deficits; this conclusion is consistent with the findings of Grossmann, Carvell, Gollomp, Stern, Vernon, and Hurtig (1991) who, using a different task, found that PD subjects "encounter more difficulty with increasingly complex grammatical phrase structures."

The RITLS errors of our study were similar in nature to those noted in a previous study of PD (Lieberman, Friedman, & Feldman, 1990) insofar as they generally occurred for only certain types of sentences that had more complex syntax. The RITLS error and RT pattern is also consistent with previous studies of the spontaneous speech of PD subjects which have noted differences in the syntactic complexity of the spontaneous speech of PD patients as well as increases in the number and duration of the short "hesitation" pauses that occur in the flow of speech (Illes et al., 1988; Illes, 1989). PD patients tended to use more nouns and verbs and fewer prepositions like at, in, to. Illes and her colleagues (1988) suggest that the differences in syntax may reflect the PD patients adapting their behavior to accommodate the increasing severity of dysarthria. However, since the speech production requirements of our comprehension procedure are minimal and were similar for all the sentence types, our subjects' errors must be direct consequences of the disease process, rather than some compensating action that minimizes speech motor activity.

Effects of Age of Acquisition of English

The possibility that the RITLS data were affected by the age at which some of the subjects acquired English was explored by testing bilingual Spanish–English speakers who had acquired English after age 7 years. Johnson and Newport (1989) demonstrate that the comprehension of complex syntax is sometimes impaired for bilingual subjects who acquired English after age 7 years. Two of our moderate PD subjects and one mild PD subject were bilingual and had acquired English after age 7 years. Moderate VOT timing deficit subjects (16 and 20) were bilingual and had acquired English at ages 13 and 20 years, respectively. A mild bilingual subject (14), who had no timing deficit, acquired English at age 13 years. However, our bilingual control group's error rate on the RITLS did not differ from that of the age-matched monolingual control groups. Therefore, the RITLS appears to be insensitive to the age-of-acquisition distinction that Johnson and Newport (1989) explored. This probably follows

from the fact that RITLS was designed to explore the basic elements of English syntax that young children acquire by age 7 years (Engen & Engen, 1983). Our control group of 10 bilingual speakers made a total of 11 RITLS errors; the highest number of RITLS errors for a member of the control group was 3 errors. In contrast, the two moderate bilingual PD subjects (16 and 20) who had VOT timing deficits made 20 and 25 errors, respectively. Moreover, mild PD subject 14, who fell into the nonoverlap VOT category, made only 1 error on the RITLS.

Effects of Medication

The drugs that our PD subjects were known to be taking enhance motor performance, reaction time, and affect when they are administered in proper doses. In toxic doses they may induce confusion, hallucinations, paranoid psychosis, and a host of other adverse mental effects. In our subjects, none of these were occurring; there was no evidence that these drugs had been taken in toxic doses. In fact, recent data clearly show that these drugs (particularly Sinemet—Carbidopa/Levodopa) enhance the performance of moderate PD subjects in cognitive tasks (Lange, Robbins, Marsden, James, Owen, & Paul, in press). The case profiles and RITLS response times and error scores noted in Tables 1 and 3, moreover, show that no general decrement in RITLS performance can be attributed to the drugs that the subjects were taking. The subjects whose medication was unknown produced no VOT overlap errors with one exception (mild 4). Only one of these subjects made more than 3 RITLS errors (mild 9 who made 5)—the mean RITLS error rate for mild subjects being 3.6 errors.

DATATOP Cognitive Tests

The results of the DATATOP tests conform to previous studies and previous data concerning syntax and cognitive deficits in PD. Although the primary symptoms of Parkinson's disease are motoric, a number of cognitive changes have been noted involving memory, abstraction, visual–spatial integration, processing time (Cummings & Benson, 1984), sorting tasks that involve planning (Flowers & Robertson, 1985), and a variety of cognitive tasks. Since the basal ganglia project to many cortical areas (Parent, 1986) the total pattern of cognitive deficits associated with PD is likely to be complex. The deficits noted are similar, but not identical, to those noted in frontal lobe lesions (Benson & Geschwind, 1972; Morris, Downes, Sahakian, Evenden, Heald, & Robins, 1988; Beatty, Staton, Weir, Monson, & Whitaker, 1989; Owen, Downes, Sahakian, Polkey, & Robbins, 1990; Lange et al., in press). For example, Beatty et al. (1989) found that semantic encoding of the lexicon appeared to be preserved in

PD, in contrast to deficits noted in frontal lobe lesions.[2] Generally, the cognitive deficits of PD involve planning and the ability to maintain a set. The deficits noted in the DATATOP tests, particularly those on the Odd man out test, fall into this category. It is important to note that moderates did not perform significantly worse than milds on the New dot test. It eliminated the possibility that moderates performed worse than milds on the RITLS because a visual deficit impaired their ability to distinguish between the elaborated line drawings.

The correlation between syntax and DATATOP tests is consistent with the data of Lieberman et al. (1990) where moderate PD subjects who had experienced some cognitive loss had higher RITLS error rates. Moderates and milds perform equally well on Verbal fluence. The Verbal fluency test measures ease and/or speed of retrieval and some dimensions of lexical structure. The absence of severe lexical deficits in our subjects argues against dementia of the Alzheimer's type and pathologic cortical damage. Subjects who have Alzheimer's dementia which derives from pathologic cortical damage show a different pattern of linguistic deficits; their syntactic abilities are preserved (Kempler, Curtiss, & Jackson, 1987) while their lexical ability is profoundly degraded (Kempler, 1988).

VOT Timing Deficits in Relation to Broca's Aphasia

VOT timing deficits similar to those noted in Broca's aphasia (Blumstein et al., 1980; Baum et al., 1990) essentially acted as a "probe" for syntax comprehension deficits that were also similar in nature to those noted for Broca's aphasia (Zurif, Caramazza, & Meyerson, 1972; Alexander et al., 1987; Baum, 1988). The nine subjects who had VOT timing deficits had significantly longer sentence comprehension response times and more errors than the subjects who had normal VOT timing (the correlation between the number of VOT and RITLS errors being significant, $r = 0.6473$, $p < 0.01$).

Neuroanatomical and neurophysiological data suggest that the speech and language deficits noted herein and similar deficits associated with

[2] Morris et al. (1988) also note that the deficits of PD are similar, but not identical, to those seen in frontal lobe lesions. The detailed pattern of deficits should be somewhat different if the circuit theory of functional neural organization (Mesulam, 1985) is correct. Lesions in frontal cortex would affect all the circuits to prefrontal cortex, whereas subcortical damage associated with PD would affect only those circuits connecting to frontal cortex through the structures affected. Since the subcortical pathways involving putamen and caudate nucleus are independent (Parent, 1986) the pattern of deficits in PD also would be variable (as we note herein). Beatty et al. (1989) suggest that the differences they note between their PD subjects and studies of frontal lobe damage are the result of damage in PD similar to that seen in dementia of the Alzheimer's type. However, whereas semantic coding is preserved in PD (as Beatty and his colleagues note) it is impaired in Alzheimer's dementia when syntax is preserved (Kempler et al., 1987; Kempler, 1988). Sahakian, Moris, Evenden, Heald, Levy, Philpot, and Robbins (1988) also show different patterns of deficits in visual memory and learning for PD and dementia of the Alzheimer's type.

Broca's aphasia have a common neurophysiologic basis—the deterioration of subcortical neural pathways, i.e., "circuits" linking Broca's area to prefrontal cortex. Damage to the putamen, caudate nucleus, and globus pallidus, as well as the internal capsule, have been noted in studies of aphasia (Naeser et al., 1982; Alexander et al., 1987; Baum et al., 1990). Pathways from caudate nucleus to globus pallidus pass through the internal capsule, as well as pathways to cortex from thalamus (Parent, 1986). These subcortical lesions could interrupt either, or both, of the independent "sensorimotoric" and "associative" basal ganglia pathways that connect cortex, putamen, globus palladus, substantia nigra, thalamus, and cortex (Delong, Georgopoulos, & Crutcher, 1983; Parent, 1986). Moreover, PET studies of aphasic and PD patients show cortical hypometabolism in prefrontal cortex (Metter et al., 1984, 1987, 1989) and caudate nucleus (Metter et al., 1984).

Other subcortical pathways may also exist linking prefrontal cortex to Broca's area (Alexander et al., 1987). However, the similar pattern of VOT timing deficits and syntax comprehension errors noted here for PD is consistent with basal ganglia involvement in Broca's aphasia. The fact that VOT timing deficits act as a "probe" for syntax comprehension errors may reflect the specificity of basal ganglia pathways to and from Broca's area and/or the robustness of particular pathways in relation to the disease process. Further studies with more severely impaired PD subjects may resolve this issue.

Our PD data also illuminate another issue—the variable pattern of deficits encountered in Broca's aphasia (Stuss & Benson, 1986; Caplan, 1987). These may derive, in part, from damage to these anatomically distinct subcortical basal ganglia pathways. Although VOT timing and sentence comprehension deficits were correlated for our PD subjects, the pattern was not completely similar. Many moderate PD subjects who had long syntax test RTs had normal VOT timing as well as the reverse pattern. No correlation was evident for the mild subjects; only two mild PD subjects had VOT timing errors and the correlation was nonsignificant (0.16). This variable pattern is consistent with neurophysiologic theories that propose independent subcortical putamen and caudate nucleus "sensorimotoric" and "associative" circuits connecting different cortical regions, which can be damaged to different degrees in PD (Albert et al., 1974; Delong et al., 1983; Parent, 1986). The subcortical lesions that result in permanent Broca's aphasia may affect one, or both, of these pathways. The cognitive impairments noted by Goldstein (1948) for the aphasias may likewise reflect lesions that interrupt pathways to prefrontal cortex.

CONCLUSIONS

The neurological basis for these VOT, syntax, and cognitive deficits appears to be the deterioration of the circuits that, in turn, reduce pre-

frontal cortical activity. Prefrontal cortex is involved in the regulation of fine motor timing (Stuss & Benson, 1986), but as Stuss and Benson note,

> one group of behavioral control functions that appear to be dependent on the prefrontal cortex concerns the ability to organize and maintain related information in fixed sequence, the establishment of related sets of this information, and the integration of these data with other information to form novel or meaningful interpretations. (1986, p. 242)

The comprehension of the meaning of a sentence is precisely the sort of cognitive problem that involves that application of stored knowledge, the "rules" of grammar, to a novel problem—a new sentence. The comprehension of a sentence involves deciding what syntactic rules apply as well as the integration of these rules with the words of the sentence and pragmatic real-world knowledge.

The cognitive and syntactic comprehension deficits that occur in PD, in this light, appear to have a common physiologic basis—the disruption of neural circuits to prefrontal cortex. Prefrontal cortex is clearly involved in nonlinguistic cognitive activity as well as motor control (Stuss & Benson, 1986). Thus it makes little sense to attribute human syntactic ability to an "encapsulated module" of the brain, i.e., a neural system that operates only in the domain of syntax. Nor can the neural domain of the module be restricted to cortical structures. The syntax module can, of course, be restricted to the mechanisms by which a person's knowledge of the syntactic rules of his/her language is encoded, but syntactic ability will deteriorate if these rules cannot be accessed or applied.

Modules can, of course, serve as functional concepts but they ultimately must be related to neurophysiology. The general model proposed by Mesulam (1985), in which specialized neural structures operate in different behavioral domains through different neural pathways, can serve to account for the neural embodiment of functional modules. The particular speech and syntactic deficits noted herein for PD appear to result from the deterioration of different basal ganglia "circuits" between Broca's area and prefrontal cortex (Lieberman, 1991). The deterioration of subcortical pathways to prefrontal cortex in PD also affects the aspects of cognition noted herein.

Our data do not directly address the question of whether a hypothetical store of syntactic rules deteriorates in PD. Insofar as our subjects often maintain sentence comprehension with longer response times, we may conclude that the problem is one of "performance," but the performance–competence distinction is itself meaningful only if competence is equated with the specific store of syntactic rules or principles. The syntax comprehension deficit noted here for PD is again similar to that noted for Broca's aphasia—an impairment in the ability to apply the rules of syntax

in an automatic, rapid manner. The performance of agrammatic aphasic subjects improves in tasks that allow more processing time (Baum, 1988). The most likely explanation for the comprehension errors noted in this experiment and in those by Lieberman et al. (1990), Grossman et al., (1991), and the simplified syntax noted by Illes et al. (1988) would appear to be deterioration of the subjects' ability to make use of the syntactic "rules" of English.

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