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Neurolinguistic Features of Spontaneous Language Production Dissociate Three Forms of Neurodegenerative Disease: Alzheimer's, Huntington's, and Parkinson's

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An analysis of the temporal (prospective) form (silent and filled hesitations, repetitions, incomplete phrases, context-related comments, interjections), syntactic form, and lexical (retrospective) form (verbal deviations, open and closed class phrases) of spontaneous language production of early and middle stage Alzheimer's, Huntington's, and Parkinson's patients was made. Results showed that the language structure was disrupted in each disease, but in different ways. Temporal interruptions of varying types were frequent in the language of Alzheimer's and Huntington's Disease patients; only long-duration silent hesitations were frequent in Parkinson's language samples. Syntactic complexity was reduced in Huntington's Disease. Verbal paraphasias were found in both the language of Alzheimer's patients, as well as moderately advanced Huntington's patients. Closed class phrases were predominant in the language of Alzheimer's patients and Huntington's patients, and open class phrases in the language of Parkinson's patients. Taken together, the results suggest that (1) there is a unique neurolinguistic profile for spontaneous language production for each neurodegenerative disease, (2) pathology of the neostriatum disrupts syntactic organization, (3) adaptive strategies are used to cope with verbal and speech-motor difficulties, and (4) adaptive strategies fail to be effective with increasing disease severity.

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INTRODUCTION

Given classical connectionist models of language processing, it is not surprising that changes in language function are seen in Alzheimer's Disease (AD), the progressive neurodegenerative disease with severe neuropathologic changes in the region of the temporoparieto-occipital junction. Given other historical and contemporary models of language functioning, particularly those that include involvement of subcortical regions (e.g., Marie, 1906; Pribram, 1976; Alexander & LoVerme, 1980; Damasio, Damasio, Rizzo, Varney, & Gersh, 1982; Naeser et al., 1982; Metter et al., 1984; Gordon 1985b), the nature of language-related changes in neurodegenerative diseases in which pathology is initially seen in basal ganglia, such as Huntington's Disease (HD) and Parkinson's Disease (PD), is of central interest. Moreover, whether alterations of both language comprehension and language production in these diseases are a function of the underlying neuropathology (Gordon, 1985b; Gordon & Illes, 1987) or of a generalized dementia (Bayles, 1982, 1984; Bayles & Boone, 1982; Bayles & Tomoeda, 1983; Bayles, Tomoeda, Kaszniak, Stern, & Eagans, 1985) has been a topic of some controversy in the recent neurolinguistic literature.

The present study was undertaken in an attempt to resolve some aspects of this controversy by evaluating the temporal, syntactic, and lexical forms of the language structure in spontaneous language production in early and middle stages of AD, HD, and PD. The specific objectives were (a) to study, *on-line*, the dynamic processes underlying planning, organization, and production of language as measured by the temporal form of the language structure; (b) to identify alterations in the syntactic and semantic aspects of language production; and (c) to determine if alterations in the structure of language arise from primary deficits attributed to the neuropathology of each disease, to a generalized dementia, to adaptive strategies used to cope with functional deficits, or to some combination of these phenomena.

METHOD

Subjects

Ten Alzheimer's patients (AD), 10 Huntington's patients (HD), 10 Parkinson's patients (PD), and 10 healthy persons (NC) matched for sex, age, and level of education were referred to the author for this study by collaborating neurologists. Subjects were male, right-handed, literate, unilingual native speakers of English, and none had any previous history of neurologic or psychiatric abnormality. Diagnosis was made on the basis of clinical and neuropsychological examination, CT scan, and, where appropriate, genetic history. Most subjects were living in the community at the time of testing.

Five subjects in each group were identified as being at an early stage in the disease, and five subjects were identified as being at a middle stage in the disease. The early AD (ADe; mean age 71.2) patients were distinguished from moderate AD (ADm; mean age 74.5) patients on the basis of the Global Deterioration Scale (Reisburg, Ferris, de Leon,

& Crook, 1982; early scores, 2.0–3.5, moderate score, 4.0–5.5). Early HD (HDe; mean age 44.0) patients were identified with an onset of symptoms within 4 years and a diagnosis within 3 years prior to testing. The patients comprising the moderate group (HDm; mean age 52.4) were each within 6 and 9 years post onset of symptoms and had a diagnosis within 10 years (Cummings & Benson, 1983). The early PD (PDe, mean age 56.4) patients and moderate PD (PDm, mean age 66.0) patients were distinguished according to the Webster scale (Webster, 1978; early score, 1–10, moderate score, 11–20). PD patients and their significant others reported no major change in cognitive ability. In contrast, patients in all other groups reported at least some cognitive change, most notably a memory loss.

Language Samples

Subjects were tape-recorded while they answered questions to open-ended autobiographical questions. Subjects appeared to have good verbal comprehension and all made an overt effort to answer interlocutors' questions. Topics pertaining to where the subject was born and raised, his work, and travel were extracted and transcribed for analysis. These topics provided samples of speech of equal difficulty (Ford, personal communication). Language samples ranged in duration from approximately 2 to 8 min, depending on the extent to which subjects responded to the target questions. The variability in duration of the language samples was accounted for in the analysis (cf. below).

Neurolinguistic Analysis

The Montreal–Stanford Neurolinguistic Protocol (MSNP) was used to analyze the language samples. The method has been described in detail elsewhere (Illes, Metter, Hanson, & Iritani, 1988) and will be reviewed briefly here.

a. Temporal form. The number of words produced per language sample (sample rate) and the number of words per actual speaking time (verbal rate, or words per minute minus hesitation time) were measured. The duration, frequency, and linguistic environment of all silent hesitations exceeding 200 msec (Ford & Holmes, 1978) were assessed. The duration of silent hesitations was determined using a Bruel & Kjaer Level Recorder with a running speed of 30 mm/sec and a temporal resolution of 60 dB/mm. Language samples were high pass filtered at 80 Hz. Frequency was determined by grouping silent hesitations according to duration. The linguistic environment of the silent hesitations was assessed by selecting five syntactic positions—at the beginning of sentences, preceding embedded clauses, between mandatory phrases, between mandatory or optional phrases and optional phrases, and within phrases—and calculating their occurrence at each position. Because interviews were not conducted under identical conditions, and because several different interviewers participated in obtaining these speech samples, the time between the end of the interlocutors' questions and the onset of a response by the speaker was not considered for analysis.

The number of words per filled hesitation (*euh, ums*) in each language sample was calculated, and the number of phonemic approximations (Joanette, Keller, & Lecours, 1980) was calculated per total number of words. The number of self-corrections (whole word repetitions) and aborted phrases (false starts) and the combination of modalizations and interjections were calculated per number of phrases in each language sample.

b. Syntactic form. Syntactic complexity was examined using a scale developed by Illes and Ford (1984, unpublished). With the assumption that the principal planning unit for spontaneous language production is the basic clause (Ford, 1978) (subject + verb; for transitive verbs; + object), a score (weight) of complexity was given to each clause produced. The scale ranged from the simplest nonembedded utterance such as a clause without a subject (score = 1), to clauses with multiple arguments (score = 4), to embedded clauses, including coordinated clauses, complements and adverbials, and relative clauses (score for nonembedded form plus a weight of 3). Weights were applied for each level of

embedding. A mean degree of complexity was thus established for the language production of each subject.

c. Lexical form. Two analyses were conducted to examine lexical form of the language structure. The first examined the extent to which the language output was distorted at the word level (number of verbal deviations per number of words). The second considered the proportion of open class and closed class subject phrases, predicate phrases, mandatory phrases, and optional phrases occurring per language sample.

RESULTS

Temporal Form

a. Production Rate

Figure 1 shows the means for sample rate and verbal rate. A MANOVA applied to these data showed that while the rates between two different diseases may be varied only by chance ($F(6, 33) = 2.38$, $p < .0511$), differences within the groups were highly significant ($F(1, 33) = 43.88$, $p < .000001$). The mean square error for the within-groups effect was computed in order to carry out the Fisher LSD test between the means of each group of the two measures. The LSD for comparisons within the disease groups was 37.71; within the normal group, it was 26.67. Three of the seven possible comparisons exceeded the LSD. These significant comparisons applied to early HD group (49.90), and to both levels of the PD group (65.00 and 48.50, respectively).

b. Silent Hesitations

b.1. Duration. The frequency of silent hesitations, grouped according to duration, was approximately the same for all groups, with silent hesitations commonly in the range of 200–600 msec. An ANOVA applied to compare only the proportion of pathologically long silent hesitations (see Gordon & Illes, 1987) showed that different disease groups varied

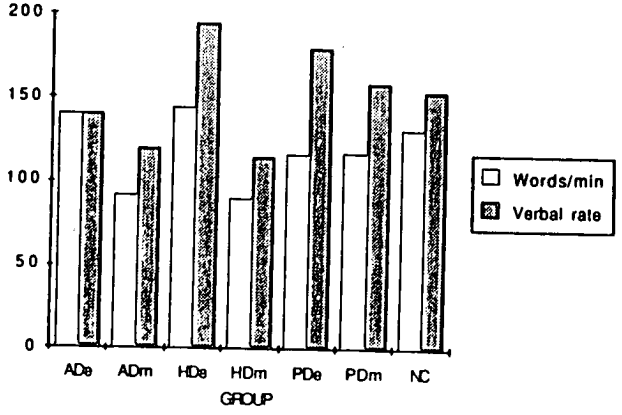

FIG. 1. Mean sample rate and verbal rate for each group.

widely on this measure ($F(6, 33) = 4.25$, $p < .002$). The LSD in number of long (> 2 sec) hesitations among means for disease groups with $p < .01$ was 21.07. Among 21 possible comparisons between groups, the means for the ADm group (21.4) and PDm group (21.2) significantly exceeded the mean for the NC group (2.1).

b.2. Linguistic environment. A MANOVA showed significant differences in the duration of silent hesitations at different syntactic positions ($F(6, 33) = 2.77$, $p < .02$). The LSD for differences between disease groups was 1,063.91 msec; for comparisons of disease groups with the normal group, the LSD was 921.38 msec. Among the possible comparisons, nine pairs of means from the beginning-of-sentence position exceeded the LSD. Two means for the PDm and HDm groups exceeded the means for all other groups. Two comparisons between means for the duration of silent hesitations between mandatory phrases and optional phrases also exceeded the LSD: PDm compared with ADm and with NC.

c. Self-corrections

An ANOVA revealed a statistically reliable separation in the use of self-corrections between groups ($F(6, 33) = 3.768$, $p < .005$; Fig. 2). Three comparisons exceeded the LSD of 73.41 for comparisons between disease groups, and 63.74 for comparisons between disease groups and the normal group: HDm > NC, ADm > PDe, and ADm > NC.

d. Aborted Phrases

An ANOVA revealed significant differences in the occurrence of aborted phrases ($F(6, 33 = 5.31$, $p < .0006$; Fig. 3). The LSD for

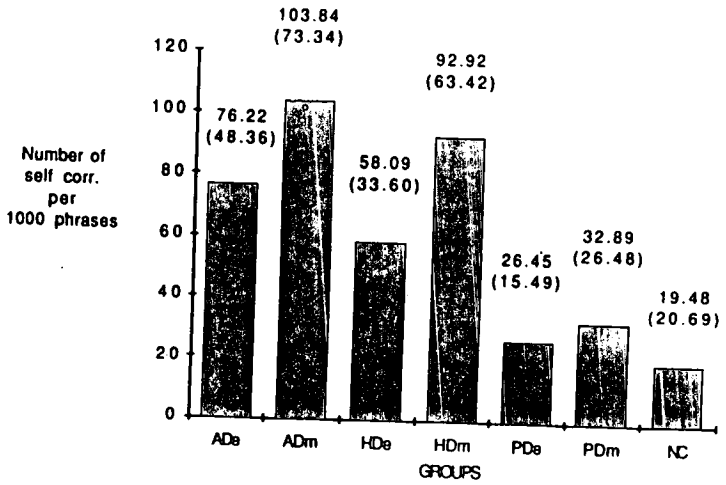

FIG. 2. Mean number of self-corrections occurring per 1,000 phrases.

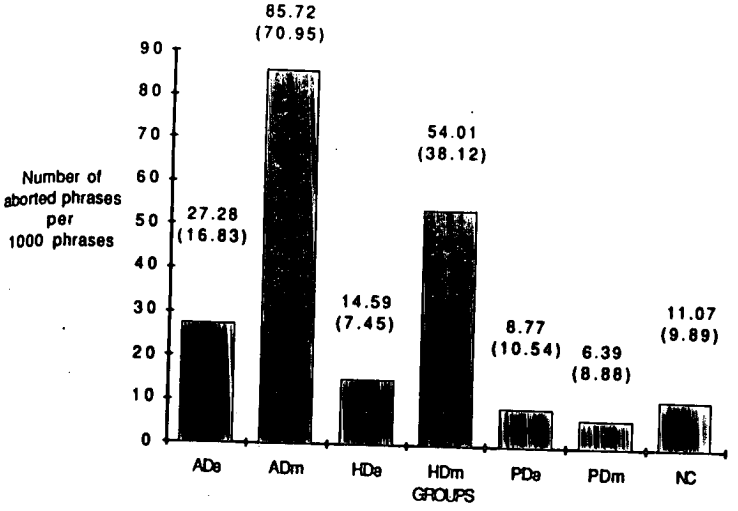

FIG. 3. Mean number of aborted phrases occuring per 1,000 phrases.

comparisons among pairs of means of the disease groups was 51.51; it was 44.61 for comparisons between means with the normal group. Of the possible comparisons, the mean of the ADm samples exceeded the means for all other groups, including its early counterpart. A similar tendency for the HDm group was not statistically significant.

e. Filled Hesitations, Phonemic Approximations, Modalizations, and Interjections

The analysis of the number of words per filled hesitation, mean number of phonemic approximations per numbers of words, and the relative occurrence of interjections and modalizations did not reliably differentiate the groups. All groups showed a great deal of variability for these measures.

Syntactic Form

The mean scores for syntactic complexity were 4.40 and 3.89 for the AD groups, 3.59 and 3.61 for the HD groups, 4.37 and 3.59 for the PD groups, and 4.38 for the NC group $F(6, 33) = 2.42$, $p < .04$). The LSD for comparisons among disease groups was 1.06 ($t = 2.75$, $p < .01$); for comparisons with the normal group it was 0.91 ($t = 2.75$; $p < .01$.). For this analysis, the LSD led to the anomalous situation in which the overall F ratio was significant, but none of the pairwise differences among means exceeded the LSD, possibly due to the small N. Because the mean scores for the two levels of the HD group were almost identical, the two groups were combined and a new LSD was computed. With $n = 10$, the LSD

was 0.75. Using this LSD, syntactic complexity was significantly less in the HD language samples than in the ADe, PDe, and NC samples.

Semantic form

a. Verbal Deviations

A MANOVA revealed significant differences in the occurrence of neologism, semantic paraphasias, and phonemic paraphasias in the language samples of each group (Fig. 4; $F(6, 33) = 5.24$, $p < 0.0073$). None of the pair-wise comparisons exceeded the Fisher LSD, not a surprising finding, however, since the number of observations was limited. Upon visual inspection of the data, it is apparent that only patients in the ADe, ADm, and HDm produced neologisms.

b. Open and Closed Class Phrases

The occurrence of open class phrases (and by extension closed class phrases, since the two are complementary) was significantly different in the language samples of the groups ($F(6, 33) = 16.87$, $p < .00001$). The mean percent of open class phrases produced by the normal group was 62.4. In comparison, the means for the other groups were ADe = 51.2, ADm = 38.8, HDe = 53.5, HDm = 55.2, PDe = 72.4, and PDm = 73.2. The LSD for comparisons between means of the disease group was 11.51; for comparison with the normal group, the LSD was 9.97, and more than half the total number of comparisons (14 of 21) exceeded the LSD.

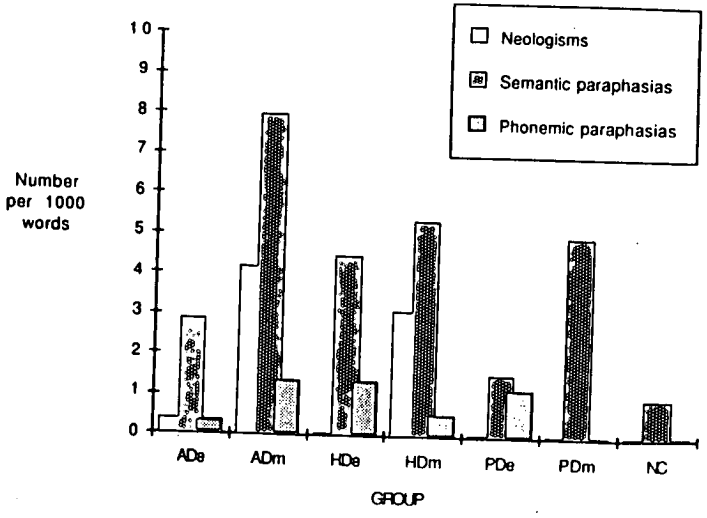

FIG. 4. Mean number of neologisms, semantic paraphasias, and phonemic paraphasias occurring per 1,000 words.

DISCUSSION

The number of words that a person is able to produce in a given test in a given amount of time is usually considered to be a sensitive measure of his ability to access a *mental lexicon*. As Bayles (1982) has suggested, there are many possible entry points into the mental lexicon, and patients with *dementia* tend to perform poorly on lexical tasks, regardless of mode of entry. The majority of these tasks, however, are not sensitive to the dynamic processes by which language responses are formulated (i.e., intention) and planned (i.e., a hierarchical process). Despite some of the intrinsic shortcomings of analyzing spontaneous production, such as not knowing precisely what a speaker is trying to say (Studdert-Kennedy, 1983) and variability in language sample length, such an analysis is sensitive to these more dynamic aspects of language production. Figure 5 summarizes the results of the extensive analysis of spontaneous language production performed for this study using the MSNP.

Temporal Form

a. Production Rate

The results of the test for production rate suggest that timing and presence of hesitations are important factors in language production in early HD and in both early and middle stage PD, and that there is a certain press of speech once speech is initiated. Two possible explanations may be given to account for the short rapid language segments in these samples. One is a speech-motor explanation and would suggest that, similiar to the motor cogwheel phenomenon seen in PD patients,

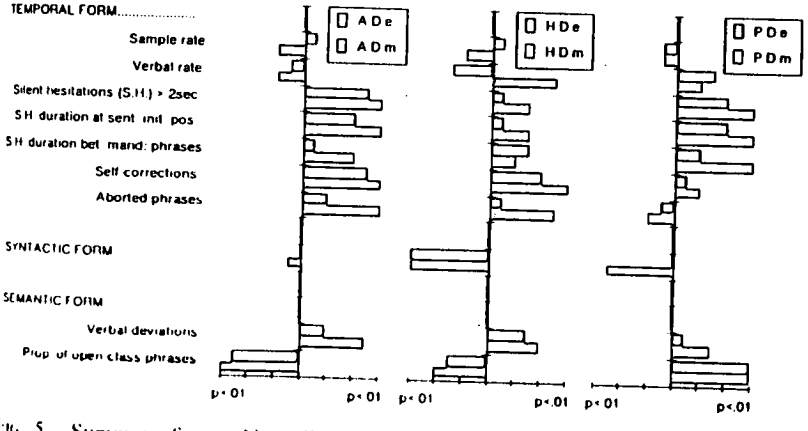

FIG. 5. Summary figure: Neurolinguistic variables significantly distinguishing disease groups. Length of each bar is determined by rank order as scores departed from the normal mean for that measure. Note: A statistically significant departure from normal is achieved for syntactic form when combining mean complexity scores for early and middle HD groups.

initiation of speech is a difficult process, but once done, speech may be very rapid. A second explanation for the phenomenon can be given in terms of coping or adaptive strategies. For example, it is possible that as the patients became aware of changes in their language, they began to speak rapidly so as not to forget an ongoing topic or a recently and successfully accessed lexical item (Gordon, 1983). The press of speech eventually disappeared in the HDm group, possibly suggesting a generalized slowing down of cognitive processes.

The absence of significant changes in rate measures for the ADe group is consistent with previous knowledge about their relative fluency as compared with normal speakers (e.g., Cummings & Benson, 1983).

b. Duration and Distribution of Silent Hesitations

Results of the analyses of silent hesitations revealed that duration, rather than frequency, is an important aspect of spontaneous language production of subjects tested in this study. Patterns for the NC group were consistent with patterns of silent hesitations in spontaneous language production of young adults described by Ford (1978). The presence of silent hesitations at the beginning of sentences in PD samples as well as AD samples suggests that their occurrence is not strictly due to a motoric difficulty in producing speech sounds. Although the hypokinetic dysarthria of PD patients has been well documented (e.g., Darley, Aronson, & Brown, 1975; Metter & Hanson, 1986), such speech-motor problems are not characteristic of AD. Therefore, it may be argued that the long silent hesitations at the beginning of sentences reflects a difficulty in the ability to either formulate or plan an upcoming linguistic sequence.

The absence of a significant effect for the HD group on this measure is surprising and is in contrast to a report by Gordon and Illes (1987). Their study differed from the present one in two respects, however. First, their neurolinguistic analysis was made on speech samples of a group of mild to severe HD patients, whereas severe patients were explicitly excluded in the present study. Second, in the Gordon and Illes study, all recordings were made by one examiner, making it possible to measure the duration of silent hesitations occurring at the very onset of subjects' responses to the interlocutor. This was not possible in the present case. In another study by Gordon (1985a), which was based on the same patients as the Gordon and Illes study (1987), Gordon demonstrated that HD patients require more prompts, or questions, to elicit approximately an equivalent amount of speech compared to normal. Although the actual number of prompts was not considered here, the evidence suggests that HD patients formulate and plan at the beginning of responses, namely at *response-initial idea boundaries*. Multiple sentences may thus be formulated at a time, and given the large cognitive

load, the corresponding silent hesitations should be longer than those associated with the planning of single sentences.

In comparison with the HD data, the long silent hesitations at *sentence boundaries* in the language samples of ADm and PDm patients may reflect a more local difficulty with decisions concerning the linguistic integration of clauses, and with the selection and positioning of noun, predicate, and object phrases. The long durations of silent hesitations between mandatory and optional phrases in the PDm samples is consistent with the results of Ford (1978), who showed that in normal speech, there is a tendency for silent hesitations to be longer before phrases that merely add information to a principal utterance without modifying it. This phenomenon is related to the fact that silent hesitations are more likely to occur before clauses or phrases that are themselves preceded by complete and meaningful speech.

In their study of language production of the same PD patients tested here, Illes et al. (1988) found an elevated number of open class optional phrases. They concluded that PD patients produce many open class optional phrases in an effort to adapt to their speech-motor difficulties and produce as much information as possible in a single sentence. In principle, this reduces the overall amount of planning and speaking that has to be done to relate a given message. It appears, however, that the production of open class optional phrases, although an effective measure to cope with functional difficulties, becomes more difficult as the disease advances, both in terms of keeping track of distant subjects and in terms of lexical access. The reduction of the effectiveness of this mechanism may be evidenced by the long silent hesitations preceding optional phrases in the language samples of the more advanced PD group, as compared to PDe and NC samples.

Given the difficulty with word naming, recall, generation, and description that patients with dementia tend to have (Bayles, 1982), it was hypothesized that an increase in the number of within-phrase hesitations would occur in all disease-group language samples. None of the disease groups deviate from the normals on this measure, however. This surprising result does not necessarily preclude a word finding difficulty, but may imply that in relatively unguided spontaneous language production, patients adopt strategies to limit and possibly conceal their word finding difficulties. The effectiveness of the various strategies does seem to diminish with increasing severity and frontal lobe involvement in each disease.

Self-corrections, Aborted phrases, Filled Hesitations, Phonemic Approximations, Modalizations, and Interjections

Like silent hesitations, filled hesitations, modalizations and interjections, repetitions, and aborted phrases may all reflect aspects of the

dynamic processes of language production. The cluster of "vocal" temporal variables may also serve a pragmatic function in that, to a large extent, they epitomize the character and context in which discourse takes place. They may serve to index a speaker's ability to monitor what he has said, what he wishes to say (Berman & Peelle, 1967), or both, or to signal the speaker's intention to the listener.

Of the vocal temporal variables, self-corrections (whole word repetitions), and aborted phrases significantly distinguished the language samples. There tended to be many "vocal" temporal interruptions in the AD and HD speech samples overall, and few in the PD samples. A possible explanation for these findings is that patients in the ADe and HDe groups repeated words and phrases in an effort to keep them roughly "on the tip of the tongue" (Brown & McNeill, 1966; Gordon, 1983; Illes, Nespoulous, & Lecours, 1986) while they planned an upcoming sequence. The increased number of self-corrections in the ADm language samples, as compared with the PDe and NC samples, may suggest that even at this later stage of Alzheimer's disease, patients may retain some awareness of their verbal difficulties. This phenomenon is illustrated in the following example:

And euh all ↓ the children are all from /fræn↓/↓ in ↓ San /fræn↓/↓ /fræn↓/↓ San Francisco.

While the self-corrective strategy appeared to be effective in facilitating lexical retrieval or keeping within context in the earlier stage of AD, the significant increase in the number of aborted phrases in the ADm (and HDm) samples may reflect the eventual failure of this strategy. Hier, Hogenlocker, and Shindler (1985) have also reported that the production of aborted phrases (termed *aposiopeses* by these authors) is a frequent occurrence in AD speech. Their interpretation, however, is that aborted phrases may represent a nonlinguistic deficit reflecting patients' failure to perceive the necessity to complete their utterances. The nature of the self-corrections, combined with many modalizations and interjections (cf. below) in the language samples of AD patients studied here, do not support this interpretation. Obler and Albert (1983) have reported that AD patients tend to make superfluous comments to the examiner during language evaluations, and these were interpreted to reflect patients' loss of knowledge of the rules of discourse. In the AD language samples studied here, however, the interjections and modalizations tended to indicate that patients were aware of their own verbal difficulties as well as the presence of the interlocutor. For example,

Then when we got to Seattle, we took the ↓ the ⇓ euh What do you call it? The ⇓ I get stuck on ↓ on What I'm trying to say.

Or,

. . . /p ↓/↓ /p rs↓/↓/p θ↓/⇓ Oh boy. I'm sure it's ⇓I↓ I feel like a ↓ like a nut.

In PD language samples, the relative lack of temporal interruptions could suggest that PD patients are not aware of their language or speech-motor performance. Another possibility, however, is that they respond to their speech-motor difficulties by avoiding temporal interruptions, particularly extraneous or empty speech. This is consistent with the well-formed and highly informative character of PD speech, described by other measures (cf. below, and discussed in Illes et al., 1988).

Syntactic Form

By grouping the HD patients, a reduction in syntactic complexity was demonstrated. An example is

Okay I↓I had a nice one, my wife and I. Years ago we went down to the Caribbean. And so I saw ↓ I had never seen the ocean before. It was really beautiful. I don't know. Have you traveled much? Maybe you've seen the Caribbean. It's just outstanding to see the beauty of the water. Just euh such a ↓ beautiful euh thing. You know, the ↓ the water ⇓ . . .

The reduction in syntactic complexity may reflect motor dysfunction. However, because reductions were similar in HDe and HDm samples, the finding may also provide evidence that pathology of certain regions of the basal ganglia, in this case the caudate nucleus, directly affects linguistic processing.

The results of analysis for syntactic complexity of the AD and PD samples suggest that the "hierarchical organization" of language remains essentially intact in these diseases. This is consistent with the patterns of language production usually reported for AD patients and with the similarity of these patterns to those in the fluent aphasias. Although syntax was essentially spared in the early course of AD, a tendency for syntax to become more simplified, with frequent violations of grammatical rules (paragrammatism) was noted in the later stage language samples.

Lexical Form

a. Verbal Deviations

Although only relatively small numbers of verbal deviations were found in the language samples in this study overall, as expected, the greatest number of all types of verbal deviations was found in the ADe and ADm language samples. Some neologisms and semantic paraphasias were also noted in HDm samples. According to Gordon (1985b), this phenomenon

may arise from the disconnection of afferents from the superior temporal gyrus to the head of the caudate nucleus in the later stage of the disease.

b. Open versus Closed Class Phrases

In the analysis of lexical form, the disease groups had different profiles. PD patients produced significantly more open class phrases than the NC group, AD patients produced significantly fewer open class phrases, and HD patients did not differ from the normals on this measure.

In order to better understand the nature of this dissociation, the open and closed class phrases were separated at the functional level: subject, predicate, mandatory, and optional phrases. The differing profiles were still evident for subject, mandatory, and optional phrases, but less apparent for the predicate phrases. This is consistent with the fact that the choice of predicates is not the same as for other classes of phrases. For example, there are only two closed class predicates, i.e., *to be* and *to have*, they are acquired differently, and they tend to behave differently in the language (Gentner, 1985).

The many closed class phrases and relative lack of open class referents in the AD language samples are consistent with the reported egocentric and empty speech of AD patients (Bayles, 1984). In PD, the increase in open class phrases appeared to serve the function of reducing the processing load for the speakers, with as much information as possible concentrated around each clause. There was no evidence that the phenomenon is due to perseveration, since each phrase, including optional phrases, added original information to the principal clause. These results for open class (optional) phrases are consistent with the study by Obler, Mildworf, and Albert (1977), who have demonstrated that in written description, PD patients use more words than normal subjects to describe the same number of themes. It must be considered, however, that the increase in the number of optional phrases may also be due in part to the reported inability of PD patients to exist from their *cognitive loop* (Bowen, Kamienny, Burns, & Yahr, 1975; Pirozzolo, Hansch, Mortimer, Webster, & Kuskowski, 1982).

SUMMARY

To summarize, the MSNP revealed differences in language production between patients identified to be in the early or middle stages of AD, HD, and PD. Language production in early AD was best characterized by self-corrective strategies, verbal deviations, and closed class phrases. Relatively more utterances were completed in the ADe language samples than in the language samples of their later counterparts. Hierarchical organization, as reflected in the syntactic complexity of the language structure, was not impaired in early AD.

HD patients differed the least from the normal speakers studied here, except on the measure of syntactic complexity, on which they were

shown to be reduced, and production rate, for which a press of speech was noted. Unlike AD patients, HD patients seem to formulate their messages at response-initial idea boundaries rather than at sentence boundaries. HD language samples did resemble those of AD patients, however, in terms of lexical form, with a tendency toward closed class phrases. In all respects, however, the HD patients studied here were less profoundly affected than the AD patients.

By contrast, PDe language samples were particularly well formed in the sense that they were intact syntactically and essentially devoid of extraneous utterances. This may be evidence for adaptation of PD patients to their speech-motor difficulties—a conclusion supported by the many open class phrases. The latter also argues against any evidence for word finding difficulty in at least some PD cases. Whether or not the increase in duration of silent hesitations preceding optional phrases reflects the onset of an impairment of lexical access remains an open question at this time.

While the MSNP is not intended to serve as a diagnostic tool, it does reveal that each of the disease groups tested here, albeit small, has a unique neurolinguistic profile. The subtle differences are likely to be due to the different loci of pathology, the course of pathology, as well as to different, and possibly frontally mediated, adaptive-compensatory strategies.

REFERENCES

Alexander, M. P., & LoVerme, S. R. 1980. Aphasia after left hemispheric intracerebral hemorrhage. *Neurology*, **30**, 1193–1201.

Bayles, K. 1982. Language function in dementia. *Brain and Language*, **16**, 265–280.

Bayles, K. 1984. Language deficits in Huntington and Parkinson disease. In *Proceedings of the Academy of Aphasia*. Los Angeles.

Bayles, K., & Boone, D. R. 1982. The potential of language tasks for identifying senile dementia. *Journal of Speech and Hearing Disorders*, **47**, 210–217.

Bayles, K., & Tomoeda, C. K. 1983. Confrontation naming in dementia. *Brain and Language*, **19**, 98–114.

Bayles, K., Tomoeda, C. K., Kaszniak, A. W., Stern, L., & Eagans, K. 1985. Verbal perseveration of dementia patients. *Brain and Language*, **25**, 102–116.

Berman, M., & Peelle, L. M. 1967. Self-generated cues: A method for aiding aphasic-apractic patients. *Journal of Speech and Hearing Disorders*, **32**, 376–389.

Bowen, F. P., Kamienny, R. S., Burns, M. M., & Yahr, M. D. 1975. Parkinsonism: Effects of Levo-dopa treatment on concept formation. *Neurology*, **25**, 701–704.

Brown, R., & McNeill, D. 1966. The "tip of the tongue" phenomenon. *Journal of Verbal Learning and Verbal Behavior*, **5**, 325–337.

Cummings, J. L., & Benson, D. F. 1983. *Dementia: A clinical approach*. Cambridge: Butterworths.

Damasio, A. R., Damasio, H., Rizzo, M., Varney, N., & Gersh, F. 1982. Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. *Archives of Neurology*, **39**, 15–23.

Darley, F. L., Aronson, A. E., & Brown, J. R. 1975. *Motor speech disorders*. Philadelphia: Saunders.

Ford, M. 1978. *Planning units and syntax in sentence production*. Thesis, Cambridge: MIT Press.

Ford, M., & Holmes, V. 1978. Planning units and syntax in sentence production. *Cognition*, 6, 35–53.

Gentner, D. 1985. Some interesting differences between nouns and verbs. *Cognition and Brain Theory*, 4, 161–179.

Gordon, W. P. 1983. Memory disorders in aphasia: 1. Auditory immediate recall. *Neuropsychologia*, 4, 325–339.

Gordon, W. P. 1985a. Neuropsychological deficits in Huntington's Disease. In *International Neuropsychological Society*. San Diego.

Gordon, W. P. 1985b. Neuropsychological assessment of aphasia. In J. Darby (Ed.), *Speech evaluation in neurology* (pp. 161–196). New York: Grune & Stratton.

Gordon, W. P., & Illes, J. 1987. Neurolinguistic characteristics of language production in Huntington's Disease: A preliminary report. *Brain and Language*, 31, 1–10.

Hier, D. B., Hagenlocker, K., & Shindler, A. 1985. Language disintegration in dementia: Effects of ætiology and severity. *Brain and Language*, 25, 117–133.

Illes, J., Metter, E. J., Hanson, W. R., & Iritani, S. 1988. Language production in Parkinson's disease: Acoustic and neurolinguistic considerations. *Brain and Language*, 33, 146–160.

Illes, J., Nespoulous, J. L., & Lecours, A. R. 1986. Hesitation patterns in neologistic jargonaphasia: A longitudinal study. *La Linguistique*, 2, 74–95.

Joanette, Y., Keller, E., & Lecours, A. R. 1980. Sequence of phonemic approximations in aphasia. *Brain and Language*, 11, 30–44.

Marie, P. 1906. La troisième circonvolution frontale gauche ne joue aucun rôle dans la fonction du langage. *La Semaine Médicale*, 26, 241–247.

Metter, E. J., & Hanson, W. R. 1986. Clinical variability in patients with hypokinetic dysarthria. *Journal of Communication Disorders*, 19, 347–366.

Metter, E. J., Riege, W. H., Hanson, W. R., Lawrence, R., Camras, J., Phelps, M. E., & Kuhl, D. E. 1984. Correlations of glucose metabolism and structural damage to language function in aphasia. *Brain and Language*, 21, 187–207.

Naeser, M. A., Alexander, M. P., Helm-Estabrooks, N., Levine, L., Laughlin, S., & Greschwind, N. 1982. Aphasia with predominantly subcortical lesion sites: Description of three capsular/putamenal aphasia syndromes. *Archives of Neurology*, 39, 2–14.

Obler, L. K. 1980. Narrative discourse styles in the elderly. In L. K. Obler & M. L. Albert (Eds.), *Language communication in the elderly* (pp. 79–90). Lexington: Lexington Books.

Obler, L. K., & Albert, M. L. 1983. Language and aging: A neurobehavioral analysis. In D. S. Beasley & G. A. Davis (Eds.), *Aging: Communication processes and disorders* (pp. 107–121). New York: Grune & Stratton.

Obler, L. K., Mildworf, B., & Albert, M. L. 1977. Writing style in the elderly. In *Proceedings of the Academy of Aphasia*. Montreal.

Pirozzolo, F. J., Hansch, E. C., Mortimer, J. A., Webster, D. D., & Kuskowski, M. A. 1982. Dementia in Parkinson's disease: A neurophysiological analysis. *Brain and Cognition*, 1, 71–83.

Pribram, K. H. 1976. Language in a sociobiological frame. *Annals of the New York Academy of Sciences*, 280, 798–809.

Reisburg, B., Ferris, H., de Leon, M. J., & Crook, T. 1982. The Global Deterioration Scale (GDS): An instrument for assessment of Primary Degenerative Dementia. *American Journal of Psychiatry*, 139, 1136–1139.

Studdert-Kennedy, M. 1983. [Review of *Temporal variables in speech: Studies in honour of Frieda Goldman-Eisler*]. H. W. Dechert and M. Raupach (Eds.). The Hague: Mouton. Haskins Laboratory, Status report on speech research, SR-74/75.

Webster, D. D. 1978. *Webster rating scale, Parkinson's disease patient evaluation*. New York: Endo Laboratories.