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Evidence that Lexical Memory is Part of the Temporal Lobe
Declarative Memory, and that Grammatical Rules are
Processed by the Frontal/Basal-Ganglia Procedural Memory System

Running Page Heading: Declarative and Procedural Memory and Language

MICHAEL T. ULLMAN
Departments of Neuroscience, Linguistics, Psychology and Neurology
Georgetown University

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ABSTRACT

Language is thought to comprise a mental lexicon, in which words are stored, and a mental grammar, which can generate unlimited rule-governed forms. Evidence is presented that the lexicon is part of a temporal lobe “declarative memory” system, previously implicated in the memory for facts and events, and that grammatical rules are processed by a frontal/basal-ganglia “procedural” system, previously implicated in the processing of motor, perceptual and cognitive skills. Patients with relative damage to one or the other brain system were given a task in which the use of lexical memory and grammatical rule processing differed, while other factors were held constant: They were asked to produce the past tense of regular (*look-looked*) and novel (*plag-plagged*) verbs, which require a grammatical *-ed*-suffixation rule, and irregular verbs (*dig-dug*), which are retrieved from memory. The general declarative memory impairment in Alzheimer's disease, which is associated with relative damage to the temporal lobe, led to more errors with irregular than regular and novel verbs. The general impairment of procedures in Parkinson's disease, which is associated with relative damage to frontal cortex or the basal ganglia, led to more errors with regular and novel verbs than with irregular verbs. Moreover, basal ganglia lesions in Parkinson's disease and in Huntington's disease which lead to the suppression of movement also led to the suppression of rule use. In contrast, a different kind of basal ganglia lesion in Huntington's disease, which leads to excess movement, also led to excess rule use. This suggests that the well-studied basal ganglia neural circuits underlying motor programming might play a comparable role in rule programming.

INTRODUCTION

This paper addresses three questions in the study of the neural basis of language: First, how many brain systems underlie language, and what class of functions does each compute? Second, where are these systems in the brain? Third, what neural mechanisms underlie their computations? Evidence is presented that two major brain systems, whose functions, neuroanatomy and neural mechanisms have been well-studied, respectively subserve the two capacities that give human language its vast expressive power: a "mental lexicon" of memorized arbitrary information associated with words, including their sound patterns and their meanings; and a "mental grammar," whose generative rules combine words into an infinite number of larger words, phrases, and sentences (e.g., see Saussure, 1916; Chomsky, 1965; Pinker, 1994).

A large body of work has suggested a neural dissociation between lexical memory and grammatical processing. In particular, lexical memory has been linked with left temporal or temporo-parietal cortex, and grammatical processing with left frontal cortex (e.g., Wernicke, 1874; Geschwin, 1965; Damasio & Damasio, 1992; Goodglass, 1993; Dronkers & Pinker, 1995). However, this neural dissociation remains controversial (e.g., Bates & Wulfeck, 1989; Blackwell and Bates, 1995; Plaut, in press), partly because tasks probing for grammar and for memory have differed in ways other than their use of the two capacities. In this paper the dissociation between lexical memory and grammatical rule processing is confirmed with a simple language task in which the use of the two linguistic capacities is contrasted while other factors, such as complexity, meaning and task demands, are held constant.

This paper links the dissociation to larger principles of neural organization. Cutting across the division of the brain into systems such as language and visual perception, there is an orthogonal division into two major kinds of memory systems (Cohen & Squire, 1980; Mishkin, Malamut, & Bachevalier, 1984; Damasio & Damasio, 1992; Lieberman & Kako, 1992; Squire, 1993). One is a *declarative memory* system for facts, events, and arbitrary visual information, subserved by a medial-temporal circuit connected largely with temporal neocortex, with the medial-temporal components consolidating memories eventually stored in neocortex (Cohen & Squire, 1980; Mishkin, Malamut, & Bachevalier, 1984; Squire, 1993; Suzuki & Amaral, 1994). The other is a *procedural memory* system for the learning and processing of motor, perceptual and cognitive skills, subserved by basal ganglia circuits which project via the thalamus to frontal cortex (Cohen & Squire, 1980; Mishkin, Malamut, & Bachevalier, 1984; Heindel et al., 1988; Saint-Cyr, Taylor & Lang, 1988; Gabrieli et al., 1993; Squire, 1993).

The forms and meanings of words are like facts and events in being arbitrary and in possibly having storage sites in temporal regions. Therefore the temporal lobe declarative memory system may subserve words as well as facts and events. Grammatical rules are like skills in requiring the coordination of procedures in real time and in possibly having neural loci in frontal regions. Therefore the frontal/basal-ganglia procedural system may process grammatical rules as well as motor, perceptual, and cognitive skills.

In this paper the memory/rule dissociation is tested with a task based on a simple linguistic system in which reliance on grammar and lexicon differs, while other factors are held constant. Regular (*look-looked*) and irregular (*dig-dug*) past tense forms of verbs are well-matched in complexity (one word), syntax (tensed), and meaning (past). But regular verbs are predictable in form (verb stem + *-ed*), and new ones are constantly being added (*faxed, moshed*), whereas irregular verbs are unpredictable (compare *sing-sang, fling-flung, bring-brought*), and constitute a fixed list. A simple theory is that irregular forms are memorized, and regular forms are generated by a rule. Regulars and irregulars interact as follows: Retrieval of an irregular blocks the rule (*dug* pre-empts *digged*); when an irregular is not successfully retrieved, application of the rule may occur, resulting in "overregularization" errors such as *digged*.

Two alternatives to this memory/rule theory have been proposed, both inspired by the probabilistic patterns found among the irregulars (*ring-rang, sing-sang, drink-drank, sit-sat*). Both alternatives posit that irregular and regular verbs are computed in a single system, and that they differ only in number and in how consistently they follow a pattern. One posits rules for irregulars (e.g., "change *i* to *a*"), with memory compressed to the minimum information necessary (Chomsky & Halle, 1968; Halle & Mohanon, 1985). The other posits a connectionist pattern-associator memory, with rules eliminated, and both regular and irregular forms produced as generalizations from previously learned similar verbs (e.g., *sang* generalized from *sat* and *rang*, *stalked* generalized from *stopped* and *walked*) (Rumelhart & McClelland, 1986; Plunkett & Marchman, 1991).

Recent psycholinguistic evidence, however, has supported the memory/rule account (Pinker & Prince, 1988; Pinker, 1991). The irregular system is acquired early, is generalized rarely, requires words to be frequent or to come from families of similar forms, and behaves like stored roots in word-formation rules. The regular system is acquired later, is generalized freely even to rare, novel, and unusual words, and behaves as the "default" in word-formation, applying even to words that could not be stored as verb roots in memory, such as nouns (*high-sticked*) and onomatopoeia (*pinged*) (Kim et al., 1991; Marcus et al., 1992; Xu & Pinker, 1992; Prasada & Pinker, 1993; Ullman, 1993; Marcus et al., 1995). These phenomena occur in many languages, even those in which regular forms are in the minority (Marcus et al., 1995). This converging evidence suggests that irregulars are words stored in associative memory, and regulars are generally computed by a

grammatical rule It is therefore of interest to confirm this distinction in the form of a neural dissociation, and to use it to illuminate the functions of the major brain systems underlying language and their relation to overall brain organization.

If indeed irregulars are stored words, and the temporal lobe declarative memory system underlies lexical memory, and if regulars are rule-products, and the frontal/basal-ganglia procedural system underlies the processing of rules, then the following double dissociations are predicted. In patients with general impairments of declarative memory, and with damage to the temporal lobe but relative sparing of frontal cortex and the basal ganglia (e.g., in Alzheimer's disease), deficits of fact retrieval and word retrieval should be associated with deficits in the production irregular past tense forms (*dig-dug*), but not with deficits in the production of regular (*look-looked*) or novel (*plag-plagged*) past tense forms. Severe impairments of lexical memory associated with such damage (e.g., in Alzheimer's disease) should be accompanied by worse performance at irregulars than regulars, overregularization errors (*dig-digged*) where the irregular fails to be recalled and the rule is applied instead, and successful application of the rule to novel verbs. In contrast, in patients with general impairments of procedures, and damage to the frontal/basal-ganglia system but relative sparing of the temporal lobe (e.g., in Parkinson's disease), motor skill processing deficits should be associated with deficits in the production of regular and novel past tense forms, but not with deficits in the production of irregular pasts. Severe impairments of procedures or grammar associated with such damage (e.g., in Parkinson's disease) should lead to greater difficulty producing regular than irregular forms, an absence of overregularizations, and trouble inflecting novel verbs. In practice, this contrast might be relative rather than absolute, because in a single patient a lesion could strike both brain systems or a brain system common to the different verb types.

Language can be linked not only to the function and neuroanatomy of the two brain systems, but also to their neural mechanisms. Within the procedural system, the basal ganglia circuits that project to frontal cortex are parallel and structurally and functionally segregated: Each receives projections from a particular set of ipsilateral cortical and subcortical regions, and projects via the thalamus to a particular ipsilateral frontal lobe area. Thus the "motor" circuit projects to frontal motor areas, and subserves motor control, the "oculomotor" circuit projects to the frontal eye fields, and subserves eye movement, and the "prefrontal" circuit projects to prefrontal areas, and may subserve cognitive functions. These circuits are structurally segregated in that they have few connections between them, and are functionally segregated in that each subserves a different function. They are parallel in that they have similar synaptic organizations, paralleling each other throughout the basal ganglia's pathways. Thus within the basal ganglia each circuit splits into the "direct pathway," whose activation leads to excitation in frontal cortex, and the "indirect pathway,"

whose activation leads to inhibition in frontal cortex (Alexander, DeLong and Strick, 1986; Alexander, Crutcher, DeLong, 1990; Young and Penney, 1993; Middleton & Strick, 1994). Therefore "it would seem likely that similar neuronal operations are performed at comparable stages" in each of the circuits, and so "detailed knowledge of the workings of once circuit may prove useful in attempts to clarify another" (Alexander, DeLong and Strick, 1986 p. 361).

Basal ganglia circuitry may project to Broca's area (Hoover & Strick, 1993; Preuss, 1995), raising the possibility that the basal ganglia subserve grammatical processing. If basal-ganglia-thalamo-cortical circuitry subserving grammatical rules does exist, it should perform neural operations comparable to those done for motor programming, in which case basal ganglia lesions across circuits may similarly affect rule and motor programming. Thus the following associations are predicted. Basal ganglia lesions leading to excess inhibition in frontal cortex, and the suppression of movement (e.g., the hypokinesia found in Parkinson's and often in Huntington's disease), may also lead to the suppression of rule use. In contrast, basal ganglia lesions leading to excess excitation in frontal cortex, and unsuppressible movements (e.g., the hyperkinesia in Huntington's disease), may also lead to excess rule use.

METHOD

Past Tense Production Task

Materials

This task was given to all cognitively impaired populations described in this paper: patients with Alzheimer's, Parkinson's or Huntington's diseases, and posterior and anterior aphasics, as well as normal control subjects.

Verbs

Subjects were presented with 120 verbs from six classes: 20 *existing irregular* verbs, which take an irregular past tense (*dig-dug*); 20 *doublet* verbs, which can take both an irregular and a regular past tense (*dive-dove/dived*); 20 *existing regular* verbs, taking regular pasts (*look-looked*), and having stems *not* phonologically similar to the stems of irregulars; 20 *attracted regular* verbs, taking regular pasts, and having stems that *are* phonologically similar to the stems of irregulars (*glide-glided*, c.f. *hide-hid*, *ride-rode*); 20 *novel regular* verbs, made-up stems which are *not* phonologically similar to the stems of existing irregulars, and whose expected pasts were therefore regular (*plag-plagged*); and 20 *novel irregular* verbs, made-up stems which *are* phonologically

similar to the stems of existing irregulars, and whose possible pasts might therefore be irregular or regular (*crive-crove/crived*, c.f. *drive-drove*, *jive-jived*).

The verbs were selected according to 6 criteria. First, the existing verbs were chosen to cover relatively wide stem and past tense frequency ranges. Second, I avoided verbs which can play the role of auxiliary or modal (*do*, *be*, *have*). Third, I eliminated verbs which were judged to be possible denominals (derived from a noun), de-adjectivals (derived from an adjective), or verbs of onomatopoeic origin; the motivation for their elimination was that past tense forms of such derived verbs have been shown to be computed differently from other past tense forms (Kim et al., 1991). Fourth, an attempt was made to avoid existing or novel verbs whose stems or expected pasts were phonologically or orthographically identical or similar to other existing words. Thus I avoided *rend*, whose irregularized past *rent* exists as a distinct word; similarly, novel forms like *flam* or *shar* were rejected as being too similar to *flame* or *share*. Fifth, I attempted to avoid stems with ambiguous pronunciations; thus I excluded verbs like *blow*, whose orthography is similar to both *flow* and *allow*. I also avoided novel verbs like *palk*, which might be incorrectly pronounced with the "l" sound, rather than like *walk*. Sixth, all novel forms had to have acceptable English spellings. Thus forms such as *krog* and *krive* were forsaken in favor of *crog* and *crive*.

In addition to these general criteria, further specific criteria were applied to each of the six verb classes:

Existing irregulars. These are verbs for which *only* an irregular past is acceptable (*dig-dug*); thus doublet verbs like *dive-dove/dived* were excluded.¹ The 20 existing irregular verbs were *swim-swam*, *dig-dug*, *swing-swung*, *cling-clung*, *wring-wrung*,² *grind-ground*, *bend-bent*, *bite-bit*, *feed-fed*, *slit-slit*, *come-came*, *make-made*, *give-gave*, *think-thought*, *stand-stood*, *hit-hit*, *split-split*, *keep-kept*, *drive-drove*, *send-sent*. However, all analyses in this paper were carried out on a 16 verb subset, with *hit*, *split*, *slit* and *grind* excluded. The first three were omitted because their pasts cannot be distinguished from their stems. *Grind* was excluded because its past tense form (*ground*) also exists as the stem of another verb (*grind* was the only such verb in the list). The mean of the natural logarithms of each past tense frequencies (Francis and Kucera, 1982) for these 16 verbs was 3.7 (standard deviation of 1.9).³ According to the memory/rule theory, irregular past tense forms (e.g., *dug*) are expected to be stored in and retrieved from memory.

Doublets. These verbs have acceptable regular and irregular pasts (*dive-dove/dived*). Doublets were analysed separately from existing irregulars because their evidence suggests that their regular as well as irregular pasts appear to be stored in memory (Ullman, 1993). The 20 doublet verbs were *knit-knit/knitted*, *wed-wed/wedded*, *wet-wet/wetted*, *thrust-thrust/thrusted*, *light-lit/lighted*, *burn-burnt/burned*, *dwell-dwelt/dwelled*, *spill-spilt/spilled*, *kneel-knelt/kneeled*, *dream-dreamt/dreamed*, *creep-crept/creeped*, *leap-leapt/leaped*, *tread-trod/treaded*, *sneak-snuck/sneaked*, *spin-spun/*

spinned, slink-slunk/slunked, slay-slew/slayed, strive-strove/strived, dive-dove/dived, shine-shone/shined. All analyses in this paper excluded *knit, wed, wet* and *thrust* because their stems and pasts cannot be distinguished. The mean of the ln-transformed frequencies for these 16 verbs' irregular pasts is 0.8 (sd = 1.0), and 0.9 (sd = 1.1) for their regular pasts. According to the memory/rule theory, not only doublet irregular pasts (e.g., *dove*), but also doublet regular pasts (e.g., *dived*), are likely to be stored in and retrieved from memory.

Existing regulars. These monosyllabic verbs were selected for their stems' phonological similarity to the stems of other regulars, and dissimilarity to the stems of irregulars. To meet this second criterion I attempted to select regular verbs whose stems' vowels and final consonant clusters were not shared with those of irregular stems. The 20 existing regulars were *scowl, tug, flush, cram, mar, chop, flap, stalk, cook, scour, slam, cross, rush, shrug, rob, drop, look, walk, stir, soar*. The mean of the ln-transformed past tense frequencies for these 20 verbs was 2.0 (sd = 1.6). According to the memory/rule theory, regular past tense forms (e.g., *looked*) are likely to be rule produced.

Attracted regulars. These verbs' stems are phonologically similar to the stems of irregulars (*glide-glided*, c.f. *ride-rode, hide-hid*). They were treated separately from the (non-attracted) existing regulars described above because evidence suggests that such regular verbs, whose stems are similar to the stems of irregulars, are attracted into the associative memory underlying irregulars, and stored alongside these irregulars --- whence their designation "attracted regulars" (Ullman 1993). The 20 attracted regulars, together with their regular pasts and plausible irregularized pasts, were *swig-swig/swug/swigged, writhe-wrothe/writhed, sneeze-snoze/sneezed, quell-quelt/quelled, yearn-yearnt/yearned, grit-grit/gritted, strut-strut/strutted, squeak-squoze/squeaked, fend-fent/fended, wink-wunk/winked, earn-earnt/earned, swell-swelt/swold/swelled, squeeze-squoze/squeezed, deem-demt/deemed, trim-trum/trimmed, skid-skid/skidded, chide-chid/chode/chided, fret-fret/fretted, blend-blent/blended, glide-glid/glode/glided*. In this paper all analyses excluded *grit, strut, skid* and *fret* because their stems and likely irregularized pasts cannot be distinguished. The mean of the ln-transformed past tense frequencies for these 20 verbs was .9 (sd = .7). According to the memory/rule theory, attracted regular past tense forms (e.g., *glided*) are likely to be stored in and retrieved from memory.

Novel regulars. These novel verb stems were selected to be phonologically similar to the stems of existing regulars and dissimilar to the stems of irregulars; therefore their only likely pasts are regular. The 20 stems were *spuff, traff, dotch, stoff, cug, slub, trab, pob, plag, crog, vask, prass, brop, prap, satch, grush, plag, tunch, scur, scash*. According to the memory/rule theory, novel regular past tense forms (e.g., *plagged*) are rule produced.

Novel irregulars. These novel verb stems were selected to be phonologically similar to the stems of existing irregulars. The 20 stems, together with their regular pasts and some plausible irregularized pasts, were *strink-strank/strunk/strinked*, *frink-frank/frunk/frinked*, *strise-stris/strose/strised*, *treave-trove/treaved*, *crive-criv/crove/crived*, *shrell-shrelt/shrelled*, *vurn-vurnt/vurned*, *steeze-stoze/steezed*, *shrim-shram/shrum/shrimmed*, *trine-trin/trone/trined*, *preed-pred/preeded*, *cleed-cled/cleeded*, *scrit-scrit/scrat/scritted*, *ret-ret/retted*, *sheel-shelt/sheeled*, *blide-blid/blode/blided*, *cleep-clept/cleeped*, *prend-prent/preended*, *shreep-shrept/shreeped*, *drite-drit/drote/drited*. All analyses were performed on the 18 verb subset excluding *scrit* and *ret*, which were omitted because their likely irregularized pasts cannot be distinguished from their stems. The memory/rule theory might expect novel irregular pasts (*crive-crove*) to be computed across the same memorized associations as existing irregulars with similar stem-past mappings (*drive-drove*) (Bybee and Slobin, 1982; Bybee and Moder, 1983; Prasada and Pinker, 1993; Ullman, 1993); in contrast, regularizations of such verbs (e.g., *crived*), are likely to be rule produced.

Sentences

Each verb stem was presented in a "stem sentence", which was followed by a "past tense sentence" prompting for a past tense form. For example:

Every day I *look* at Susan.

Just like every day, yesterday I _____ at Susan.

All sentences were written to conform to three criteria, whose purposes were to ensure consistency among the items and to facilitate the task for language-impaired subjects. First, every stem and past sentence were in the completive aspect. Second, every stem sentence began with "Every day I," and every past sentence began with "Just like every day, yesterday I." Third, the verb was always followed by a two word argument or adjunct, each word being of high frequency, few syllables, and uninflected. Moreover, for novel verbs these words were selected to minimize the possibility of conjuring up a meaning for the novel form from an existing similar-sounding verb; thus I avoided "Every day I *broop* a penny" to prevent confusion with *drop*.

Procedure

The items were first randomized by computer program (Perlman, 1986), and then gone over by hand to ensure that similar-sounding verb forms did not follow each other too closely. All subjects received items in the same order; this was done for testing convenience.

Each subject was tested separately. The subject was first given several practice items, for which he or she was asked to read each stem and past tense sentence out loud, filling in the missing word in the past tense sentences. Each sentence pair was printed on a single sheet of paper in large font.

If the subject misread the verb stem, he or she was stopped and asked to read it again. If reading was laborious, both sentences were read by the experimenters, with appropriate intonation to elicit a response for the missing word. All sessions were audio-taped. During the testing of each subject, the experimenters wrote down all responses for each verb item. If any response was unclear, or if the experimenters disagreed about a response, the tape was played back until a consensus was reached. Special attention was paid to weak final consonants such as the final [t] in *looked* and *kept*. An item was counted as correct if it elicited only correct responses. Thus any incorrect responses, without, before, or after a correct response, resulted in an incorrect score for that item. For doublet and novel irregular verbs (*dive, crive*), there is no single correct past tense; therefore regular pasts (*dive, crived*) were counted as correct if they were produced at any point before re-prompting, and analogously with irregular pasts (*dove, crove*). Similarly, unmarked forms (e.g., *look-look, keep-keep*) were counted if they were produced before re-prompting. Over-regularizations (*digged*), as well as multiply- or syllabically-suffixed forms (*plaggeded, plag-id*) were counted even if they were produced after any re-prompting, to increase the sample size, because they were relatively rare. These scoring criteria were used for all cognitively-impaired populations and for the normal control subjects.

Object Naming Task

To measure lexical memory, subjects were asked to name 84 line-drawings of objects from the Boston Naming Test (Goodglass, Kaplan and Weintraub, 1983), presented one at a time. Subjects were allowed several attempts on each item. Responses were scored as correct if the object was correctly named during any attempt, as long as the next item had not yet been presented. No phonemic or semantic prompting was given. A score of 100% indicates that all items were named correctly.

Dementia Task: IMC

To estimate the severity of any dementia, subjects were given the Information, Memory and Concentration (IMC) subtest of the Blessed Dementia Scale (Blessed, Tomlinson, & Roth, 1968). The IMC dementia test has five subsections: (1) The subject is asked for general information, such as his or her name and the current season; (2) tests for "personal memory", such as date of birth; (3) requests "non-personal memories", such as the name of the current president and a date during World War II; (4) is a 5 minute delay recall test of a fictitious person and his address; (5) asks the subject to list the months backwards and count forwards and backwards between 1 and 20. Higher IMC scores indicate greater dementia; a score of 0 indicates an absence of dementia.

Fact Retrieval

Memory for facts and events was measured with the remote memory subsection of the IMC dementia test --- that is, the second and third subsections in which subjects are asked questions about personal and non-personal facts and events. Higher scores correspond to more errors.

EXPERIMENT 1: PATIENTS WITH ALZHEIMER'S DISEASE

Patients with probable Alzheimer's disease (AD)⁴ are impaired at learning new words, facts, events, and visual information, as well as recalling and recognizing old ones (Lawson and Barker, 1968; Bayles, 1982; Corkin, 1982; Muscovitch, 1982; Hier et al., 1985; Nicholas et al., 1985; Huff et al., 1986; Kempler, Curtiss and Jackson, 1987; Rissenberg and Glanzer, 1987; Beatty et al., 1994; Huff et al., 1988; Sagar et al., 1988; Sahakian et al., 1988; Dall'Ora et al., 1989; Kopelman 1989; Nebes, 1989; Carlessimo and Oscar-Berman, 1992; Price et al., 1993).

In contrast, AD patients (ADs) appear to be relatively spared at learning new perceptual, motor and cognitive skills (Eslinger and Damasio, 1986; Heindel, Butters, & Salmon, 1988; Heindel, Salmon et al., 1989; Knopman, 1991; Knopman and Nissen, 1987; Bondi and Kaszniak, 1991; Grosse et al., 1991; Heindel et al., 1991; Deweer et al., 1993; Gabrieli et al., 1993), and at processing motor and cognitive skills learned before disease onset (Cummings and Zarit, 1987; Beatty et al., 1988; Crystal, Grober and Masur, 1989; Beatty et al., 1994).

Moreover, evidence suggests that ADs may have relatively spared syntax, morphology, and phonology, even in the face of lexical and conceptual deficits. This has been shown in English in spontaneous speech (Kempler, Curtiss & Jackson, 1987; Hier, Hagenlocker & Shindler, 1985; Bayles, 1982; Appell, Kertesz and Fisman, 1982; Nicholas, Obler, Albert, & Helm-Estabrooks, 1985; Murdoch, Chenery, Wilks and Boyle, 1987; Price et al., 1993), elicited sentence production (Schwartz, Marin, & Saffran, 1979), sentence comprehension (Schwartz, Marin, & Saffran, 1979; Kempler et al., 1987), and identification or correction of errors (Cushman and Caine, 1987; Whitaker, 1976). Similar contrasts have also been shown in French (Irigaray, 1973; see Obler, 1981).

These behavioral dissociations may stem from neuroanatomical dissociations. In AD, medial temporal structures tend to have higher densities of neurofibrillary tangles (NFTs) than any other area, while high-order temporal areas have higher densities than many other neocortical areas, including Broca's area (Brodmann's areas 44 and 45), which tends to be relatively spared, as do the basal ganglia and the cerebellum (Kemper, 1984, 1994; Arnold et al., 1991; Dustin, Brion & Flament-Durand, 1992). Similarly, Brun and Gustafson (1976) found that medial temporal and temporo-parietal areas suffered the greatest cell loss in AD. A number of PET studies of regional

brain metabolism in AD suggest that hypometabolism is worse and more frequent in the temporal and parietal than in the frontal lobes, and that the basal ganglia and cerebellum are relative spared (see Rapoport, 1991; Grady and Rapoport, 1992). Note that NFT densities and hypometabolism, unlike densities of neuritic plaques, are good predictors of cognitive decline (Wilcock & Esiri, 1982; Rapoport, 1991).

If temporal neocortex underlies the storage of words as well as facts, but is not involved in processing grammatical rules, we should find correlations between difficulties remembering facts and difficulties remembering words, and each of these with difficulties producing irregularized past tense forms, but not with difficulties producing regularized pasts. In addition, AD patients with severe lexical memory impairments should be worse at producing irregular than rule-generated past tense forms.

Subjects

The tasks were given to 24 patients diagnosed with probable AD lacking complications, according to the guidelines of the National Institute of Aging (NIA) and the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) (Khachaturian, 1985; McKhann, Drachman, Folstein, Katzman, & Price, 1984). All patients were Native speakers of standard American English; 21 were monolingual, 3 bilingual;⁵ 16 were female, 8 male; 22 were right-handed, 2 ambidextrous. The 24 patients had a mean age of 73 years, and a mean of 15 years of education.

Results and Discussion

Across the 24 AD patients, object naming difficulties correlated with difficulties producing existing (*dug*) and novel (*crove*) irregular pasts, but not existing (*looked*) or novel (*plagged*) regular pasts, or regularizations of novel irregulars (*crived*) (with irregularizations partialled out) (Table 1, a). This pattern also held when IMC dementia scores were partialled out (Table 1, b), suggesting that the correlation pattern cannot be explained by the patients' dementia. It might be argued that the lack of a correlation between object naming and past tense production of regulars was due to the high production rate of existing regulars, resulting in a relative lack of variance (92% correct, $sd=12\%$, vs 84%, $sd=16\%$ for existing irregulars). However, the correlation pattern held even when the past tense production variable was rank-ordered (Table 1, c); moreover, irregulars were produced no less successfully, and with smaller standard deviations, than novel regulars (*plagged*) and regularizations of novel irregulars (*crived*) (respectively, 86% %, $sd=18\%$ and 75%, $sd=21\%$), which showed the same pattern as existing regulars (*looked*). Object naming

difficulties correlated negatively with overregularization rates (*digged*) ($r(22)=-.58$ $p=.003$), even with IMC scores partialled out ($r(21)=-.45$ $p=.029$). This supports the hypothesis that overregularizations are rule-produced upon the failure to retrieve their corresponding irregulars (*dug*) from memory (Marcus et al. 1992).

Fact retrieval difficulties correlated with object naming difficulties ($r(22) = -.61$, $p < .001$), and also with difficulties producing irregular pasts, but not with difficulties producing any of the three regularized pasts (*looked*, *plagged*, *crived*) (Table 1, d). This correlation pattern remained with IMC dementia scores (excluding the IMC fact retrieval component) partialled out (Table 1, e), even when the past tense variable was rank ordered (Table 1, f). Similarly, fact retrieval difficulties correlated negatively with overregularization rates ($r(22)=.57$ $p=.004$), even with IMC scores (excluding fact retrieval) partialled out ($r(21)=.45$ $p=.030$). Surprisingly, the correlation between fact retrieval difficulties and difficulties producing novel irregularized pasts (*crove*) was either not significant (Table 1, d and e) or marginally significant (Table 1, f); this suggests that there are few neural substrates in common between fact retrieval and the production of novel irregulars.

If words and facts are stored in temporal neocortex rather than in medial temporal structures, we might expect that performance at the acquisition of new facts, which is known to depend upon medial temporal structures, should not correlate with performance at the production of irregulars. Indeed, the ADs' performance at the 5-minute recall test in the IMC, in which subjects are asked to recall a fictitious name and address, did not correlate with performance at producing irregular past tense forms (or with performance at any other of the past tense types; see Table 1, g); this was even more striking when performance at fact retrieval was partialled out ($r(21)= -.05$ $p=.809$). In contrast, fact retrieval performance still correlated with performance producing irregular pasts, even with performance at 5-minute recall partialled out ($r(21)= -.55$ $p=.007$). This contrast also held with Spearman rank correlations ($r(21)= -.20$ $p=.371$ vs $r(21)= -.45$ $p=.032$).

If one set of neural structures subserves the regular rule, while another set underlies the production of existing and novel irregular pasts, then the ADs' production rates of the three types of regularized pasts should be inter-correlated, as should their production rates of existing and novel irregulars, while correlations between performance at regular and irregular past types should not be significant. As shown in the upper section of Table 1, difficulties producing existing regulars (*looked*), novel regulars (*plagged*), and regularizations of novel irregulars (*crived*) were highly inter-correlated ($p < .001$ in all three cases), but were not correlated with success at existing or novel irregulars ($p > .25$ in four cases, and a borderline significant correlation between novel regulars and novel irregular pasts, which may be due to their shared novelty). Moreover, the significant correlations among the regular past types occurred despite the existing regular's high success rate, whose resulting ceiling effects should serve to mask such correlations. The correlation between the

production rates for existing and novel irregular past tense forms barely approached significance; this is consistent with the hypothesis that they share only some underlying mechanisms.

If irregular but not regularized past tense forms are lexicalized, and if the neural systems underlying lexical memory are lesioned in AD, while those subserving grammatical rules are relatively spared, then those AD patients with the greatest impairments of lexical memory should be worse at producing irregular than regularized past tense forms.

AD symptoms are variable. The 5 ADs with the worst object naming scores were therefore selected for comparison with age- and education-matched control subjects. These 5 anomic ADs were all right-handed; 4 were native speakers of American English, 1 was bilingual; 4 were female, 1 male. The 5 had a mean age of 74, and a mean 14 years of education. Their scores were compared with those of 14 normal control subjects, who had a mean age of 74 and a mean of 16 years of education; 11 were native speakers of American English, 3 bilingual; 12 were right-handed, 2 ambidextrous; 9 were female, 5 male. There was no statistically significant difference between the 5 anomic ADs' and the 14 control subjects' ages ($t(17)=.32$ $p=.751$) or years of education ($t(17)=1.64$ $p=.119$).

To determine whether the 5 anomic ADs were more impaired at the production of irregular pasts (*dug*) than regular pasts (*looked*), compared to controls, a mixed between-subject (AD and control groups) and within-subject (regular and irregular verb classes) analysis of variance (ANOVA) was carried out. This revealed a statistically significant interaction between group and verb class ($F(1,17)=20.45$ $p<.001$), as did analogous mixed analyses of covariance (ANCOVAs) with age ($F(1,16)=24.03$ $p<.001$), education ($F(1,16)=16.33$ $p<.001$) or IMC dementia scores ($F(1,16)=14.14$ $p=.002$) held constant. Following up on the ANOVA with *t*-tests showed that regular pasts were produced statistically significantly more successfully than irregular pasts by the ADs (paired $t(4)=8.33$ $p=.036$ over subjects, indep. measures $t(34)=4.86$ $p<.001$ over items), but not by the control subjects (paired $t(13)=1.29$ $p=.221$ over subjects, indep. measures $t(34)=1.22$ $p=.229$ over items). (see Table 2 for means). The ADs' superior performance on the regular verbs could not be due to any frequency advantage because the irregular items had higher past tense frequencies. Neither does it appear to be caused by an averaging artifact: 4 of the 5 ADs had higher regular than irregular scores (mean advantage of 38 percentage points); all 4 individual subject independent measures *t*-tests over items were statistically significant. In contrast, the remaining subject (EF) was only 6 percentage points more successful at the irregulars than on the regulars, a difference which was not statistically significant. (see Table 2 for individual subject analyses).

The 5 ADs produced significantly more overregularizations (*digged*) than their control subjects, (independent measures ($t(17)=6.45$ $p<.001$), as well as more overregularizations as a proportion of their irregulars errors (independent measures $t(10)=2.46$ $p=.034$; analysis carried out on only

those control subjects who made at least one irregular error). This further supports the hypothesis that overregularizations are produced upon failure to retrieve the stored irregular past tense form.

Novel regular pasts (*plagged*) have not been heard before, and therefore could not be memorized, and must be created anew. According to the memory/rule theory, they must be generated by the application of the rule to the stem; therefore the anomic ADs may be better at producing the pasts of novel regulars than of existing irregulars, despite the conceptual difficulties associated with carrying out the task with novel verbs. A mixed between-subject (the 4 anomic ADs able to carry out the production task with novel verbs, and the 14 control subjects) and within-subject (novel regular and existing irregular verb classes) ANOVA revealed a statistically significant interaction between subject group and verb class ($F(1,16)=7.22$ $p=.016$), as did analogous ANCOVAs holding constant age ($F(1,15)=9.26$ $p=.008$), education ($F(1,15)=5.82$ $p=.029$), or IMC dementia scores ($F(1,15)=11.72$ $p=.004$).

Follow-up *t*-tests showed that the ADs were more successful at producing past tense forms for novel regulars than for existing irregulars (indep. measures $t(7)=2.15$ $p=.069$ over subjects, indep. measures $t(34)=2.80$ $p=.008$ over items), while their control subjects showed the *opposite* tendency (nonsignificantly: paired $t(13)=1.57$ $p=.140$ over subjects; indep. measures $t(34)=1.19$ $p=.243$ over items). Three of the four AD subjects were worse at producing pasts for existing irregulars than novel regulars (mean difference of 18 percentage points), while the fourth subject showed the opposite pattern (21 percentage point advantage for the irregulars)⁶ (see Table 2).

If novel irregularizations (*crive-crove*) are associatively computed alongside existing irregulars (Bybee and Slobin, 1982; Bybee and Moder, 1983; Prasada and Pinker, 1993; Ullman, 1993), their production should be impaired among anomic ADs. In contrast, regularizations of these same verbs (*crive-crived*) should be produced with fewer difficulties if they are rule-products. Although in a mixed between-subject (the 4 anomic ADs able to perform the task on novel verbs, and the 14 controls) and within-subject (irregularizations and regularizations of novel irregulars) ANOVA the interaction between group and past type was not statistically significant ($F(1,16)=1.19$ $p=.292$), the ADs produced one fourth as many irregularizations (*crive-crove*) as their controls ($t(16)=1.72$ $p=.105$), but a slightly, though non-significantly, larger number of regularizations (*crived*) ($t(16)=.44$ $p=.670$). Thus the anomic ADs appeared to have trouble computing novel irregular forms, resorting to the regularized form instead.

Interestingly, rule-generated forms appeared to have been produced with no impairment: Despite the conceptual difficulty of performing the task with novel verbs, the 5 ADs were not significantly worse than their control subjects at producing novel regular pasts (*plagged*), and produced more regularized pasts of novel irregulars (*crived*) than the controls. They also produced more overregularizations as a percentage of irregular errors than their control subjects, with is

consistent with *facilitated* rule use among the ADs, compared to controls. Similarly, once general intellectual deterioration was adjusted for, the ADs were *better* than their controls at producing regularized pasts. In an ANCOVA between the 5 anomic ADs and their controls, holding IMC dementia scores constant, the ADs' IMC-adjusted mean for the production of regular pasts (*looked*) was actually 20 percentage points *higher* than that of their control subjects 0% for ADs, 90% for controls ($F(1,16)=6.71, p=.020$), while the ADs' *unadjusted* mean was 9 percentage point below that of the controls. A similar though non-significant pattern held for the analogous ANCOVA for novel verbs: For novel regulars (*plugged*) the ADs' IMC-adjusted mean had a 17 point advantage ($F(1,15)=1.30, p=.273$), 4% for ADs, 87% for controls versus a 9 point disadvantage with the unadjusted means, and for regularizations of novel regulars (*crived*) the ADs' IMC-adjusted mean had a 33 point advantage ($F(1,15)=.75, p=.399$). 3% for ADs, 60% for controls Thus once there has been an adjustment of the ADs' general intellectual impairment, which may have given them trouble understanding or paying attention to the task, we find that they are better than their control subjects at producing regularized past tense forms, as if rule production had been *facilitated*.

Summary and Conclusion

Difficulties with object naming and with fact retrieval were correlated with each other. Each of these was correlated with difficulties producing existing irregular (*dug*), and, to a lesser extent, novel irregular (*crove*) past tense forms, and, negatively, with overregularization rates (*digged*). In contrast, neither difficulties with object naming nor with fact retrieval were correlated with difficulties producing any of the three types of regular past tense forms (*looked, plugged, crived*). This contrast held even when past tense performance was rank ordered, or when IMC dementia scores or performance at 5-minute fact recall were partialled out. Production rates of the three types of regular pasts were highly inter-correlated, but were not correlated with production rates of existing or novel irregular pasts. The 5 most anomic ADs (those with the worst performance at object naming) had more trouble producing irregularized (*dug, crove*) than regularized (*looked, plugged, crived, digged*) past tense forms, which appear to have been produced without impairment, and possibly with facilitation.

These results suggest that the pasts of existing irregulars (*dug*) are stored in and retrieved from lexical memory, which is part of the same neural system as the memory for facts and events. The production of novel irregular pasts (*crove*) also appears to depend partly on neural structures underlying lexical memory, and to a lesser extent, on those underlying the memory for facts and events. In contrast, the results suggest that production of the three types of regular pasts (*looked, plugged, crived*) is not subserved by the neural system subserving lexical and fact-event memory.

The results therefore support the memory/rule hypothesis, with a brain system that is damaged in AD subserving the memory of facts and words, but not the processing of grammatical rules. The damage to temporal cortex in AD, and relative sparing of frontal cortex and the basal ganglia, support the view that temporal cortex underlies the memory for facts and words, as part of the declarative memory system, but not grammatical processing, which may be subserved by the frontal/basal-ganglia procedural system.

Further Discussion

Alzheimer's disease and the study of the neural bases of grammar

This study supports previous claims that grammatical processing is relatively spared in AD. In contrast, deficits in AD have been reported not only for lexical memory and conceptual information, but also for discourse (Bayles, 1982). Thus within language, the *sparing* of grammar in AD appears to be relatively selective, as opposed to the relatively selective *impairment* of grammar in anterior aphasia (Broca's aphasia). This contrast may provide a useful construct for the study of language, particularly of grammar. The selective sparing of a domain such as grammar may elucidate it in ways that its selective impairment could not. While selective impairment can reveal which neural structures are *necessary* for a computation, selective sparing can reveal those that are *sufficient*. Thus by studying ADs as well as anterior aphasics, we may be able to determine which neural structures are both necessary and sufficient for grammatical processing. Moreover, selective sparing may avoid certain problematic interpretations of selective impairment, such as possible lesion side effects (i.e., damage to one set of neural structures resulting in the malfunction of another set through their connectivity).

Given that grammatical processing is relatively spared in AD, it seems likely that brain areas which are heavily lesioned in the disease are not necessary for grammatical rule processing. Thus the relatively high densities of neurofibrillary tangles in area 22, and very high densities in area 38 (Arnold et al., 1991), may cause problems for claims that the anterior portion of area 22 (Dronkers et al., 1994), or the left temporal pole in general (Mazoyer et al., 1993), might play a role in grammatical processing. Similarly, Arnold et al. (1991) found very high NFT densities in anterior insular cortex, suggesting that this region may also not be necessary for grammatical rule processing.

Dissociations in irregular and regular spelling-to-sound transformations

This study has shown a dissociation in AD between the impaired production of irregular past tense forms, and the relatively spared production of regular past tense forms. A similar dissociation has also been shown in a non-grammatical domain. Studies have revealed that Alzheimer's disease is associated with relatively intact reading and writing of regular spelling-to-sound transformations

(forms with predictable pronunciations in English, such as *mint*, or the novel form *rint*), while irregular spelling-to-sound transformations are problematic (forms with unpredictable pronunciations, such as *pint*, or *yacht*)).

Balota and Ferraro (1993) found a frequency by regularity interaction for reading, with irregular spelling-to-sound transformations showing a greater disadvantage for low frequency than high frequency forms, compared to regulars. This is consistent with the view that while irregularly spelled forms are lexicalized, regularly spelled forms are not. Similarly, Schwartz, Marin and Saffran (1979) found that a subject with degenerative dementia showed few problems with regular words or high-frequency irregular words; low-frequency irregular words were not tested. In contrast, three anterior-lesioned patients to whom they gave the task (including VS and HT, performed at chance at the regularly spelled words. Warrington (1975) tested three patients with diffuse cerebral lesions of unknown origin (possibly Alzheimer's disease), two of whom showed signs of increased pathology in left temporal, temporo-parietal, or adjacent posterior regions. These patients had difficulty reading irregular words, even those of high frequency, while their reading of regular words was relatively spared. An analogous regular-irregular contrast was found in *writing* to dictation (Rapcsak et al., 1989), wherein AD patients were better at spelling regular than irregular words.

Such dissociations, respectively termed "surface dyslexia" and "surface agraphia" for reading and writing, are also found in patients with lesions (e.g., from strokes) in left temporal areas and adjacent posterior regions (e.g., Coltheart et al., 1983). This pattern contrasts with that of many patients with lesions in left anterior areas, who are often worse at reading or writing regular than irregular spelled forms ("phonological dyslexia" and "phonological agraphia"). Lesions in temporal and adjacent posterior areas are also associated with greater difficulties producing irregular than regular past tense forms, whereas anterior lesions are associated with the opposite pattern (see Ullman et al., in press).

These parallel double dissociations between the domains of spelling and inflectional morphology suggest a neural commonality between regular spelling-to-sound transformations and regular inflectional morphology, and likewise for irregulars. The apparent relative sparing of the learning and processing of motor, perceptual and cognitive skills in AD, in the face of the impaired learning, recall and recognition of words, facts, events, and arbitrary visual information, suggests that regular transformations in the spelling-to-sound and inflectional morphology domains involve the procedural system, while irregulars in both domains are learned and stored in declarative memory. It is important to point out that I am *not* claiming that representations of all types of arbitrary information are randomly distributed across temporal or temporo-parietal cortex. Rather I am suggesting that many different types of arbitrary or unpredictable information, including

information about facts and events, conceptual information, visual information, unpredictable word forms such as irregular pasts and words with irregular spelling-to-sound transformations, are subserved by the declarative memory system. Such memories are presumably consolidated in medial temporal structures, and eventually stored in cortex. However, representations sharing certain features, or learned in a particular modality, might very well be clustered together, such that a lesion might affect representations of one type of information more than others. Indeed, there have been a number of case studies of patients with temporal lobe pathology who have category-specific deficits, even limited to a particular modality (e.g., Warrington and Shallice, 1984; McCarthy and Warrington, 1988; Hart and Gordon, 1992; see Rapp and Caramazza, 1995. for a discussion). Crucially, in this paper I am emphasizing the neural commonality, rather than distinctiveness, of representations of these different types of information, which I suggest can be attributed to their common dependence upon declarative memory.

EXPERIMENT 2: PATIENTS WITH PARKINSON'S DISEASE

Parkinson's disease (PD) is associated with severe degeneration of dopaminergic neurons in the substantia nigra pars compacta of the basal ganglia, while in non-demented PD patients temporal and parietal areas are relatively spared of pathology (Dubois et al., 1991). This basal ganglia degeneration results in the inhibition of frontal cortical areas to which the basal-ganglia-thalamic circuits project, which appears to explain why PD patients (PDs) have difficulty initiating and carrying out movement (hypokinesia) (Young and Penney, 1993). The basal ganglia degeneration might also account for findings suggesting impairments in the learning of new motor (Harrington et al., 1990; Ferraro et al., 1993; Jackson et al., 1995), perceptual (Bondi and Kaszniak, 1991; Allain, Lieury, Quemener, et al., 1995), and possibly cognitive (St. Cyr et al., 1988; Allain, Lieury, Quemener, et al., 1995) skills. However, such deficits in skill learning have not always been found, and the issue remains controversial (Heindel et al., 1989; Harrington et al., 1990; Dubois et al., 1991; Allain, Lieury, Thomas, et al., 1995).

Grammatical processing may also be impaired. Several studies have found impaired sentence comprehension among Parkinson's patients, with syntactically more complex sentences yielding more errors (Lieberman et al., 1990; Lieberman et al.; 1992, Grossman et al., 1991; Grossman et al., 1992; Natsopoulos et al., 1991). However, it is unclear whether impairments of working memory or other non-linguistic functions could account for these results. Illes et al. (1988) and Illes (1989) reported that the spontaneous speech of moderate PDs had lower syntactic complexity

than the speech of mild PDs, as measured by a syntactic complexity metric developed by the authors and based on lexical functional grammar. They also reported a significantly higher ratio of open class words (e.g., nouns and verbs) to function words (e.g., determiners and prepositions) for PD patients than controls.

In contrast, learning words, facts, events, and visual information is relatively spared, particularly in non-demented PDs, and especially when the information must be recognized rather than recalled (Lees and Smith, 1983; Flowers, Pearce & Taylor et al., 1986; St. Cyr et al., 1988; see Dubois et al., 1991 for a review). Similarly, in non-demented PDs, words, facts, events, and visual information learned before onset of the disease (remote memory) are relatively spared for recognition, and to a lesser extent for recall (Warburton, 1967; Matison et al., 1982; Freedman et al., 1984; Globus et al., 1985; Huber et al., 1986; Sagar et al., 1988; Levin et al., 1989; Bayles 1990).

If the basal ganglia underlie grammatical rule processing as well as motor programming, the suppression of movement (hypokinesia) in PD should be associated with the suppression of rule use. If the basal ganglia are less important in word-finding, such patients may be relatively spared at the production of irregulars. Thus if the left basal ganglia project not only to left cortical motor areas, which subserve right-side movement, but also to left frontal areas subserving grammatical processing (see above), right-side hypokinesia should be associated with the suppression of rule use, and therefore with difficulty producing past tense rule-products (*looked, plagged, crived, digged*), but perhaps not with difficulty producing irregular pasts (*dug, crove*).

Subjects

The tasks were given to 38 subjects who were diagnosed by their primary neurologist as having idiopathic Parkinson's disease; each diagnosis was confirmed by neurologists from the Movement Disorders Unit at Massachusetts General Hospital. Analyses were carried out only on the 28 patients who learned standard American English as a primary language before the age of 5 (i.e., first language or bilingual), who were not severely demented (IMC score ≤ 5), and who had no other medical complications. One additional patient was excluded from analyses because he was severely *hyperkinetic*, presumably from levodopa medication. Of the 28 patients, 25 were first language speakers, and 3 bilingual; 6 were female, 22 male; 26 were right-handed, 2 ambidextrous. The mean age of the 28 patients was 69, with a mean of 15 years of education. 23 of the patients were receiving levodopa with a peripheral decarboxylase inhibitor (Sinemet).

In addition to the past tense production, object naming, and IMC dementia tasks, all PD patients were given a test measuring the severity of their hypokinesia (akinesia and bradykinesia). This

consisted of four hand and foot movement tests taken from the Unified Parkinson's Disease Rating Scale (UPDRS) (Fahn et al. 1987). Left and right limb movements were scored separately. The four tests were conducted while the patient was sitting down and facing the experimenter: (1) In Finger Taps, the patient taps the thumb with the index finger in rapid succession, with the widest amplitude possible. (2) In Hand Movements, the patient opens and closes the hand in rapid succession with the widest amplitude possible. (3) In Rapid Alternating Movements of Hands (also called Pronation-Supination Movements of Hands), the patient turns both hands palms-up and then palms-down, as quickly and with as large an amplitude as possible. (4) In Leg Agility, the patient bends the knee and taps the heel on the ground in rapid succession, picking up the entire leg about three inches off the ground with each tap. In evaluating a patient's performance on these tasks, several factors are taken into consideration: the number of repetitions performed, their amplitude, hesitations in movement, and fatigue.

Results and Discussion

Across the 28 PD patients, right-side hypokinesia correlated with difficulties producing the three types of regularized pasts (*looked, plagged, crived*), but not with difficulties producing the two types of irregular pasts (*dug, crove*) (Table 3, a). This pattern held even with IMC dementia scores partialled out (Table 3, b). Although a similar pattern was observed for the analogous correlations with left-side motor impairment (Table 3, c and d), the correlation coefficients for the three regular past types were each smaller (less negative) for these left-side correlations than for the analogous right-side correlations, both in the surface correlations, and with IMC scores were partialled out. Paired *t*-tests suggested these differences were not due to chance alone ($t(2)=3.46$ $p=.074$, $t(2)=3.93$ $p=.059$). In contrast, this difference did not hold with the two irregular past types ($p > .4$ in both cases, with left-side hypokinesia actually a slightly better predictor).

More importantly, the superiority of right-side hypokinesia in predicting difficulties with regularized pasts was confirmed in partial correlations: With left-side hypokinesia partialled out, right-side hypokinesia correlated with difficulties producing existing regulars (statistically significant) and novel regulars (borderline statistically significant), but not with difficulties producing existing or novel irregular pasts (Table 3, e). In contrast, left-side hypokinesia was not predictive of the difficulty producing any of the past tense types, once right-side hypokinesia was partialled out (Table 3, f).

If the left basal ganglia play a role in rule programming as well as motor programming, but are less important in the production of irregular past tense forms, then right-side hypokinesia should

correlate with difficulties producing regularized forms, but not irregular forms, even when object naming scores are partialled out. Indeed, this is what we found (Table 3, g). In contrast, if irregular forms are retrieved from (*dug*) or computed in (*crove*) lexical memory, while regularized forms are not, object naming should correlate with difficulties producing irregular but not regularized forms, when right-side hypokinesia is held partialled out. Indeed, object naming was weakly predictive of irregularized pasts, but not at all of regular pasts (Table 3, h).

The memory/rule theory is further supported by the finding (see upper section in Table 3) that difficulties producing the three types of regular pasts (*looked*, *plugged*, *crived*) were strongly inter-correlated ($p < .01$ for all three correlations), while the correlations between difficulties producing these regular pasts and the pasts of existing or novel irregulars were not significant ($p > .3$ in four cases, and borderline significant for the correlation between existing regulars and irregulars, which may indicate that existing regulars are sometimes memorized).

If the left basal ganglia underlie the processing of both right-side motor skills and grammatical rules, but are less important in the retrieval and storage of words, then PD patients with severe right-side hypokinesia should be worse at producing rule-generated than stored past tense forms.

PD symptoms are variable. The 5 PDs with the worst right-side hypokinesia were therefore selected for comparison with age- and education-matched controls. These 5 hypokinetic PDs were all right-handed native speakers of American English; 1 was female, 4 male. Their mean age was 72, with a mean of 16 years of education. The performance of the 5 PDs was compared with that of the same 14 controls with which the ADs were also compared. There was no statistically significant difference between the PDs' and controls' age (independent measures $t(17)=1.10$ $p=.286$) or years of education ($t(17)=.38$ $p=.706$).

To determine whether the 5 PDs with the most severe right-side hypokinesia were more impaired at the production of regulars (*looked*) than irregulars (*dug*), compared to controls, we carried out a mixed between-subject (the 5 PDs and their control subjects) and within-subject (existing regular and irregular verb classes) ANOVA. This revealed a statistically significant interaction between group and verb class ($F(1,17)=7.65$ $p=.013$), as did analogous mixed ANCOVAs holding constant age ($F(1,16)=6.09$ $p=.025$), education ($F(1,16)=7.72$ $p=.013$), or IMC dementia scores ($F(1,16)=5.82$ $p=.028$). The PDs were marginally better at producing past tense forms for irregular than regular verbs (paired $t(4)=1.98$ $p=.118$ over subjects, indep. measures $t(34)=1.34$ $p=.190$ over items), while the controls showed the opposite pattern (paired $t(13)=1.29$ $p=.221$ over subjects, indep. measures $t(34)=1.22$ $p=.229$ over items). Four of the five PDs had higher scores on regular than irregular verbs (mean advantage of 11 percentage points), while the remaining subject was 6 percentage points better at irregulars (Table 4).

The 5 PDs produced no overregularizations, despite their opportunity to do so, with a mean of 12% of their irregular items yielding errors. In contrast, the control subjects overregularized 14% of their irregular errors, even though they made fewer irregular errors than the PDs ($t(17)=2.03$ $p=.059$), and thus had less opportunity to overregularize.

Novel regular pasts (*plagged*) have not been heard before, and therefore could not have been memorized, but must rather be created anew. The memory/rule theory predicts such forms are rule-products, and therefore the hypokinetic PDs should be impaired at producing them. A mixed between-subject (PD and control groups) and within-subject (novel regulars and existing irregular verb classes) ANOVA revealed a statistically significant interaction between subject group and verb class ($F(1,17)=21.03$ $p<.001$), as did analogous ANCOVAs holding constant age ($F(1,16)=21.73$ $p<.001$), education ($F(1,16)=20.28$ $p<.001$) or IMC dementia scores ($F(1,16)=20.10$ $p<.001$). Follow up t -tests showed that the PDs were less successful at novel regulars than existing irregulars (paired $t(4)=10.47$ $p<.001$ over subjects, indep. measures $t(34)=3.49$ $p=.001$ over items), while this difference was not significant for the controls, despite their larger subject sample size (paired $t(13)=1.57$ $p=.140$ over subjects, indep. measures $t(34)=1.19$ $p=.243$ over items). All 5 PDs showed this superiority at irregulars, thereby precluding an averaging artifact (Table 4).

Interestingly, the 5 PDs were also more successful at producing existing regulars (*looked*) than novel regulars (*plagged*). There was a significant interaction between group and verb class ($F(1,17)=6.05$ $p=.025$), and the difference between the existing and novel regular success rates was significantly greater for the PDs (mean difference=1 $t(17)=2.46$ $p=.025$). This is not surprising, given that existing regular past tense forms have been heard before, and therefore have a non-zero probability of having been memorized, while novel regular past tense forms could not have been memorized, and therefore their production should be more susceptible to suppression of the rule.

If regularizations (*crive-crived*) of novel irregulars are also rule-generated, while their irregularizations (*crive-crove*) are associatively computed alongside existing irregulars (Bybee and Slobin, 1982; Bybee and Moder, 1983; Prasada and Pinker, 1993; Ullman, 1993), the hypokinetic PDs should have more difficulty than their controls at producing regularizations, but perhaps not irregularizations. A mixed between-subject (PDs and controls) and within-subject (irregularizations and regularizations of novel irregulars) ANOVA revealed the suggestion of an interaction between group and past type ($F(1,17)=2.77$ $p=.114$), as did ANCOVAs holding age ($F(1,16)=2.77$ $p=.115$) or IMC dementia scores constant, ($F(1,16)=3.29$ $p=.089$), while the interaction from the ANCOVA holding education constant was statistically significant ($F(1,16)=5.47$ $p=.033$). Follow-up independent measures t -tests revealed that the PDs were indeed less successful than the controls at

producing regularizations (*crived*), ($t(17)=2.67$ $p=.016$), while there was a non-significant trend in the opposite direction for irregularizations ($t(17)=-.66$ $p=.516$). This contrast shows that the PDs' difficulty with regularizations of novel verbs cannot be fully explained by a general impairment at processing novel verbs.

Arguments against Alternative Explanations

Although the findings suggest that neural damage in PD results in greater difficulties with rule-generation than with the production of irregularized forms, at least four alternative explanations might account for the results.

First, could frequency effects explain the findings? The 5 hypokinetic PDs' relative impairment at producing pasts for the existing regulars could be explained by the fact that the past tense frequencies of those items were on average lower than those of the irregular items. Thus if both verb types were stored in memory, regulars would be retrieved less successfully. However, such frequency effects could not account for the PDs' lack of overregularizations (*digged*) or relative difficulty with novel regulars (*plagged*, *crived*). It is also not clear how frequency effects could account for the correlation patterns: the inter-correlations among regularized past tense types, but not between regularized and irregularized types, as well as the contrasting correlations between production of regular and irregular forms with hypokinesia and object naming. In addition, as we will see below, the most hypokinetic PDs were also given a new past tense production task in which the regular and irregular items were closely matched on past tense frequency as well as articulatory difficulty; again, the subjects were worse at producing regular than irregular past tense forms. Finally, "attracted regular" pasts (*glided*), which independent evidence suggests can be stored alongside irregulars (Ullman, 1993), and which had *lower* frequencies than the irregular or existing regular pasts in our task, yielded analyses unlike the non-attracted existing regulars (*looked*): The attracted regular pasts correlated with object naming, but not with hypokinesia, and were not produced less successfully than the irregular pasts (*dug*).

Second, although PDs have visual difficulties (Dubois et al., 1991), such impairments cannot explain the results. Given the nature of the task, in which all verbs are presented in their stem forms, the stems of existing or novel regulars should not be harder to read or process than the stems of existing or novel irregulars. Moreover, the novel irregular verbs (*crive*) yielded a relative deficit in the production of regularized pasts (*crived*), compared to irregularized pasts (*crove*), even though they shared the same stems. In any case, subjects were asked to read the stem sentence again if any error was made in reading the verb stem.

Third, PDs might make more errors on the production of *-ed*-suffixed past tense forms if they produce only as much phonological material as is necessary to form a real word. Such a tendency might arise, for example, from articulatory difficulties (see immediately below), which might encourage the patient to attempt to produce a form that was easier to pronounce than the past tense form, but was still a real word. Because regular pasts are phonologically composed of their stems plus the suffix (*look* + *-ed*), while few irregular pasts contain initial phonological material that sounds like a real English word (e.g., *dug*), such a tendency should yield more errors for regular than irregular verbs. However, past tense forms for attracted (*glided*) and doublet (*dived*) verbs, which also contain their stems, yet which previous evidence suggests can be stored alongside irregulars (Ullman, 1993), were not produced significantly less successfully than irregulars. The contrast between these results and the impaired production of non-attracted existing regulars (*looked*) can be better explained by a rule impairment than by the production of embedded words.

A fourth alternative explanation is a motor or articulatory deficit. The impairments of mouth movements or of articulation found in PD (Ramig and Gould, 1986) might result in greater difficulty pronouncing the final consonant clusters of regular than irregular pasts. All regular forms have a minimum of one final consonant (the alveolar stop in the /ed/ suffix), which is appended to any stem-final consonants (e.g., *looked*, *chopped*). In contrast, irregular pasts often have no more final consonants than their stems (e.g., *dig-dug*). Indeed, the past tense forms of all the existing regular items in the past tense production task, and all but two of the novel regulars, had a two-consonant final cluster; the remaining two novel regulars were even more complex (*vasked*, *tunched*). In contrast, 13 of the 16 existing irregular past tense forms had 1 final consonant, and the remaining 3 verbs had two (*bent*, *sent*, *kept*).

Four arguments against an *articulatory deficit account* are presented:

Pattern of Errors

Articulatory difficulties might yield unmarked forms as errors (*look-look*, *plag-plag*) because omitting the final alveolar stop (the *-ed*) should simplify articulation. Indeed, for the 5 hypokinetic PDs, unmarked forms consisted of more than half (53%) of the errors for existing regulars. % of the errors for existing regulars. If articulatory problems result in such final-consonant omissions, we might expect analogous errors for similar-sounding irregulars (*keep-kep*). However, this did not occur. All 28 PDs produced *kept*, without a single utterance of *kep*, even though the experimenters paid special attention to the production of forms with weak or absent final alveolar stops. Similarly, among the novel irregulars, there were no instances of *cleep-clep*, *shreep-shrep*, or *sheel-shel*, even though 14 of the 28 PDs produced *clept*, 9 uttered *shrept*, and 3 said *shelt*.

Regulars and Irregulars Matched on Past Tense Pronounceability

Although the lack of forms like *kep* suggests that the PDs' difficulties with regulars cannot be explained by articulatory difficulties, there were far fewer irregular than regular double-consonant-final pasts, and thus the lack of forms like *kep* might result from the scarcity of such verbs. To test the articulation deficit hypothesis directly, a new verb list was created: 21 pairs of regular and irregular verbs, with the regulars and irregulars matched one-to-one on the word-final pronounceability of their past tense forms (*passed-los*). These verbs were presented to 4 of the 5 most hypokinetic PDs from the first testing session (the fifth was in a nursing home and was unavailable for testing) in a new past tense production task like the original one (Every day I *lose* a quarter. Just like every day, yesterday I _____ a quarter.)

The 21 irregular-regular past tense pairs were *sent-gained, spent-planned, lent-dined, lost-passed, dealt-sailed, felt-failed, meant-joined, kept-stopped, slept-slipped, sold-rolled, told-called, bound-frowned, found-owned, held-pulled, heard-stirred, made-played, rode-showed, wrote-tried, ate-stayed, built-ruled, left-lived*. New sentences were written for each verb, with the same criteria as in the original task. The irregulars were statistically significantly more frequent (mean ln-transformed frequency of 4.05) than the regulars (3.51) (paired $t(20)=2.20$ $p=.040$) (Francis & Kucera, 1982).

For each existing regular verb in the original past tense production task (20 verbs) and in the new past tense production task (21 verbs), an uninflected word was selected, closely matched one-to-one on word-final pronounceability and frequency (e.g., *scowled-scald*). These 41 uninflected words were presented one-to-a-page to the same 4 hypokinetic PDs. The forms were read out loud by the experimenter, and the subjects were asked to repeat the form out loud. Thus the subjects were able to both hear and read the forms, minimizing potential aural comprehension or reading problems.

The uninflected words were monomorphemic where possible; in no case was the final alveolar stop a separate morpheme. There was also an attempt to avoid words which contained a phonological equivalent of a high-frequency word (e.g., *duct, c.f. duck; pact, c.f. pack*). The 20 uninflected words matched to the 20 regular verbs in the original production task are listed here together with their matched regular past tense forms: *scowled-scald, tugged-sect, flushed-crust, crammed-tempt, marred-shard, chopped-opt, flapped crypt, stalked-react, cooked-eject, scoured-curd, slammed-prompt, crossed-frost, rushed-trust, shrugged-strict, robbed-erupt, dropped-adopt, looked-fact, walked-act, stirred-lard, soared-sword*. The uninflected words (mean ln-transformed frequency = 1.9) were not statistically significantly lower in frequency than their matched regular past tense forms (2.0) (paired $t(19)=.782$ $p=.444$). The 21 uninflected words matched to the 21 regulars in the new production task were *gained-saint, planned-brand, dined-*

grind, passed-fast, sailed-shield, failed-yield, joined-faint, stopped-apt, slipped-script, rolled-mold, called-cold, frowned-mound, owned-fond, pulled-build, stirred-bird, played-aid, showed-code, tried-pride, stayed-shade, ruled-fold, lived-gift. The uninflected words (ln-transformed frequency = 3.3) were not statistically significantly lower in frequency than their matched past tense forms (3.6) (paired $t(20)=1.41$ $p=.174$).

The 4 hypokinetic PDs were more successful at producing past tense forms for the irregular verbs (mean of 8 (paired $t(3)=1.85$ $p=.161$, over subjects; paired $t(20)=3.01$ $p=.007$, over items). All four subjects produced more irregular than regular pasts, although, not surprisingly, in the 2 patients with less severe hypokinesia, the difference was small (see Table 5). These results indicate that articulatory difficulties do not fully explain the PDs' relative deficit at producing regular past tense forms, and suggest a rule impairment.

The 4 PDs were also better at uttering the 41 uninflected words (9 past tense forms from the two production tasks (paired $t(3)=1.8$ $p=.168$, over subjects; paired $t(40)=7.0$ $p<.001$), over items). As shown in Table 5, all four subjects showed this pattern; only for the 2 patients with more severe hypokinesia was the difference statistically significant. Similar results were obtained with analogous individual and group paired t -tests comparing each of the 4 subjects' performance at producing the 20 regular pasts in the first production task with their success at repeating their 20 matched uninflected forms, and analogously for the 21 regulars in the second task with their repetition of the 21 matched uninflected forms. These results again argue against a solely articulatory deficit account.

To further test the hypothesis that the PDs were better at producing irregular than regular past tense forms because the former had higher frequencies, we analysed the 4 retested hypokinetic PDs' past tense production performance at a subset of verb pairs in which the regular verbs had a *higher* ln-transformed past tense frequency (1.54) than the irregular verbs (1.38) (Francis & Kucera, 1982). These six irregular-regular verb pairs were thus simultaneously equated on pronounceability and past tense frequency: *lost-passed, slept-slipped, sold-rolled, bound-frowned, rode-showed, ate-stayed*. The PDs produced past tense forms more successfully for the irregular verbs (9 (paired $t(3)=2.83$ $p=.066$, over subjects; paired $t(5)=3.2$ $p=.025$, over items), with all 4 subjects showing this pattern (Table 5). These results indicate that the PDs' superior performance at producing irregulars was not explainable by frequency factors alone, and provide further support against the articulatory deficit account.

Stored Regular Past Tense Forms are Like Irregular Past Tense Forms

Unlike the existing and novel regular forms examined thus far, certain regular past tense forms, with specific characteristics, are likely to be learned in and retrieved from lexical memory (Ullman,

1993). If a rule impairment explains the results discussed thus far, past tense forms of irregulars and of these memorized regulars should yield similar patterns in analyses, in contrast to the differing patterns we have observed between irregulars and the rule produced regulars such as *looked* and *plugged*. However, if the PDs' observed problems can be explained by articulatory difficulties, the irregulars and memorized regulars should yield the same differences as those between irregulars and rule produced regulars.

As described above, regulars whose stems are phonologically identical or similar to the stems of irregulars, such as doublet regulars (*dive-dived/dove*) or "attracted regulars" (*glide-glided*, c.f. *hide, ride*), appear to be attracted into associative memory, and memorized alongside those irregulars (Ullman, 1993). All 28 PDs and control subjects were tested on the past tense production of doublet and attracted regular verbs.

If the PD impairment is rule based, then the production of stored forms like *glided* or *dived* should be like the production of irregulars (*dug*) in correlating with object naming difficulties, but not with hypokinesia. In contrast, if the PD impairment is primarily articulatory in nature, the correlation pattern of these stored regular pasts should be similar to that of existing non-attracted regulars (*looked*) and novel regulars (*plugged*), correlating with hypokinesia, but not with object naming difficulties. The 28 PDs' difficulties producing attracted regulars (*glided*) correlated with object naming difficulties, partialing out right-side hypokinesia ($r(25) = .61$ $p < .001$), but not with right-side hypokinesia, partialing out object naming difficulties ($r(25) = -.06$ $p = .774$). Similarly, the 28 PD patients' difficulties producing doublet regulars (*dived*) correlated with object naming difficulties, with right-side hypokinesia and difficulties producing doublet irregulars (*dove*) partialled out ($F(1,24) = .52$ $p = .007$), while they did not correlate with hypokinesia, with the other two variables partialled out ($F(1,24) = .18$ $p = .374$). As expected, difficulties producing doublet irregulars (*dive-dove*) also correlated with object naming difficulties, partialing out right-side hypokinesia ($r(25) = .51$ $p = .007$), but not with right-side hypokinesia, partialing out object naming difficulties ($r(25) = .26$ $p = .184$).

To test the hypothesis that the PDs were not more impaired at irregular than attracted regular pasts, a mixed between-subject (the 5 most hypokinetic PDs and their control subjects) and within-subject (attracted regular and existing irregular verb classes) ANOVA was carried out. There was no statistically significant interaction between group and verb class ($F(1,17) = .15$ $p = .707$), and similarly for analogous ANCOVAs holding constant age ($F(1,16) = .78$ $p = .391$), education ($F(1,16) = .11$ $p = .750$) or IMC dementia scores ($F(1,16) = .96$ $p = .341$). Follow-up paired *t*-tests showed that the 5 PDs were not statistically significantly worse at producing past tense forms for attracted regulars (*glided*) (8) (*dug*) (8) while this difference was borderline significant for the control subjects (9). In contrast, as we have seen above, a different pattern held for non-attracted

existing regulars such as *looked*: The PDs were worse at producing past tense forms for these regular verbs than for irregulars, while their controls showed the opposite pattern. Interestingly, the mean ln-transformed past tense frequency of the 16 attracted regulars' past tense forms (*glided*) (mean of 1.0) was lower (indep. measures $t(34)=2.36$ $p=.024$) than that of the non-attracted regulars (*looked*) (mean of 2.0), let alone of the irregulars (*dug*) (mean of 3.7). This further supports the claim that the PDs' problems with non-attracted regulars (*looked*) is not explained by their having lower past tense frequencies than the irregular items.

Similarly, in a mixed between-subject (5 PDs and 14 controls) and within-subject (production of doublet regular pasts (*dived*) and doublet irregular pasts (*dove*)) ANOVA, there was no statistically significant interaction between group and verb class ($F(1,17)=.03$ $p=.859$), and similarly for analogous ANCOVAs holding constant age ($F(1,16)=.07$ $p=.789$), education ($F(1,16)=.03$ $p=.864$) or IMC dementia scores ($F(1,16)=.50$ $p=.489$). The PDs were not significantly worse than their controls at producing either doublet regulars (*dived*) (2) or doublet irregulars (*dove*) (5). In contrast, we have seen borderline or statistically significant interactions from analogous ANOVAs, ANCOVAs, and t -tests comparing the 5 PDs' and controls' regularization (*crived*) and irregularization (*crove*) rates for novel irregulars. These results are problematic for an articulatory deficit account, which predicts similar past tense production patterns for *dived* and *crived*, while they support a rule impairment.

Thus attracted and doublet regular pasts (*glided*, *dived*), which previous evidence had suggested are likely to be retrieved from memory rather than being rule-created (Ullman, 1993), had past tense production patterns similar to irregulars (*dug*), and different from existing non-attracted regulars (*looked*) and novel regularized forms (*plagged*, *crived*). This suggests a PD rule impairment, but is inconsistent with an articulatory deficit account. The low mean past tense frequency of attracted regulars also suggests that frequency differences cannot explain the PDs' impairments with non-attracted regulars (*looked*). Finally, the results bolster the claim that doublet and attracted regulars are stored in and retrieved from lexical memory (Ullman, 1993).

Summary and Conclusion

Across the 28 PDs, right-side hypokinesia correlated with difficulties producing different types of rule-generated (*looked*, *plagged*, *crived*), but not irregular (*dug*, *crove*) past tense forms, even when IMC dementia scores were partialled out. This pattern of correlations with right-side hypokinesia remained when left-side hypokinesia was partialled out, but none of the correlations of past-tense performance with left-side hypokinesia were significant with right-side hypokinesia partialled out. Right-side hypokinesia correlated with difficulties producing rule-generated pasts but

not irregular pasts with object naming scores partialled out, while object naming difficulties correlated, albeit weakly, with difficulties producing irregular, but not rule-generated, pasts, with right-side hypokinesia partialled out. Production rates of the different types of rule-generated pasts were highly inter-correlated, but did not correlate with production rates of existing or novel irregulars. The 5 most hypokinetic PDs had greater difficulty producing rule-generated pasts (*looked, plagged, crived*) than irregularized pasts (*dug, crove*), and did not overregularize (*digged*). The 5 anomic ADs and the posterior aphasic showed the opposite pattern.

Evidence was presented against four alternative accounts for these findings: frequency effects, visual difficulties, a tendency to produce embedded words, and an articulatory deficit. Three lines of evidence were presented against the possibility that an articulatory deficit could fully explain the data. First, the 5 hypokinetic PD subjects did not produce the same types of phonological errors with irregulars (*kept-kep*) as they did with regulars (*look-look*). Second, in a new set of regular and irregular verbs, matched one-to-one on pronounceability and frequency (*passed-lost*), the retested hypokinetic PD subjects were worse at producing the regular than irregular past tense forms; moreover, these PDs were also worse at producing regular pasts than at repeating pronounceability- and frequency-matched uninflected words (*passed vs fast*). Third, two types of regular past tense forms that are retrieved from memory (attracted and doublet regulars like *glided* and *dived*) were not produced less successfully than irregulars, and showed a correlation pattern similar to that of the irregulars (*dug, crove*), not the non-attracted or novel regulars (*looked, plagged, crived*).

These results suggest that in PD, degeneration of the nigro-striatal dopaminergic neurons in the left basal ganglia leads not only to the suppression of motor programming, but also the suppression of grammatical rule programming. However, our results are also consistent with rule programming being subserved by left frontal areas, without involvement of the basal ganglia: If basal-ganglia-thalamo-cortical circuits do indeed project to frontal cortex, the nigro-striatal degeneration may lead to cortical dysfunction, causing the observed rule deficits. Degeneration of the dopaminergic neurons projecting from the ventral tegmental area to frontal cortex seems to be a less likely explanation: The apparent selective influence of the left, but not right, basal ganglia, and the lack of correlations between difficulties with object naming and rule-generation suggest that it is the specific neural structures whose degeneration leads to right-side hypokinesia (the nigro-striatal neurons in the left basal ganglia) which also lead to rule impairments. This also argues against implication of brain areas other than the frontal lobes or the basal ganglia, particularly of temporal and parietal areas, which moreover are relatively spared of pathology in non-demented PD patients (Dubois et al., 1991), such as those that we tested.

While the findings do not support a role of for the basal ganglia in lexical retrieval or storage, they do not preclude such a role either. Rule programming, with its requirement for coordination in real time, may be more susceptible than lexical operations to disruption caused by the PD degeneration. This possibility is not precluded by the double dissociations between PD and AD/posterior aphasia: While the temporal lobe system might subserve the lexicon, but not grammar, the frontal/basal-ganglia system might subserve both, perhaps with a different role than the temporal lobe system (e.g., retrieval vs storage).

EXPERIMENT 3: PATIENTS WITH HUNTINGTON'S DISEASE

In PD patients, we found that the suppression of motor programming, caused by basal ganglia degeneration leading to the inhibition of frontal motor regions, was associated with suppression of rule programming. A complementary demonstration of a role for the basal ganglia in rule programming comes from Huntington's disease (HD). Like PD, HD is associated with the loss of neurons in the basal ganglia, although in the neostriatum (caudate nucleus and putamen) rather than in the nigro-striatal projections as in PD. This HD degeneration is often in projections to the inhibitory "indirect" pathway, resulting in excess excitation in motor and other frontal cortical areas receiving basal ganglia-thalamic projections (Reiner et al., 1988). This is thought to explain why HD patients have unsuppressible movements (chorea, a type of *hyperkinesia*) (Young and Penney, 1993). Interestingly and surprisingly, the neostriatal cell loss is not restricted to projections to the inhibitory indirect pathway. It also occurs in projections to the excitatory "direct" pathway, resulting in excess inhibition in motor and other frontal cortical areas receiving basal ganglia projections. This may explain why HD patients can also have *hypokinesia* (bradykinesia) *co-existing* with their chorea (Young and Penney, 1993). The neostriatal degeneration also may account for findings suggesting that HD patients have difficulty learning new motor, perceptual and cognitive skills (Harrington et al., 1990; Heindel, Butters, & Salmon, 1988; Heindel, et. al, 1989; Knopman and Nissen, 1991; Martone et al., 1984; Butters et al., 1985; St. Cyr et al., 1988), while the learning of new words, facts and arbitrary visual information remains relatively intact (Heindel, Butters, & Salmon, 1988; Martone et al., 1984; Butters et al., 1985; St. Cyr et al., 1988).

If basal-ganglia-thalamo-cortical circuitry does indeed subserve grammatical rule programming, and if the neural operations underlying rule programming are indeed similar to those underlying motor programming, as might be expected if they have similar synaptic organizations (Alexander, DeLong and Strick, 1986; Alexander, Crutcher, DeLong, 1990), then in HD, excess movement

(chorea) should be associated with excess rule use, while the suppression of movement (hypokinesia) should be associated with the suppression of rule use.

Method

Subjects

The tasks were given to 18 subjects with Huntington's disease. Their diagnosis criteria were positive family history and clinical symptoms. One of the 18 was excluded from analyses because his severe dysarthria prevented understanding of his responses, in particular of his word endings. The 17 remaining subjects were all first language speakers of English; 13 were right handed, 2 left-handed, 2 ambidextrous; 5 were female, 12 male. The 17 subjects had a mean age of 45 and a mean of 14 years of education (Table 6). One subject was taking a dopamine agonist --- levodopa with a peripheral decarboxylase inhibitor (Sinemet); four were taking dopamine blockers --- two Haloperidol (Haldol), and two Perphenazine (Trilafon); two were taking a GABA-minergic agonist --- Clonazepam (Klonopin); no subjects were taking dopamine depleters (e.g., Reserpine) or cholinergic agonists (e.g., Physostigmine or Deanol). The HDs' performance was compared with that of 8 age- and education-similar normal control subjects (Table 6); these were the same control subjects compared with the posterior aphasic JLU.

Materials and Procedure

HD and control subjects were given the past tense production, object naming, and IMC dementia tests. In addition, each HD subject was given a number of subtests from the Unified Huntington's Disease Rating Scale (UHDRS) (The Huntington Study Group, in press). The UHDRS includes a number of assessments of specific motor impairments, including numerical measures of chorea (hyperkinesia) and bradykinesia (hypokinesia). For the assessment of chorea, each of 7 body areas (e.g., face, trunk, right upper extremities, left upper extremities, etc.) is subjectively assigned a numerical measure of "maximal chorea", which ranges from 0 (no chorea) through 4 ("marked/prolonged"); all 17 HDs had chorea scores for different body areas between 0 and 3. For a given patient, these chorea values for each of the seven body parts are summed, for a maximum total chorea score of 28 (4*7); among the 17 HDs, total chorea scores ranged from 2 to 21. The numerical measure for bradykinesia is also subjectively assigned, and ranges from a description of an absence of bradykinesia (score of 0) to one involving movements that are "markedly slow, [with] long delays in initiation" (score of 4). The scores for the 17 patients ranged from 0 to 3 ("moderately slow, some hesitation"). The 17 HDs were also assessed on three other subtests of the UHDRS: the Independence Scale and the Shoulson Total Functional Capacity

(TFC), both subjective measures of the subject's ability to live independently, and the Physical Disability, a subjective measure of general motor disability.

Results and Discussion

The HD patients inflected irregular, regular and novel verbs at similar success rates (see Table 6). There were no significant differences between the production of past tense forms for existing regular and irregular verbs (*looked* vs *dug*; paired $t(16)=.67$ $p=.513$), existing regular and novel regular verbs (*looked* vs *plagged*; $t(30)=.89$ $p=.378$; independent measures t -test comparing performance between existing regulars for all 17 HD patients and novel regulars for the 15 HDs able to carry out the task for novel verbs), or existing irregular and novel regular verbs (*dug* vs *plagged*; $t(30)=.51$ $p=.611$). However, the types of errors made by the HD patients revealed their underlying deficits.

Excess Movement (Hyperkinesia) Associated with Excess Rule Use

The HD patients made two unusual errors, producing forms with multiple (*plaggeded*) or syllabic (*plagg-id*) suffixes, particularly for the regular and novel verbs (Table 6). Many forms were produced that were both multiply- and syllabically-suffixed (e.g., *look-ided*, *prapp-ided*, *shrug-ided*, *plagged-id*); these were counted as multiply-suffixed in the percentages. Multiply-suffixed forms with more than two suffixes were also occasionally encountered (e.g., *droppededed*, *choppededed*). The 17 HD patients produced multiply- and syllabically-suffixed forms for regular and novel verbs at a rate of 6.6% (includes cutter and leotta, at 0 each - thus over all 17 HDs (errors made by 9 patients; one outlier at 7 in contrast to 0.2 (Mann-Whitney $U_c(1) = 3.64$, $p = .056$; nonparametric test used because of the outlier), 1). Across the HD patients, the chorea measure (hyperkinesia) correlated significantly with the rate of producing multiply- or syllabically-suffixed forms ($r_r(15) = .57$, $p = .018$; Spearman rank correlation used because of the outlier), even when IMC dementia scores were partialled out ($r_r(14) = .54$, $p = .032$).

Irregular past forms ending with *t* or *d* (e.g., *kept*, *bent*) elicited no perseverations of the final consonant (e.g., *keptet*, *bentet*, *bitit*, 0 out of 170 opportunities), and only one syllabic pronunciation (*kep-it*, 1 out of 51 opportunities), suggesting that the errors with regular and novel verbs could not be explained by motor perseverations of *-t* or *-d* or other motor problems. Moreover, if motor perseverations explained the production of multiply- or syllabically-suffixed forms, the HD patients should also have produced forms like *look-it* or *lookedet*, with the syllabic suffix or one of the multiple suffixes containing a *t* rather than a *d*. However, no such forms were produced; rather all syllabic suffixes, and all secondary or tertiary suffixes in multiply-suffixed forms, were *-d*-final. In contrast, as described above, the one apparently syllabically-suffixed irregular form (*kep-it*) was produced with a *t*, not a *d* as in *kep-id* or *look-id*. Finally, multiply- and syllabically-suffixed

forms were produced for verbs whose phonology required each of the three allomorphs ([t], [d], [ed]) (e.g., *tuncheded*, *sheeleded*, *prendeded*, and *grush-id*, *plag-id*, *preed-id*), suggesting the commonality of an underlying rule.

The 17 HDs also produced many overregularizations (*digged*) (mean 8 while their control subjects produced none ($t(23) = 2.73, p = .012$). Moreover, the chorea measure correlated, approaching significance, with the HD rate of producing overregularizations ($r(15) = .42, p = .094$), even when IMC dementia scores were partialled out ($r(14) = .48, p = .062$).

Although both the AD and HD patients overregularized, they are predicted to do so for different reasons: deficient word-finding for AD, but overactive rules for HD. Unlike the AD patients, the HD patients' object naming scores were close to those of their control subjects (compare Tables 9 and 2). Indeed, the 17 HDs overregularized at the same rate as the full group of 24 ADs (8 but were significantly better at object naming (74 More importantly, across all 17 HD patients, naming did not correlate with the overregularization rate ($r(15) = -.24, p = .356$), unlike the 24 AD patients (see above). In contrast, the overregularization rate correlated with chorea, partialing out object naming (approaching significance) ($r(14) = .46, p = .072$), but not with object naming, partialing out chorea ($r(14) = -.32, p = .229$). Because the *-ed* in overregularization errors (*digged*) is not present in correct irregular forms (*dug*), such errors cannot be attributed to motor perseverations of the *-t* or *-d*, further underscoring a role for the basal ganglia in rule programming.

The HD patients also occasionally produced suffixed irregular *pasts* (*dugged*): 1 and all control subjects ($t(55) = 2.88, p = .006$). With the two types of overregularizations combined (*digged* and *dugged*), the following statistically significant analyses were obtained: 9 versus 0% for their control subjects ($t(23) = 2.65, p = .014$); the production of these forms correlated with the chorea measure ($r(15) = .47, p = .055$; with IMC scores partialled out, ($r(14) = .52, p = .040$), but not with object naming ($r(15) = -.20, p = .447$). The production rate of such forms correlated with chorea, partialing out object naming ($r(14) = .51, p = .044$), but not with object naming, partialing out chorea ($r(14) = -.29, p = .283$).

The HD patients also occasionally produced multiply- and syllabically-suffixed forms for irregular verbs (see Table 6), while their control subjects produced none. 10 yielded either overregularizations (*digged*), suffixed irregular *pasts* (*dugged*), multiply-suffixed forms (*diggeded*), or syllabically-suffixed forms (*digg-id*). Across the 17 HD patients, the production of all suffixed irregulars (*digged/dugged/diggeded/digg-id*) correlated with chorea ($r(15) = .46, p = .065$; with IMC scores partialled out ($r(14) = .50, p = .047$), but not with object naming ($r(15) = -.09, p = .736$). Likewise, the irregular suffixation measure correlated with chorea, partialing out object naming ($r(14) = .47, p = .065$), but not with object naming, partialing out chorea ($r(14) = -.16, p = .560$).

A number of other measures of disease progression or of behavioral or motor impairments, including hypokinesia, correlate neither with chorea, nor with the rate of producing multiply- and syllabically-suffixed forms for regular and novel verbs, nor with the overregularization rate, nor with rate of suffixing irregulars (Table 7). This suggests that it is the specific kind of lesion leading to chorea that also leads to overactive rule use.

Suppressed Movement (Hypokinesia) Associated with Suppressed Rule Use

If in HD the suppression of movement is associated with the suppression of rule use, we might expect hypokinesia to be associated with omission of the *-ed* suffix, resulting in the production of unmarked forms like *look* and *plag*. Indeed, the 17 HDs produced more unmarked forms than controls for existing regulars (*look*) ($t(23)=2.39$ $p=.026$) (mean 9 errors made by 11 of the 17 patients, range 5-30) for novel regulars (*plag-plag*) ($t(21)=2.41$ $p=.025$) (mean 9 errors made by 11 of the 15 patients able to perform the task for novel verbs; one outlier at 4 although this difference was not statistically significant) for existing irregulars (*dig-dig*) ($t(23)=1.63$ $p=.118$) (mean errors made by 8 of the 17 patients; range 6-3)

Moreover, hypokinesia, as measured by the bradykinesia UHDRS subtest, correlated with the production of unmarked forms for novel regulars and for existing regulars both in a simple correlation and when partialing out chorea (Table 8, rows a and b, columns 1 and 2). Analogous correlations with the production of unmarked forms for irregular verbs (*dig-dig*) approached significance, (Table 8, a and b, column 3), though this is attributable to the strong association between object naming and hypokinesia ($r(15)= -.51$ $p=.036$): When object naming was also partialled out, hypokinesia did not correlate at all with the production rate of unmarked irregulars, but did correlate with the production rate of unmarked novel regulars (significantly) and existing regulars (borderline significant) (Table 8, c). A similar pattern was obtained with IMC dementia scores partialled out (Table 8, d).⁷

These findings are unlikely to be fully explained by an articulatory deficit. Like irregular verbs (*dig*), but unlike novel verbs (*plag*) or non-attracted regulars (*look*), the production of unmarked forms for attracted regulars (*glide*) was not greater than that of the control subjects ($t(23)=1.67$ $p=.108$) and did not correlate with hypokinesia in any of the correlations (Table 8, rows a-d, column 4). Because attracted regular past tense forms (*glided*) are likely to be stored in memory (Ullman, 1993), a rule deficit account predicts that the production of their unmarked forms should have a similar pattern to that of irregular verbs (*dig*), while an articulatory deficit account predicts that it should be similar to that of novel regulars (*plag*) and non-attracted existing regulars (*look*). Note that these results also argue against a substrating explanation. Interestingly, object naming correlated

with the production of unmarked forms for attracted regulars (*glide*) ($r(13) = -.59$ $p = .021$), as well as for irregulars (*dug*) ($r(13) = -.75$ $p < .001$), but not for non-attracted existing regulars (*look*) ($r(13) = -.36$ $p = .16$), further supporting the lexicalization of past tense forms for irregulars and attracted regulars, but not for non-attracted regulars.

Given that in both HD and PD the suppression of movement (hypokinesia) appears to be caused by high levels of inhibition of cortical areas to which the damaged basal ganglia circuits project, we might expect similar correlation patterns in the two diseases for the production of unmarked forms. Indeed, PDs showed a correlation pattern highly similar to that which we have just described for the HDs. Across the 28 PDs, right-side hypokinesia correlated with the production rate of unmarked forms for novel regulars (*plag-plag*) ($r(26) = .52$ $p = .005$) and for existing regulars (*look-look*) ($r(26) = .64$ $p < .001$), but only borderline significantly for existing irregulars (*dig-dig*) ($r(26) = .34$ $p = .074$) and attracted regulars (*glide-glidir*) ($r(26) = .34$ $p = .062$). With object naming held partialled out, right-side hypokinesia still correlated with the production of unmarked forms for novel regulars ($r(25) = .39$ $p = .043$) and existing regulars ($r(25) = .58$ $p = .002$), but not for existing irregulars ($r(25) = .16$ $p = .428$) or attracted regulars ($r(25) = .14$ $p = .498$). In contrast, partialing out right-side hypokinesia, object naming correlated with the production of unmarked forms for existing irregulars ($r(25) = -.50$ $p = .008$), and attracted regulars ($r(25) = -.62$ $p < .001$), but not for non-attracted existing regulars ($r(25) = -.15$ $p < .460$). The similarity of this correlation pattern to that of the HDs supports the hypothesis that in both HD and PD, basal ganglia degeneration leading to movement suppression also leads to rule suppression.

Chorea correlated neither with hypokinesia (Table 7, g), nor with the production of unmarked forms for any of the four verb classes, even when partialing out hypokinesia (Table 8, f and g). Similarly, we saw above that hypokinesia (as well as a variety of other measures of disease progression and of behavioral and motor impairments) correlated neither with the rate of producing multiply- and syllabically-suffixed forms for regular and novel verbs, nor with the overregularization rate nor with rate of suffixing irregulars. Thus one type of basal ganglia lesion in HD, which leads to the excitation of frontal cortical areas and excess movement, is also associated with excess rule use, but not with suppressed movement or rule use. In contrast, another type of basal ganglia lesion in HD, which leads to the inhibition of frontal cortical areas and suppressed movement, is also associated with suppressed rule use, but not with excess movement or rule use. Moreover, in HD there is inconsistent cell loss in brain structures outside the basal ganglia, yet consistently high levels of cell loss within the basal ganglia (Vonsattel et al. 1985; see also Roos 1986) Finally, a third type of basal ganglia lesion, in PD, which also leads to the inhibition of frontal cortical areas and suppressed movement, is also associated with suppressed rule use. This combination of findings strongly indicates that it is the specific kind of lesion leading to chorea in

HD that also leads to overactive rule use, and the specific kind of lesion leading to hypokinesia in HD, and probably in PD as well, that also leads to suppressed rule use.

Summary and Conclusion

The 17 HDs inflected irregular (*dug*), regular (*looked*) and novel (*plagged*) verbs at similar rates of success. However, they produced more multiply- and syllabically- suffixed forms for regular and novel verbs (*lookeded*, *look-id*, *plaggeded*, *plag-id*) than their control subjects. The HDs' rate of producing such errors correlated with chorea, even with IMC dementia scores partialled out. These errors are unlikely to be explained by articulatory perseverations of the *-t* or *-d*, or by other motor problems because there was a lack of forms like *keptit*, *lookedet*, *kep-it*, *look-it* or *kep-id*. The HDs also produce more overregularizations (*digged*) and suffixed irregular pasts (*dugged*) than their control subjects. The HDs' rate of producing such errors correlated with chorea, even with object naming or IMC dementia scores partialled out. These errors cannot be attributed to motor perseverations of the *-t* or *-d* because such phonemes are not always present in the stem form (*dig-digged*). Unlike in AD, overregularizations were unlikely to be explained by word-finding deficits, because their overregularization rate did not correlate with object naming difficulties.

The HDs produced significantly more unmarked forms than their control subjects for regular and novel verbs (*look*, *plag*), but not for irregular verbs (*dig*). Hypokinesia correlated with the production rate of unmarked for regular and novel verbs, but not for irregular verbs, once object naming or IMC dementia scores were partialled out. The production of unmarked forms for existing and novel regulars is unlikely to be explained by articulatory problems, because the production rate of unmarked forms (*glide*) for the stored attracted regulars was like that of irregulars in not being significantly greater than that of the control subjects, and in not correlating with hypokinesia. PD patients showed a pattern of correlation between hypokinesia and the production of unmarked forms that was highly similar to that of the HD patients.

Seven measures of disease progression or of behavioral and motor impairments, including hypokinesia, correlated neither with chorea, nor with the production rate of multiply- or syllabically-suffixed forms, nor with overregularization rates. In contrast, chorea did not correlate with the production of unmarked forms. This contrast, in combination with the correlations between chorea and excess rule use in HD, and between hypokinesia and suppressed rule use in HD and PD, indicates that it is the specific kind of lesion leading to excess movement that also lead to excess rule use in HD, and that it is the specific kind of lesion leading to suppressed movement that also lead to suppressed rule use in HD and PD.

GENERAL DISCUSSION

Summary

Impairments of Declarative and Lexical Memory

Previous evidence has associated Alzheimer's disease (AD) with deficits in learning new facts, events, images, and words, as well as recalling and recognizing old ones. This suggests a general impairment of declarative memory, as well as impairments of lexical memory. In contrast, previous evidence has suggested that in AD, the learning and processing of new motor, perceptual, and cognitive skills remains relatively unimpaired, as does the processing of previously learned motor and cognitive skills. This suggests a relative sparing of the procedural system. AD patients also appear to be relatively unimpaired at processing syntax, morphology and phonology, suggesting that grammar may be relatively spared. These dissociations may be explained by findings indicating that in AD there is greater damage to medial temporal, lateral temporal, and temporo-parietal areas than to frontal cortex and the basal ganglia, as measured by density of neurofibrillary tangles, level of cell loss, and degree of hypometabolism.

For the AD patients in this study, difficulties remembering facts correlated with difficulties remembering words; each of these correlated with difficulties producing existing irregular past tense forms (*dug*), and, negatively, with overregularization errors (*digged*), and to a lesser extent with difficulties producing novel irregular pasts (*crove*), but not with difficulties producing existing or novel regular pasts (*looked, plagged, crived*). This pattern held even when partialing out dementia or when partialing out performance at 5-minute recall, which is thought to depend on the integrity of medial temporal lobe structures. Production rates of the different *-ed*-suffixed forms were inter-correlated, but not were not correlated with production rates of existing or novel irregular pasts.

The 5 most anomic AD patients, as measured by an object naming task, were worse at producing past tense forms of irregular verbs than regular verbs (*dug* vs *looked*), despite the irregular items' higher past tense frequencies, and overregularized often (*digged*). They were also worse at producing pasts of irregular verbs than novel regular verbs (*dug* vs *plagged*), and had more trouble with novel irregular pasts (*crove*) than their regularized alternatives (*crived*), compared to controls. Several other studies have revealed a similar irregular-regular dissociation for ADs in a non-grammatical domain: They have greater difficulty reading irregularly than regularly spelled words (e.g., *yacht* vs *rint*, *mint*).

Impairments of Procedures and Rules

Parkinson's disease (PD) is associated with degeneration of the nigro-striatal dopaminergic cells in the basal ganglia, resulting in inhibition of motor and other frontal cortical areas to which the basal-ganglia-thalamic circuits project. This is thought to explain the suppression of movement in PD (hypokinesia), and may account for findings suggesting impairments learning or processing new motor, perceptual and cognitive skills. PD patients also have trouble understanding sentences, and their speech can be syntactically simple, suggesting they may have difficulties with grammar. In contrast, the learning of new words, facts, events, and images, and the remembering of old ones, is relatively spared, especially for the recognition of this information.

For the PD patients in this study, right-side hypokinesia, which is caused by degeneration in the left basal ganglia, was correlated with difficulties producing existing and novel regular past tense forms (*looked, plagged, crived*), but not with difficulties producing existing or novel irregular pasts (*dug, crove*). This pattern held even when partialing out dementia or left-side hypokinesia. In contrast, left-side hypokinesia correlated with none of the measures of past-tense performance once right-side hypokinesia was partialled out. Production rates of the different *-ed*-suffixed forms were inter-correlated, but not were not correlated with production rates of existing or novel irregular pasts.

The 5 PD patients with the most severe right-side hypokinesia were worse at producing past tense forms of regular than irregular verbs (*looked* vs *dug*), and never overregularized (*digged*). They were also worse at producing pasts for novel regulars than existing irregulars (*plagged* vs *dug*), and had more trouble producing regularized pasts for novel irregulars (*crived*) than their irregular alternatives (*crove*), compared to controls.

The PD findings are unlikely to be explained by frequency differences, visual deficits, a tendency to produce embedded words, or articulatory difficulties. In particular, three lines of evidence argue against an articulatory deficit account. First, the 5 hypokinetic PD subjects did not make phonological errors on irregulars (*keep-kep*) analogous to those made on regulars (*look-look*). Second, in a new set of regular and irregular verbs, matched one-to-one on pronounceability and frequency (*passed-lost*), the retested hypokinetic PD subjects were worse at producing regular than irregular past tense forms. In this retest the PDs were also worse at producing regular pasts than at repeating pronounceability- and frequency-matched uninflected words (*passed* vs *fast*). Third, two types of regular past tense forms that are retrieved from memory ("attracted regulars" like *glided* and "doublet regulars" like *dived*) were not produced less successfully than irregulars, and showed a correlation pattern similar to that of irregular pasts (*dug*), not regular or novel pasts (*looked, plagged*).

A Role for The Basal Ganglia in Grammatical Rule Processing

In Huntington's patients, excess movement (chorea, a type of hyperkinesia), caused by a specific kind of basal ganglia lesion which leads to excess excitation of frontal cortical areas, was associated with excess use of the *-ed*-suffixation rule: the production of multiply- and syllabically-suffixed regular and novel verbs (*lookeded, look-id, plaggeded, plagg-id*), and overregularizations (*digged, dugged*). These forms were unlikely to be explained by motor perseverations of *-t* or *-d* because forms like *keptit, lookedet, kep-it, look-it* or *kep-id* were lacking, and overregularization errors were produced from stems lacking a final *-t* or *-d* (e.g., *digged*). Unlike in AD, overregularizations were not associated with difficulties remembering words, underscoring a role for the basal ganglia in grammatical rule processing.

Suppressed movement (hypokinesia), caused in Huntington's and Parkinson's patients by basal ganglia lesions leading to the inhibition of frontal cortical areas, was associated with suppressed use of the *-ed*-suffixation rule. In HD and HD, hypokinesia correlated with the production of unmarked forms for novel and existing regular verbs (*plag, look*), but not with the production of unmarked forms for irregular verbs (*dig*), partialing out object naming. This contrast was unlikely to be caused by articulatory difficulties, because hypokinesia was not associated with the production of unmarked forms for attracted regulars (*glide*), whose past tense forms (*glided*) can be stored in memory.

A number of measures of disease progression and of behavioral and motor impairments, including suppressed movement (hypokinesia), were *not* correlated with either excess movement (chorea) or excess rule use. Conversely, chorea did *not* correlate with the production of unmarked forms. This contrast, in combination with the correlations between chorea and excess rule use in HD, and between hypokinesia and suppressed rule use in HD and PD, as well as the lack of consistent cell loss outside the basal ganglia in HD, indicates that it is the specific kind of lesion leading to excess movement in HD that also lead to excess rule use, and that it is the specific kinds of lesion leading to suppressed movement in HD and PD that also lead to suppressed rule use. Thus the basal ganglia appear to play a similar role in motor and rule programming.

Conclusion

Previous evidence has suggested that irregularly inflected forms are stored in and retrieved from associative memory, while regularly inflected forms are generally produced in a distinct system by a grammatical symbol-processing-like rule.

In this paper, the demonstration of double dissociations over irregularized and regularized inflected forms between the ADs and the posterior aphasic on the one hand, and the PDs and

anterior aphasics on the other, suggests that one neural system subserves the computation of irregulars, while another subserves the computation of regulars.

Previous studies have suggested that arbitrary information such as facts and events are consolidated in medial temporal structures, and eventually stored in neocortex. In this paper and in other studies it has been shown that for ADs, posterior aphasics, PDs, and anterior aphasics, there are correlations or co-occurrences of the degree of damage to temporal or temporo-parietal regions, and the degree of impairment at remembering images, facts, events, and words, at the production of irregular pasts, and at the reading of irregular pasts and plurals, and the reading of irregularly spelled forms. This suggests that these functions are subserved by a temporal lobe system subserving the memory of arbitrary information: that is, declarative memory.

Previous studies have suggested that the basal ganglia, which project to frontal cortex, are necessary for learning and processing skills, and that grammatical processing may depend on frontal cortex and/or the basal ganglia. In this paper and in other studies it has been shown that for ADs, posterior aphasics, PDs, and anterior aphasics, there are correlations or co-occurrences of the degree of damage to frontal cortex or the basal ganglia, and the degree of impairment at learning or processing motor, perceptual and cognitive skills, at processing of grammar, and at the production of existing and novel regular pasts, the reading of regular pasts and plurals, and the reading of regularly spelled forms. This suggests that these functions are subserved by a frontal/basal-ganglia system subserving skills and rules: that is, the procedural system.

In this paper and in other studies it has been shown that for ADs, posterior aphasics, PDs, and anterior aphasics, there is a *lack* of correlations and co-occurrences between the degree of damage to temporal and temporo-parietal areas, and the degree of impairment of skill- and rule-like functions; and between the degree of damage to frontal cortex or the basal ganglia, and the degree of impairment of the memory of arbitrary information. This suggests that the temporal lobe declarative memory system does not subserve the processing of skills and rules, and that the frontal/basal-ganglia procedural system may not underlie the memory of arbitrary information.

Finally, this study has shown that the kind of basal ganglia lesions in PD and HD which are thought to lead to the suppression of movement, also lead to the suppression of rule use. In contrast, a different kind of basal ganglia lesion in HD, which is thought to lead to excess movement, also leads to excess rule use. This suggests that the basal ganglia may play a similar role in motor and rule programming.

These findings and conclusions allow us to address the the three questions about the neural basis of language that were asked in the beginning of this paper:

(1) How many brain systems underlie language, and what class of functions does each compute? Lexical memory appears to be part of the declarative memory system underlying the learning and storage of arbitrary information; this information may be represented associatively. Grammatical rules appear to be processed by the procedural system underlying the learning and processing of skills and rules; grammatical rules may be computed according to symbol-processing-like principles.

(2) Where in the brain are the systems underlying language? Lexical information may be consolidated in medial temporal structures, and eventually stored in temporal or temporo-parietal neocortex. The frontal/basal-ganglia system appears to contribute to the learning and processing of grammatical rules.

(3) What neural mechanisms underlie the systems' computations of language functions? This paper has addressed this question only for the procedural system; the evidence indicates that the well-studied basal ganglia circuits underlying motor programming may play a comparable role in rule programming.

Further Discussion

How many brain systems underlie language?

I have argued that at least two major brain systems underlie language. However, I have not claimed that there are only two brain systems. First, it would not be surprising if other major brain systems also subserved language. Second, grammatical rules might be subserved by specialized cortical neural structures in addition to those underlying the procedural system, just as specialized cortical motor programs appear to complement the role of the basal ganglia in the domain of movement (e.g., in the motor humunculus). Third, the evidence presented in this paper only addresses grammatical rule processing. No claims are made about the neural basis of other aspects of grammar, such as various principles (e.g., binding principles; Chomsky, 1981) and filters (e.g., case filter; Chomsky and Lasnik, 1977).

Specialization within each brain system

This paper attempts to *synthesize*, by implicating two major brain systems in a variety of language and non-language functions. But I have not argued that there is no specialization *within* each of the two brain systems. Indeed, an enormous amount of research in the past two decades has suggested a many-dimensional partitioning of stored information, such as verbs vs nouns, and animate vs non-animate categories (e.g., Warrington and Shallice, 1984; McCarthy and Warrington, 1988; Hart and Gordon, 1992), all of which might be learned and stored in the declarative memory

system. Similarly, within the procedural system there are known to be not only different functionally segregated circuits, such as the "motor circuit," "oculomotor circuit," or "limbic circuit," but also sub-circuits, each subserving different functions within a given circuit, and each apparently structurally segregated from and parallel to the others (Alexander, Crutcher, & DeLong, 1990). Thus it would not be surprising if different grammatical and non-grammatical rules were subserved by different sub-circuits. Moreover, because the functional segregation is maintained at the level of cortex, these hypothesized rule sub-circuits might also be maintained to the level of cortex.

Pertinence of non-language research

By tying lexical memory to declarative memory, and grammatical rule processing to the procedural system, it follows that findings from previous and future studies of these two systems should also pertain to language. A large body of research may therefore be pertinent to the study of language. For example: (1) Advances in explaining the neural mechanisms of the basal ganglia in movement may also be relevant in accounting for their role in rule processing. (2) The implication of other brain structures in the declarative or procedural systems would suggest that such brain structures may also play a role in language; thus the cerebellum, which has been implicated in procedural memory (e.g., Sanes, Dimitrov, & Hallet, 1990; Grafman et al., 1992), may also be involved in grammatical rules.

Pertinence of language findings to the two brain systems

Conversely, findings within the domain of language may shed light on each of the two brain systems. Thus the multiple- and syllabic-suffixation found among the HD patients might help elucidate the nature of the basal ganglia impairment in HD, as well as the normal function of the basal ganglia. In particular, the all-or-nothing and easily detectable nature of grammatical rules such as the *-ed*-suffixation rule might prove useful in the study of basal ganglia function.

Temporal cortex: Storage or retrieval?

Although the results from this study indicate that temporal and temporo-parietal regions subserve lexical memory, it does not distinguish between a role in the storage or retrieval of lexical information. However, there are at least two lines of evidence that suggest that these regions subserve the storage of lexical information, whether or not they are also involved in retrieval. First, studies of declarative memory suggest that the memories are stored in lateral cortex to which they are connected (see Introduction). Second, AD patients, who have high levels of degeneration in temporal and temporo-parietal cortex, compared to other cortical areas (see page~%pagerefADNeuropathology), are impaired not only at the recall, but also at the recognition of words as well as other arbitrary information (see page~%pagerefExperiment3:AD).

Temporal cortex and the sparing of grammatical rule processing

Findings from this study suggest that temporal and temporo-parietal regions may not subserve grammatical rule processing. Moreover, they suggest that rule processing for the ADs and posterior aphasic may be *facilitated*: First, multiply- or syllabically-suffixed forms for regular and novel verbs were produced by the 24 ADs (posterior aphasic (2 multiply-suffixed regulars, or Second, both the posterior aphasic and the ADs produced significantly more overregularizations (*digged*) as a percentage of irregular errors than their control subjects. Third, the 5 anomic AD subjects' dementia-adjusted means for regular verbs and for novel verbs were higher than those for their controls (see page~%pagerefADRuleFacilitation).

What could account for this apparent rule facilitation? It does not appear to be explainable by the same set of factors resulting in rule over-activation in HD: While in AD object naming correlated negatively with the rate of producing overregularizations (*digged*), indicating that such forms were produced when the irregular could not be remembered, in HD this correlation was not significant, implicating non-lexical impairments. However, it may also be possible that the temporal lobe damage in AD and posterior aphasia not only leads directly to lexical difficulties, but also indirectly to rule facilitation: Several studies have suggested that lesions to the hippocampus can result in increased dopamine transmission in the portion of the striatum to which the hippocampus projects (part of the basal ganglia "limbic circuit") (Jaskiw, Karoum, & Weinberger, 1990; Lipska et al., 1992; Brene et al., 1993; Springer and Isaacson, 1982). Moreover, independent evidence suggests that increasing dopamine levels in the basal ganglia can result in excess movements of the sort found in HD (see Gray et al., 1991). Given that temporal and posterior parietal cortex also project to the striatum, in the "prefrontal" circuits (Alexander, DeLong and Strick, 1986; Alexander, Crutcher, DeLong, 1990), rule facilitation in AD and posterior aphasia might be explained if damage to striatal input cells in other circuits, such as the circuitry which we may underlie grammatical rules, also resulted in increased dopamine transmission (see immediately below).

Interaction between the two brain systems and blocking

A widely observed phenomenon in language is interaction between an exceptional form, such as an irregular (*dug*), and its corresponding general form, such as the corresponding overregularized form (*digged*), which is *blocked* by the exceptional form; failure to compute the exceptional form can result in overregularization errors (*digged*). Thus people who have trouble remembering irregular past tense forms, such as children (Marcus et al., 1992), or AD patients or posterior aphasics (shown in this paper), tend to overregularize.

I have argued that irregular past tense forms are stored in temporal or temporo-parietal cortex. These cortical areas project excitatorily directly to the striatum (Alexander, DeLong and Strick, 1986; Alexander, Crutcher, DeLong, 1990). If projections from temporal cortical representations of irregular past tense forms impinged primarily upon striatal projections to the basal ganglia's "indirect pathway," successful computation of an irregular (*dug*) should excite striatal projections to the indirect pathway, leading to the inhibition of frontal cortical areas to which the basal ganglia circuitry projects, and the suppression of its programming (Young and Penney, 1993). If this circuitry projects to frontal cortical areas subserving the *-ed*-suffixation rule, the programming of this rule would be suppressed; thus the computation of *digged* would be suppressed. However, when an irregular (*dug*) was not successfully computed because its memory representation was weak (in children) or degraded (in Alzheimer's disease and posterior aphasia), rule programming would not be suppressed, and an overregularization (*digged*) might be computed. Similarly, if no irregular exists (*look, plag*), rule programming should also proceed unsuppressed.

Basal ganglia circuits also receive excitatory striatal inputs from their own frontal cortical output regions, forming a closed loop (Alexander, DeLong and Strick, 1986; Alexander, Crutcher, DeLong, 1990). Thus frontal cortical areas subserving rule programming should project excitatorily back to the striatum. If these frontal inputs to the striatum were similar to the posterior inputs in that they also impinged upon portions of the striatum projecting to the "indirect pathway," successful rule programming would lead to the suppression of further rule programming. This could explain why people without brain damage tend not to utter multiply-suffixed forms (*lookeded, diggeded*): Computation of the rule suppresses additional rule computation, in a negative feedback loop. In contrast, this closed loop suppression would tend to be ineffective in patients with Huntington's disease, whose indirect pathway is lesioned, and therefore dysfunctional. The indirect pathway dysfunctionality in HD could also explain HD overregularizations: Although the irregular past (*dug*) might be successfully computed in posterior cortex, their excitatory outputs to the striatum would not successfully activate the dysfunctional indirect pathway, resulting in a lack of suppression of rule programming in frontal cortex, and overregularizations like *digged*. Moreover, the rule might also be applied to the successfully computed irregular past (*dug*), resulting in forms such as *dugged*. Indeed, for the 17 HDs, while the ADs, PDs and controls did not produce a single such form among them (the difference was significant in all three cases: 17 HDs vs 24 ADs, $t(39)=2.21$ $p=.033$; 17 HDs vs 28 PDs, $t(43)=2.39$ $p=.021$; 17 HDs vs all 40 controls subjects, $t(55)=2.88$ $p=.006$). In contrast, in PD, degeneration of the nigro-striatal dopaminergic projections leading to the inhibition of cortical areas should cause suppression of rule programming even when the irregular is not successfully computed, thus explaining the lack of overregularizations among the hypokinetic PDs, and the production of unmarked forms for regular and novel verbs.

Language in AD, PD, HD

The findings in this study also shed light on language impairments in AD, PD and HD.

AD: The study provides further support for the view that in AD grammatical processing is spared, while lexical memory is not. Moreover, the results also elucidate the nature of the naming deficit in AD, for which three accounts have been proposed: lexical, semantic, and visual (for discussions, see Huff, 1990; also Nebes, 1989). The relative deficit in the production of irregular past tense forms suggests that the AD naming deficit is not solely visual or semantic, because irregulars and regulars do not differ any principled manner either visually or semantically.

PD: The findings suggest that PD is associated with impairments of grammatical rule processing, as a result of the nigro-striatal degeneration in the basal ganglia. Our results are also consistent with previous findings suggesting a relative sparing of lexical memory in PD, even for word recall: While the PDs' control subjects correctly named 8 ($t(40)=-.07$ $p=.944$), and the 5 most hypokinetic PDs named 8 ($t(17)=-.79$ $p=.442$).

HD: Our findings indicate that HD is associated with impairments of grammatical rule processing, caused by neostriatal degeneration in the basal ganglia. Moreover, these impairments are of two types, analogous to the two major types of motor impairments found in HD: excess rule use and suppressed rule use.

NOTES

¹ Non-doublet irregulars were defined as those verbs whose regularized pasts' mean acceptability ratings by normal young adults in past tense sentential contexts were less than 3.5, from an acceptability ratings scale of 1 to 7 (7 being most acceptable), as tested in Ullman (1993). This cutoff corresponded well to our own judgments, distinguishing doublets (*{%it dive-dove/dived}*) from non-doublet irregulars (*{%it dig-dug/*digged}*).

² No instances of *{%it wrang}* were produced by the Broca's or anomic aphasics. *{%it Wrang}* was counted as incorrect for the remaining subject groups; however, only 4 of the 24 probable Alzheimer's disease patients, and 1 of the 17 Huntington's disease patients, produced this form, making it unlikely that its inclusion as correct would have yielded different results. Interestingly, 9 of the 28 Parkinson's patients produced *{%it wrang}*; thus if the form had been included as correct, it would have strengthened our claim that Parkinson's disease impairs the production of rule-constructed regulars more than irregulars.

³ In this paper all frequencies are natural logarithm transformed, with 1 added to the raw frequency count before the transformation to avoid logarithms of zero.

⁴ AD cannot be diagnosed with certainty before autopsy, and can be difficult to differentiate from other degenerative dementias. The patients reported by Schwartz, Marin and Saffran (1979), Whitaker (1976), and Irigaray (1973; see Obler, 1981) involved unspecified degenerative dementias.

⁵ Categorization as standard American English was made by the experimenter. In this paper, bilinguals are defined as having had important exposure (input from at least one primary caretaker and/or older siblings) to English and one or more other languages before the age of five.

⁶ This unexpectedly large difference for subject EF may have been explained by a negative association between dementia and the processing of novel regulars. There was an interaction between group, verb class, and IMC scores ($F(1,14)=8.83$ *{%it p}=.010*), with follow-up tests suggesting that it could be explained by a negative correlation between IMC scores and performance at producing novel regulars for the ADs, but not for the controls. That is, as their dementia (which ranged from 12 to 21) increased, the 5 ADs' performance on novel regulars diminished, while this did not hold for the controls (whose IMC scores ranged from 0 to 4). This relationship is not too surprising, given that dementia might be associated with conceptual difficulty understanding the task for novel verbs. Because subject EF had the highest IMC score of all 24 ADs, she may have had the most trouble with novel forms. In support of this view, she also produced fewer regular and irregular pasts for novel irregulars (*{%it crive-crove/crived}*) than the other 4 anomic ADs; that is, her performance at all novel verbs, regular and irregulars alike, as worse than the other ADs.

⁷ When the patient who was an outlier in the production of unmarked forms for novel regulars (*{%it plag-plag}*) (4 and Pearson's parametric correlations were carried out, the same pattern of results was obtained as was found with this patient included in Spearman rank-order correlations: The production of unmarked novel regulars correlated with bradykinesia (*{%it r}(12)=.76* *{%it p}=.002*), even partialing out chorea (*{%it r}(11)=.75* *{%it p}=.003*), partialing out chorea and object naming (*{%it r}(10)=.69* *{%it p}=.012*), and partialing out chorea and IMC dementia scores (*{%it r}(10)=.70* *{%it p}=.011*). The production of unmarked novel regulars did not correlate with chorea (*{%it r}(12)= -.27* *{%it p}=.353*), or with chorea partialing out bradykinesia (*{%it r}(11)= -.22* *{%it p}=.480*).

REFERENCES

- Albin, R. L., Young, A. B., & Penney, J. B. (1989). The functional anatomy of basal ganglia disorders. *Trends in Neuroscience*, *12*(10), 366-375.
- Alexander, G. E., Crutcher, M. D., & DeLong, M. R. (1990). Basal ganglia-thalamocortical circuits: Parallel substrates for motor oculomotor 'prefrontal' and 'limbic' functions. In H. B. M. Uylings, C. G. V. Eden, J. P. C. DeBruin, M. A. Corner, & M. G. P. Feenstra (Eds.), *Progress on Brain Research* (Vol. 85, pp. 119-146): Elsevier Science Publishers B.V.
- Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, *9*, 357-381.
- Allain, H. (1995). Procedural memory and Parkinson's disease. *Dementia*, *6*, 174-178.
- Allain, H., Lieury, A., Thomas, V., Reymann, J. M., Gandon, J. M., & Belliard, S. (1995). Explicit and procedural memory in Parkinson's disease. *Biomedical & Pharmacotherapy*, *49*, 179-186.
- Arnold, S. E., Hyman, B. T., Flory, J., Damasio, A. R., & Hoesen, G. W. V. (1991). The topographical and neuroanatomical distribution of neurofibrillary tangles and neuritic plaques in the cerebral cortex of patients with Alzheimer's disease. *Cerebral Cortex*, *Jan/Feb 1*, 103-116.
- Balota, D. A., & Ferraro, F. R. (1993). A dissociation of frequency and regularity effects in pronunciation performance across young adults, older adults, and individuals with senile dementia of the Alzheimer's type. *Journal of Memory and Language*, *32*, 573-592.
- Bates, E., & Wulfeck, B. (1989). Crosslinguistic studies of aphasia, *The crosslinguistic study of sentence processing* (pp. 328-371). New York: Cambridge University Press.
- Bayles, K. A. (1982). Language Function in Senile Dementia. *Brain and Language*, *16*, 265-280.
- Bayles, K. A. (1990). Language and Parkinson's disease. *Alzheimer Disease and Associated Disorders*, *4*, 171-180.
- Beatty, W. W., Winn, P., Adams, R. L., Allen, E. W., Wilson, D. A., Prince, J. R., Olson, K. A., Dean, K., & Littleford, D. (1994). Preserved cognitive skills in dementia of the Alzheimer type. *Archives of Neurology*, *51*, 1040-1046.
- Beatty, W. W., Zavadil, K. D., Bailly, R. C., Rixen, G. J., Zavadil, L. E., Farnham, N., & Fisher, L. (1988). Preserved musical skill in a severely demented patient. *International Journal of Clinical Neuropsychology*, *10*(4), 158-164.
- Blackwell, A., & Bates, E. (1995). Inducing agrammatic profiles in normals: Evidence for the selective vulnerability of morphology under cognitive resource limitation. *Cognitive Neuroscience*, *7*, 228-257.
- Blessed, G., Tomlinson, B. E., & Roth, M. (1968). The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *British Journal of Psychiatry*, *114*, 797-811.
- Bondi, M. W., & Kasniak, A. W. (1991). Implicit and explicit memory in Alzheimer's disease and Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology*, *13*(2), 339-358.

Brene, S., Lindfors, N., Herrera-Marschitz, M., & Persson, H. (1993). Differential regulation of preprotachykinin-A mRNA expressions in striatum by excitation of hippocampal neurons. *European Journal of Neuroscience*, 5, 839-845.

Brun, A., & Gustafson, L. (1976). Distribution of cerebral degeneration in Alzheimer's disease. *Archiv fur Psychiatrie und Nervenkrankheiten*, 223, 15-33.

Butters, N., PhD, Jessica Wolfe, P., Maryann Martone, B., Eric Granholm, B., & Laird S. Cermack, P. (1985). Memory disorders associated with Huntington's disease: Verbal recall, verbal recognition and procedural memory. *Neuropsychology*, 23(6), 729-743.

Bybee, J. L., & Moder, C. L. (1983). Morphological classes as natural categories. *Language*, 59(2), 251-270.

Bybee, J. L., & Slobin, D. I. (1982). Rules and schemas in the development and use of the English past tense. *Language*, 58(2), 265-289.

Caramazza, A., & Zurif, E. B. (1976). Dissociation of algorithmic and heuristic processes in language comprehension: Evidence from aphasia. *Brain and Language*, 3, 572-582.

Carlesimo, G. A., & Oscar-Merman, M. (1992). Memory deficits in Alzheimer's patients: A comprehensive review. *Neuropsychological Review*, 3(2), 119-169.

Chomsky, N. (1965). *Aspects of the theory of syntax*. Cambridge, MA: The MIT Press.

Chomsky, N. (1981). *Lectures on government and binding*. Dordrecht: Foris.

Chomsky, N., & Halle, M. (1968). *The sound pattern of English*. New York: Harper and Row.

Chomsky, N., & Lasnik, H. (1977). Filters and control. *Linguistic Inquiry*, 8, 425-504.

Cohen, N. J., & Squire, L. R. (1980). Preserved learning and retention of pattern-analyzing skills in amnesia: Dissociation of knowing how and knowing that. *Science*, 210, 207-210.

Coltheart, M., Masterson, J., Byng, S., Prior, M., & Riddoch, J. (1983). Surface Dyslexia. *Quarterly Journal of Experimental Psychology*, 35(A), 469-495.

Coltheart, M., Patterson, K., & Marshall, J. C. (1980). *Deep dyslexia*. London: Routledge.

Corkin, S. (1982). Some relationships between global amnesias and the memory impairments in Alzheimer's disease, *Alzheimer's disease: A report of progress* (pp. 149-164). New York: Raven Press.

Coslett, H. B. (1986,). *Dissociation between reading of derivational and inflectional suffixes in two phonological dyslexics*. Paper presented at the Academy of Aphasia, Nashville.

Coslett, H. B. (1988, October). *A selective morphologic impairment in writing: Evidence from a phonological dysgraphic*. Paper presented at the Academy of Aphasia, Montreal.

Crystal, H. A., Grober, E., & Masur, D. (1989). Preservation of musical memory in Alzheimer's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, 52, 1415-1416.

Cummings, J. L., & Zarit, J. M. (1987). Probable Alzheimer's disease in an artist. *Journal of the American Medical Association*, 258(19), 2731-2734.

Cushman, L. A., & Caine, E. D. (1987). A controlled study of processing of semantic and syntactic information in Alzheimer's disease. *Archives of Clinical Neuropsychology*, 2, 283-292.

Dall'Ora, P., Sala, S. D., & Spinnler, H. (1989). Autobiographical memory. Its impairments in the amnesic syndromes. *Cortex*, 25, 187-217.

Damasio, A. R. (1992). Aphasia. *New England Journal of Medicine*, 326, 531-539.

- Damasio, A. R., & Damasio, H. (1992). Brain and language. *Scientific American*, 267(3), 88-95.
- de Saussure, F. (1959). *A course in general linguistics*. London: Peter Owen.
- Deweer, B., Pillon, B., Michon, A., & Dubois, B. (1993). Mirror reading in Alzheimer's disease: Normal skill learning and acquisition of item-specific information. *Journal of Clinical and Experimental Neuropsychology*, 15(5), 789-804.
- Dronkers, N. F., Wilkins, D. P., Jr., R. D. V. V., Redfern, B. B., & Jaeger, J. J. (1994,). *Platform Session 3: 1. A reconsideration of the Brain Areas involved in the Disruption of Morphosyntactic Comprehension*. Paper presented at the Academy of Aphasia Conference.
- Dubois, B., F. Boller, Pillon, B., & Agid, Y. (1991). Cognitive deficits in Parkinson's disease, *Handbook of Neuropsychology* (Vol. 5, pp. 195-240). Amsterdam: Elsevier.
- Dustin, P., Brion, J. P., & Flament-Durand, J. (1992). What's new in the pathology of neuronal cytoskeleton: The significance of neurofibrillary tangles. *Pathol Res Pract*, 188, 248-253.
- Eslinger, P. J., & Damasio, A. D. (1986). Preserved motor learning in Alzheimer's disease: Implications for anatomy and behavior. *Journal of Neuroscience*, 6, 3006-3009.
- Fahn, S., & Elton, R. L. (1987). Unified Parkinson's disease rating scale. In C. D. Marsden & M. Goldstein (Eds.), *Recent Developments in Parkinson's Disease II* (pp. 153-163). New York: MacMillan.
- Ferraro, F. R., Balota, D. A., & Connor, L. T. (1993). Implicit memory and the formation of new associations in nondemented Parkinson's disease individuals and individuals with senile dementia of the Alzheimer's type. *Brain and Cognition*, 21, 163-180.
- Flowers, K. A., Pearce, I., & Pearce, J. M. (1984). Recognition memory in Parkinson's disease. *Journal of Neurology*, 47, 1174-1181.
- Francis, N., & Kucera, H. (1982). *Frequency analysis of English usage: Lexicon and grammar*. Boston MA: Houghton Mifflin.
- Freedman, M., Rivoira, P., Butters, N., Sax, D. S., & Feldman, R. G. (1984). Retrograde amnesia in Parkinson's disease. *Le Journal Canadien Des Sciences Neurologiques*, 11, 297-301.
- Gabrieli, J. D. E., Corkin, S., Mickel, S. F., & Growdon, J. H. (1993). Intact acquisition and long-term retention of mirror-tracing skill in Alzheimer's disease and in global amnesia. *Behavioral Neuroscience*, 107(6), 899-910.
- Geschwind, N. (1965). Disconnexion syndromes in animals and man. *Brain*, 88, 237-294.
- Globus, M., Mildworf, B., & Melamed, E. (1985). Cerebral blood flow and cognitive impairment in Parkinson's disease. *Neurology*, 35, 1135-1139.
- Goodglass, H. (1993). *Understanding aphasia*. San Diego, CA: Academic Press.
- Goodglass, H., & Kaplan, E. (1972). *The assessment of aphasia and related disorders*. Philadelphia: Lea and Febiger.
- Goodglass, H., Kaplan, E., & Weintraub, S. (1983). *Boston Naming Test*. Philadelphia: Lea and Febiger.
- Grady, C. L., & Rapoport, S. I. (1992). Cerebral metabolism in aging and dementia, *Handbook of mental health and aging* (2nd ed., pp. 201-228). New York: Academic Press.
- Grafman, J., Litvan, I., Massaquoi, S., Stewart, M., Sirigu, A., & Hallet, M. (1992). Cognitive planning deficit in patients with cerebellar atrophy. *Neurology*, 42, 1493-1496.
- Gray, J. A., Feldon, J., Rawlins, J. N. P., Hemsley, D. R., & Smith, A. D. (1991). The neuropsychology of schizophrenia. *Behavioral and Brain Sciences*, 14, 1-84.

- Grodzinsky, Y. (1986). Language deficits and the theory of syntax. *Brain and Language*, 27, 135-159.
- Grosse, D. A., Wilson, R. S., & Fox, J. H. (1991). Maze learning in Alzheimer's disease. *Brain and Cognition*, 15, 1-9.
- Grossman, M., Carvell, S., Gollomp, S., Stern, M. B., Vernon, G., & Hurtig, H. I. (1991). Sentence comprehension and praxis deficits in Parkinson's disease. *Neurology*, 41, 1620-1626.
- Grossman, M., Carvell, S., Stern, M. B., Gollomp, S., & Hurtig, H. I. (1992). Sentence comprehension in Parkinson's disease: The role of attention and memory. *Brain and Language*, 42, 347-384.
- Growdon, J. H., & Corkin, S. (1986). Cognitive impairments in Parkinson's disease. In M. D. Yahr & K. J. Bergmann (Eds.), *Advances in Neurology* (Vol. 45, pp. 383-392). New York: Raven Press.
- Halle, M., & Mohanan, K. P. (1985). Segmental phonology of modern English. *Linguistic Inquiry*, 16(1), 57-116.
- Harrington, D. L., Haaland, K. Y., Yeo, R. A., & Marder, E. (1991). Procedural memory in Parkinson's disease: Impaired motor but not visuoperceptual learning. *Journal of Clinical and Experimental Neuropsychology*, 12, 323-339.
- Hart, J., & Gordon, B. (1992). Neural subsystems for object knowledge. *Nature*, 359, 60-64.
- Heindel, W. C., Butters, N., & Salmon, D. P. (1988a). Impaired learning of a motor skill in patients with Huntington's disease. *Behavioral Neuroscience*, 102, 141-147.
- Heindel, W. C., Butters, N., & Salmon, D. P. (1988b). Impaired learning of a motor skill in patients with Huntington's disease. *Behavioral Neuroscience*, 102(1), 141-147.
- Heindel, W. C., Salmon, D. P., & Butters, N. (1991). The biasing of weight judgments in Alzheimer's and Huntington's diseases. *Journal of Clinical and Experimental Neuropsychology*, 13, 189-203.
- Heindel, W. C., Salmon, D. P., Schults, C. W., Walicke, P. A., & Butters, N. (1989). Neurophysiological evidence for multiple implicit memory systems: A comparison of Alzheimer's, Huntington's, and Parkinson's Disease Patients. *Journal of Neuroscience*, 2(7), 582-587.
- Hickok, G., Zurif, E., & Canseco-Gonzalez, E. (1993). Structural description of agrammatic comprehension. *Brain and Language*, 45.
- Hier, D. B., Hagenlocker, K., & Shindler, A. G. (1985). Language disintegration in dementia: Effects of etiology and severity. *Brain and Language*, 25, 117-133.
- Hoover, J. E., & Strick, P. L. (1993). Multiple output channels in the basal ganglia. *Science*, 259, 819-821.
- Huber, S. J., Shuttleworth, E. L., & Paulson, G. W. (1986). Dementia in Parkinson's disease. *Archives of Neurology*, 43, 987-990.
- Huff, F. J. (1990). *Handbook of Neuropsychology, Language in normal aging and age-related neurological diseases* : Elsevier Science Publishers B.V.
- Huff, F. J., Corkin, S., & Growdon, J. H. (1986). Semantic impairment and anomia in Alzheimer's disease. *Brain and Language*, 28, 235-249.
- Huff, F. J., Mack, L., Mahlmann, J., & Greenberg, S. (1988). A comparison of lexical-semantic impairments in left hemisphere stroke and Alzheimer's disease. *Brain and Language*, 34, 262-278.

- Illes, J. (1989). Neurolinguistic features of spontaneous language production dissociate three forms of neurodegenerative disease: Alzheimer's, Huntington's, and Parkinson's. *Brain and Language*, 37, 628-642.
- Illes, J., Metter, E. J., Hanson, W. R., & Iritani, S. (1988). Language production in Parkinson's disease: Acoustic and linguistic considerations. *Brain and Language*, 33, 146-160.
- Irigaray, L. (1973). *Le Langage des D'ements*. The Hague: Mouton.
- Jackson, G. M., Jackson, S. R., Harrison, J., Henderson, L., & Kennard, C. (1995). Serial reaction time learning and Parkinson's disease: evidence for a procedural learning deficit. *Neuropsychologia*, 33(5), 577-593.
- Jaskiw, G. E., Karoum, F. K., & Weinberger, D. R. (1990). Persistent elevations of dopamine and its metabolites in the nucleus accumbens after mild subchronic stress in rats with ibotenic acid lesions of the medial prefrontal cortex. *Brain Research*, 534, 321-323.
- Kemper, T. (1984). Neuroanatomical and neuropathological changes in normal aging and dementia. In M. L. Albert & J. E. Knoefel (Eds.), *Clinical neurology of aging* (1st ed., pp. 9-52). New York: Oxford University Press.
- Kemper, T. L. (1994). Neuroanatomical and neuropathological changes during aging and dementia, *Clinical Neurology of Aging* (pp. 3-67). New York: Oxford University Press.
- Kempler, D., Curtiss, S., & Jackson, C. (1987). Syntactic preservation in Alzheimer's disease. *Journal of Speech and Hearing Research*, 30, 343-350.
- Kertesz, A. (1982). *Western Aphasia Battery*. New York: Grune and Stratton.
- Khachaturian, Z. S. (1985). Diagnosis of Alzheimer's disease. *Archives of Neurology*, 42, 1097-1105.
- Kim, J. J., Pinker, S., Prince, A., & Prasada, S. (1991). Why no mere mortal has ever flown out to center field. *Cognitive Science*, 15, 173-218.
- Knopman, D. (1991). Long-term retention of implicitly acquired learning in patients with Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, 13, 880-894.
- Knopman, D., & Nissen, M. J. (1987). Implicit learning in patients with probable Alzheimer's disease. *Neurology*, 37, 784-788.
- Kopelman, M. D. (1989). Remote and autobiographical memory temporal context memory and frontal atrophy in Korsakoff and Alzheimer's patients. *Neuropsychologia*, 27, 437-460.
- Lieberman, P., Kako, E., Friedman, J., Tajchman, G., Feldman, L. S., & Jiminez, E. B. (1992). Speech production syntax comprehension and cognitive deficits in Parkinson's disease. *Brain and Language*, 43, 169-189.
- Marcus, G. F., Brinkmann, U., Clahsen, H., Wiese, R., & Pinker, S. (1995). German inflection: The exception that proves the rule. *Cognitive Psychology*, 29, 189-256.
- Marcus, G. F., Pinker, S., Ullman, M., Hollander, M., Rosen, T. J., & Xu, F. (1992). Overregularization in language acquisition. *Monographs of the Society for Research in Child Development*, 57(4, Serial No. 228).
- Marin, O. S. M., Saffran, E. M., & Schwartz, M. F. (1976). Dissociations of language in Aphasia: Implications for normal functions. *Annals of the New York Academy of Sciences*, 868-884.
- Martone, M., Butters, N., Payne, M., Becker, J. T., & Sax, D. S. (1984). Dissociations between skill learning and verbal recognition in amnesia and dementia. *Archives of Neurology*, 41, 965-970.

- Matison, R., Mayeux, R., Rosen, J., & Fahn, S. (1982). 'Tip-of-the-tongue' phenomenon in Parkinson's disease. *Neurology*, *32*, 567-570.
- Mazoyer, B. M., Tzourio, N., Frak, V., Syrota, A., Murayama, N., Levrier, O., Salamon, G., Dehaene, S., Cohen, L., & Mehler, J. (1993). The cortical representation of speech. *Journal of Cognitive Neuroscience*, *5*(4), 467-479.
- McCarthy, R. A., & Warrington, E. K. (1988). Evidence for modality-specific meaning systems in the brain. *Nature*, *334*, 428-430.
- McKhann, G., Drachman, D., Folstein, M., Katzman, R., & Price, D. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's disease. *Neurology*, *34*, 939-944.
- Middleton, F. A., & Strick, P. L. (1994). Anatomical evidence for cerebellar and basal ganglia involvement in higher cognitive function. *Science*, *266*, 458-461.
- Mishkin, M., Malamut, B., & Bachevalier, J. (1984). Memories and Habits: Two Neural Systems, *Neurobiology of Learning and Memory* (pp. 65-77). New York: Guilford Press.
- Muscovitch, M. (1982). A neuropsychological approach to perception and memory in normal and pathological aging. In F. I. M. Craik & S. Trehub (Eds.), *Aging and cognitive processes*. New York: Plenum Press.
- Natsopoulos, D., Katsarou, Z., Bostantzopoulou, S., Grouis, G., Mentenopoulos, G., & Logothetis, J. (1991). Strategies in comprehension of relative clauses by Parkinsonian patients. *Cortex*, *27*, 255-268.
- Nebes, R. D. (1989). Semantic Memory in Alzheimer's Disease. *Psychological Bulletin*, *106*(3), 377-394.
- Nicholas, M., Obler, L. K., Albert, M. L., & Helm-Estabrooks, N. (1985). Empty speech in Alzheimer's disease and fluent aphasia. *Journal of Speech and Hearing Research*, *28*, 405-410.
- Obler, L. (1981). Review: *Le Langue des dements*. By Luce Irigaray. The Hague: Mouton, 1973. 357 pp. *Brain and Language*, *12*, 375-386.
- Perlman, G. (1980). Unixstat.
- Pinker, S. (1984). *Language learnability and language development*. (Vol. 7). Cambridge, MA: Harvard University Press.
- Pinker, S. (1991). Rules of language. *Science*, *253*, 530-535.
- Pinker, S. (1994). *The Language instinct*. New York: William Morrow.
- Plaut, D. C. (in press). Double dissociation without modularity: Evidence from connectionist neuropsychology. *Journal of Clinical and Experimental Neuropsychology*.
- Plunkett, K., & Marchman, V. (1991). U-Shaped learning and frequency effects in a multi-layered perceptron: Implications for child language acquisition. *Cognition*, *38*, 43-102.
- Prasada, S., & Pinker, S. (1993). Generalisation of regular and irregular morphological patterns. *Language and Cognitive Processes*, *8*(1), 1-56.
- Preuss, T. M. (1995). The argument from animals to humans in cognitive neuroscience. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 1227-1241). Cambridge, MA: MIT Press.
- Price, B. H., MD, Hakan Gurvit, M., Sandra Weintraub, P., Changiz Geula, P., Elizabeth Leimkuhler, P., & Marsel Mesulam, M. (1993). Neuropsychological patterns and language deficits in 20 consecutive cases of autopsy-confirmed Alzheimer's disease. *Archives of Neurology*, *50*, 931-937.

- Ramig, L. A., & Gould, W. J. (1986). Speech characteristics in Parkinson's disease. *Neurol. Consult.*, 4, 1-8.
- Rapcsak, S. Z., Arthur, S. A., Bliklen, D. A., & Rubens, A. B. (1989). Lexical agraphia in Alzheimer's disease. *Archives of Neurology*, 46, 65-68.
- Rapoport, S. I. (1991). Positron emission tomography in Alzheimer's disease in relation to disease pathogenesis: A critical review. *Cerebrovascular and Brain Metabolism Reviews*, 3, 297-335.
- Rapp, B. C., & Caramazza, A. (1995). Disorders of lexical processing and the lexicon. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences*. Cambridge, MA: MIT Press.
- Reiner, A., Albin, R. L., Anderson, K. D., D'Amato, C. J., Penney, J. B., & Young, A. B. (1988). Differential loss of striatal projection neurons in Huntington's disease. *Proceedings of the National Academy of Science, USA*, 85, 5733-5737.
- Rissenberg, M., & Glanzer, M. (1987). Free recall and word finding abilities in normal aging and senile dementia of the Alzheimer's type. *Journal of Gerontology*, 42, 318-322.
- Roos, R. A. C. (1986). Neuropathology of Huntington's chorea. In P. J. Vinken, G. W. Bruyn, & H. L. Klawans (Eds.), *Handbook of Clinical Neurology* (Vol. 5, pp. 315-326). Amsterdam: Elsevier.
- Rumelhart, D. E., & McClelland, J. L. (1986). On learning the past tenses of English verbs. In J. L. McClelland, D. E. Rumelhart, & PDP Research Group (Eds.), *Parallel distributed processing: Explorations in the microstructures of cognition* (Vol. 2, pp. 216-271). Cambridge, MA: MIT press.
- Sagar, H. J., Cohen, N. J., Sullivan, E. V., Corkin, S., & Growdon, J. H. (1988). Remote memory function in Alzheimer's and Parkinson's disease. *Brain*, 111, 185-206.
- Sahakian, B. J., Morris, R. G., Evenden, J. L., Heald, A., Levy, r., Philpot, M., & Robbins, T. W. (1988). A comparative study of visuospatial memory and learning in Alzheimer-type dementia and Parkinson's disease. *Brain*, 111, 695-718.
- Saint-Cyr, J. A., Taylor, A. E., & Lang, A. E. (1988). Procedural learning and neostriatal dysfunction in man. *Brain*, 111, 941-959.
- Sanes, J. N., Dimitrov, B., & Hallet, M. (1990). Motor learning in patients with cerebellar dysfunction. *Brain*, 113, 103-120.
- Schwartz, M., Saffran, E., & Marin, O. S. M. (1980). The word order problem in agrammatism I: Comprehension. *Brain and Language*, 10, 249-262.
- Schwartz, M. F., Linebarger, M. C., Saffran, E. M., & Pate, D. S. (1987). Syntactic transparency and sentence interpretation in aphasia. *Language and Cognitive Processes*, 2, 85-113.
- Schwartz, M. F., Marin, O. S. M., & Saffran, E. M. (1979). Dissociations of language function in dementia : A case study. *Brain and Language*, 7, 277-306.
- Sherman, J. C., & Schweickert, J. (1989). Syntactic and semantic contributions to sentence comprehension in Agrammatism. *Brain and Language*, 37, 419-439.
- Shoulson, I., & Fahn, S. (1979). Huntington's disease: Clinical care and evaluation. *Neurology*, 29, 1-3.
- Springer, J. E., & Isaacson, R. L. (1982). Catecholamine alteration in basal ganglia after hippocampal lesions. *Brain Research*, 252, 185-188.
- Squire, L. R., Knowlton, B., & Musen, G. (1993). The structure and organization of memory. *Annual Review of Psychology*, 44, 453-495.

Suzuki, W. A., & Amaral, D. G. (1994). Perirhinal and parahippocampal cortices of the macaque monkey: Cortical afferents. *Journal of Comparative Neurology*, 350, 497-533.

Taylor, A. E., Saint-Cyr, J. A., & Lang, A. E. (1986). Frontal lobe dysfunction in Parkinson's disease: The cortical focus of neostriatal outflow. *Brain*, 109, 845-883.

The Huntington's Study Group. (1996). The Unified Huntington's Disease Rating Scale: Reliability and consistency, *Movement Disorders* .

Tukey, J. W. (1977). *Exploratory data analysis*. Reading, MA: Addison-Wesley.

Tulving, E. (1983). *Elements of episodic memory*. Oxford: Clarendon Press.

Ullman, M. T. (1993). *The Computation of Inflectional Morphology*. Unpublished doctoral dissertation, Massachusetts Institute of Technology, Cambridge, MA.

Vonsattel, J.-P., MD, Richard H. Meyers, P., Thomas J. Stevens, B., Robert J. Ferrante, M., Edward D. Bird, M., & Edward P. Richardson, J., MD. (1985). Neuropathological classification of Huntington's disease. *Journal of Neuropathology and Experimental Neurology*, 44(6), 559-577.

Warburton, J. W. (1967). Memory disturbance and the Parkinson syndrome. *British Journal of Medical Psychology*, 40, 169-171.

Warrington, E. K. (1975). The selective impairment of semantic memory. *Quarterly Journal of Experimental Psychology*, 27, 635-657.

Warrington, E. K., & Shallice, T. (1984). Category specific semantic impairments. *Brain*, 107, 829-854.

Wernicke, C. (1874). The aphasic symptom. In R. S. C. a. M. W. Wartofsky (Ed.), *Boston studies in the philosophy of science* (Vol. 4,). Boston: Reidel.

Whitaker, H. (1976). A case of the isolation of the language function, *Studies in Neurolinguistics* (Vol. 1-2,). New York: Academic Press.

Xu, F., & Pinker, S. (1995). Weird past tense forms. *Journal of Child Language*, 22(3), 531-556.

Young, A. B., & Penney, J. B. (1993). Biochemical and functional organization of the basal ganglia, *Parkinson's disease and movement disorders* (pp. 1-11). Baltimore: Williams and Wilkins.

TABLES

Correlations for the 24 AD patients				
Existing Regular (<i>looked</i>)	Novel Regular (<i>plugged</i>)	Regularization of Novel Irregular (<i>crived</i>)	Irregularization of Novel Irregular (<i>crove</i>)	Existing Irregular (<i>dig</i>)
<i>looked</i>	r(19)=.78 p<.001	r(18)=.68 p=.001	r(19)=.16 p=.482	r(22)=.24 p=.265
<i>plugged</i>		r(18)=.86 p<.001	r(19)=.39 p=.077	r(19)=.07 p=.773
<i>crived</i>			NA	r(18)=-.03 p=.921
<i>crove</i>				r(19)=.35 p=.115
(a) Object naming				
	r(22)=.25 p=.235	r(19)=.24 p=.303	r(18)=.24 p=.303	r(19)=.41 p=.062
(b) Object naming, with IMC dementia scores partialled out				
	r(21)=.11 p=.607	r(18)=.08 p=.725	r(17)=.02 p=.940	r(18)=.47 p=.036
(c) Object naming vs rank ordered past tense production, with IMC dementia scores partialled out				
	r(21)=.24 p=.261	r(18)=.26 p=.267	r(17)=.12 p=.616	r(18)=.42 p=.063
(d) Fact retrieval				
	r(22)=-.31 p=.145	r(19)=-.28 p=.214	r(18)=-.25 p=.298	r(19)=-.17 p=.471
(e) Fact retrieval, with IMC dementia scores (excluding fact retrieval component) partialled out				
	r(21)=-.17 p=.429	r(18)=-.10 p=.687	r(17)=.08 p=.734	r(18)=-.26 p=.264
(f) Fact retrieval vs rank ordered past tense production, with IMC dementia scores partialled out				
	r(21)=-.23 p=.299	r(18)=-.11 p=.645	r(17)=.21 p=.396	r(18)=-.39 p=.088
(g) Recall of information 5 minutes after presentation (IMC subsection)				
	r(22)=-.01 p=.968	r(19)=0 p=.991	r(18)=-.20 p=.405	r(19)=.10 p=.657
				r(22)=-.34 p=.107

Table1: Correlation matrix for the 24 ADs among production rates of past tense types in the past tense production task, and between production rates of past tense types and object naming or fact retrieval. For regularizations of novel irregulars (*crived*), partial correlations are reported with irregularizations (*crove*) held constant. In this paper all p 's for r 's, t 's, and nonparametric difference tests, are reported as two-tailed.

The 5 most anomic AD patients								
Subject		JB	JE	EF	PJ	EP	AD m	NC m
n		1	1	1	1	1	5	14
Age		75	73	80	73	68	74	74
Education (yrs)		18	12	12	14	13	14	16
Language		Eng	Eng	Eng	Bil	Eng		
Sex		F	M	F	F	F		
Handedness		R	R	R	R	R		
Past Tense Production								
IMC dementia		18	11	21	20	12	16	1
Existing Regular	<i>(look)</i>							
Correct	<i>(looked)</i>	95%	100%	75%	75%	100%	89%	98%
Novel Regular	<i>(plag)</i>							
Correct	<i>(plagged)</i>	90%	85%	60%	NA	100%	84%	93%
Novel Irregular	<i>(crive)</i>							
Regularized	<i>(crived)</i>	89%	67%	39%	NA	94%	72%	67%
Irregularized	<i>(crove)</i>	11%	0%	17%	NA	0%	7%	27%
Existing Irregular	<i>(dig)</i>							
Correct	<i>(dug)</i>	69%	56%	81%	38%	56%	60%	96%
Overregular	<i>(diggd)</i>	19%	13%	13%	38%	38%	24%	1%
(as % of errors)	<i>(diggd)</i>	60%	29%	67%	60%	86%	60%	7%
Existing Regular vs Existing Irregular	<i>t (34)</i>	2.18	3.83	-.44	2.38	3.83	4.86	1.22
	<i>p</i>	.036	.001	.665	.023	.001	<.001	.229
Novel Regular vs Existing Irregular	<i>t (34)</i>	1.61	1.96	-1.37	NA	3.83	2.80	-1.19
	<i>p</i>	.116	.058	.179	NA	.001	.008	.243

Table 2: Background information and test results for the 5 most anomic AD patients (those with the worst object naming scores) and their normal control subjects (NC). Means and analyses for novel verbs are carried out over the 4 patients able to perform the task for novel verbs. For the control subjects, overregularizations as a percentage of irregular errors was calculated over only those subjects who made at least one error on irregulars.

Correlations for the 24 PD patients				
Existing Regular (<i>looked</i>)	Novel Regular (<i>plagged</i>)	Regularization of Novel Irregular (<i>crived</i>)	Irregularization of Novel Irregular (<i>crove</i>)	Existing Irregular (<i>dig</i>)
<i>looked</i>	r(26)=.85 p<.001	r(25)=.49 p=.009	r(26)=.09 p=.664	r(26)=.35 p=.067
<i>plagged</i>		r(25)=.63 p<.001	r(26)=-.11 p=.564	r(26)=.20 p=.316
<i>crived</i>			NA	r(25)=-.05 p=.789
<i>crove</i>				r(26)=.23 p=.236
(a) Right-side hypokinesia (RS)				
	r(26)=-.72 p<.001	r(26)=-.71 p<.001	r(25)=-.50 p=.008	r(26)=.04 p=.828
(b) RS, with IMC dementia scores partialled out				
	r(25)=-.64 p<.001	r(25)=-.63 p<.001	r(24)=-.39 p=.047	r(25)=-.03 p=.887
(c) Left-side hypokinesia (LS)				
	r(26)=-.66 p<.001	r(26)=-.69 p<.001	r(25)=-.46 p=.016	r(26)=-.06 p=.743
(d) LS, with IMC dementia scores partialled out				
	r(25)=-.55 p=.003	r(25)=-.59 p=.001	r(24)=-.34 p=.095	r(25)=-.15 p=.440
(e) RS, with LS partialled out				
	r(25)=-.41 p=.033	r(25)=-.34 p=.081	r(24)=-.23 p=.253	r(25)=.18 p=.370
(f) LS, with RS partialled out				
	r(25)=-.14 p=.493	r(25)=-.24 p=.229	r(24)=-.08 p=.691	r(25)=-.19 p=.353
(g) RS, with object naming scores partialled out				
	r(25)=-.67 p<.001	r(25)=-.66 p<.001	r(24)=-.40 p=.042	r(25)=.19 p=.334
(h) Object naming, with RS partialled out				
	r(25)=.17 p=.395	r(25)=.20 p=.315	r(24)=.21 p=.312	r(25)=.37 p=.061

Table 3: Correlation matrix for the 28 PDs among production rates of past tense types in the past tense production task, and between production rates of past tense types and right-side hypokinesia (RS), left-side hypokinesia (LS), or object naming. For regularizations of novel irregulars (*crived*), partial correlations are reported with irregularizations (*crove*) held constant.

The 5 most hypokinetic PD patients								
Subject		DC	RD	WL	PR	HT	PD m	NC m
n		1	1	1	1	1	5	14
Age		68	65	78	65	82	72	74
Education (yrs)		14	12	16	20	16	16	16
Language		Eng	Eng	Eng	Eng	Eng		
Sex		M	M	M	M	F		
Handedness		R	R	R	R	R		
Medication		Sinemet Artane	Sinemet	Artane	Sinemet Parlodel	Sinemet Parlodel		
Right-side hypokinesia		14	8.5	9	10	6.5	10	
IMC dementia		5	1	2	4	0	2	1
Object naming		75%	94%	81%	85%	93%	86%	89%
Past Tense Production								
Existing Regular	<i>(look)</i>							
Correct	<i>(looked)</i>	65%	95%	85%	55%	100%	80%	98%
Novel Regular	<i>(plag)</i>							
Correct	<i>(plagged)</i>	50%	80%	70%	50%	75%	65%	93%
Novel Irregular	<i>(crive)</i>							
Regularized	<i>(crived)</i>	50%	50%	39%	28%	22%	38%	67%
Irregularized	<i>(crove)</i>	6%	50%	6%	50%	67%	36%	27%
Existing Irregular	<i>(dig)</i>							
Correct	<i>(dug)</i>	75%	100%	100%	69%	94%	88%	96%
Overregular	<i>(digged)</i>	0%	0%	0%	0%	0%	0%	1%
(as % of errors)	<i>(digged)</i>	0%	NA	NA	0%	0%	0%	14%
Existing Irregular vs Existing Regular	<i>t</i> (34) <i>p</i>	.63 .531	.89 .379	1.63 .112	.83 .415	-1.12 .270	1.34 .190	-1.22 .229
Novel Irregular vs Existing Regular	<i>t</i> (34) <i>p</i>	1.54 .134	1.94 .060	2.55 .016	1.12 .270	1.51 .141	3.49 .001	1.19 .243

Table 4: Background information and test results for the five most hypokinetic PD patients and their control subjects (NC).

Retest of the hypokinetic PD patients						
Subject		DC	RD	PR	HT	PD m
n		1	1	1	1	4
IMC dementia		8	0	NA	0	NA
Right-side hypokinesia		13.5	6.5	14	7	10.3
Past Tense Production						
Irregular (21 items)	<i>(lost)</i>	81%	100%	67%	100%	87%
Regular (21 items)	<i>(passed)</i>	67%	95%	24%	95%	70%
Irregular vs	<i>t</i> (20)	1.0	1.0	2.9	1.0	3.01
Regular	<i>p</i>	.329	.329	.009	.329	.007
Irregular (6 items)	<i>(lost)</i>	100%	100%	67%	100%	92%
Regular (6 items)	<i>(passed)</i>	67%	83%	0%	83%	58%
Irregular vs	<i>t</i> (5)	1.6	1.0	3.2	1.0	3.01
Regular	<i>p</i>	.175	.363	.025	.363	.025
Repetition of Uninflected Words vs Past Tense Production of Regular Verbs						
Uninflected (41 items)	<i>(fast)</i>	93%	100%	95%	100%	97%
Regular (41 items)	<i>(passed)</i>	66%	95%	39%	98%	74%
Uninflected vs	<i>t</i> (40)	2.90	1.4	6.5	1.0	7.0
Regular	<i>p</i>	.006	.160	<.001	.323	<.001

Table 5: Results from the retest of 4 hypokinetic PDs, for purposes of testing the articulatory deficit and frequency accounts. IMC dementia and hypokinesia scores are from this retest session. In the new past tense production task, the 21 regular and 21 irregular items were matched on pronounceability. The 6 pairs of regular and irregular items were drawn from the list of 21 pairs, but were selected such that the regular verbs had a slightly *higher* mean past tense frequency than their matched irregulars. The 41 uninflected words for repetition-reading were matched one-to-one on pronounceability to the 41 regular items in the original (20) and new (21) past tense production tasks.

The 17 HD patients			
		HD m N=17	NC m N=8
Age		45	48
Education (yrs)		14	15
Years since onset		6	
Independence (100 normal, 10 min)		77	
Shoulson TFC (13 normal, 0 min)		7.1	
Physical Disability (100 normal, 10 min)		79	
Total chorea (0 none, 28 max)		10	
Hypokinesia (0 none, 4 max)		1.6	
IMC dementia (0 none, 38 max)		7	.5
Object naming		74%	91%
Past Tense Production			
Existing Regular	<i>(look)</i>		
Correct	<i>(looked)</i>	80%	99%
Multiple Suffixed	<i>(lookeded)</i>	5%	0%
Syllabic Suffixed	<i>(look-id)</i>	1%	0%
Unmarked	<i>(look)</i>	9%	0%
Novel Regular	<i>(plag)</i>		
Correct	<i>(plagged)</i>	72%	94%
Multiple Suffixed	<i>(plaggeded)</i>	4%	0%
Syllabic Suffixed	<i>(plag-id)</i>	4%	0%
Unmarked	<i>(plag)</i>	9%	0%
Novel Irregular	<i>(crive)</i>		
Regularization	<i>(crived)</i>	57%	63%
Multiple Suffixed	<i>(criveded)</i>	4%	1%
Syllabic Suffixed	<i>(crive-id)</i>	3%	0%
Irregularization	<i>(crove)</i>	20%	32%
Unmarked	<i>(crive)</i>	11%	4%
Existing Irregular	<i>(dig)</i>		
Correct	<i>(dug)</i>	76%	99%
Overregular	<i>(digged)</i>	8%	0%
(as % of errors)	<i>(digged)</i>	28%	0%
Multiple Suffixed	<i>(diggeded)</i>	1%	0%
Syllabic Suffixed	<i>(dig-id)</i>	.4%	0%
Unmarked	<i>(dig)</i>	6%	1%

Table 6: Background information and test results for the 17 HD patients and thier control subjects (NC). Means for novel verbs are calculated over the 15 patients who were able to perform the task for novel verbs. Overregularizations as a percentage of irregular errors were calculated over only those subjects who made at least one error on irregulars (15 of the 17 HDs, 2 of the 8 controls).

Correlations with inappropriately suffixed forms				
	Chorea	Inappropriately Suffixed Reg, Novel (e.g., <i>plaggeded</i>)	Overregular (<i>digged</i>)	Suffixed Irregular (e.g., <i>diggeded</i>)
(a) Years since disease onset	$r(15)=-.15$ $p=.557$	$r(15)=.35$ $p=.167$	$r(15)=-.06$ $p=.824$	$r(15)=.16$ $p=.528$
(b) Independence Scale	$r(15)=-.01$ $p=.957$	$r(15)=.05$ $p=.842$	$r(15)=-.13$ $p=.620$	$r(15)=-.04$ $p=.872$
(c) Shoulson Total Functional Capacity	$r(15)=-.13$ $p=.629$	$r(15)=.09$ $p=.730$	$r(15)=-.35$ $p=.175$	$r(15)=-.22$ $p=.401$
(d) IMC dementia	$r(15)=-.25$ $p=.329$	$r(15)=0$ $p=.990$	$r(15)=-.15$ $p=.577$	$r(15)=-.11$ $p=.688$
(e) Object naming	$r(15)=.11$ $p=.673$	$r(15)=.21$ $p=.420$	$r(15)=-.24$ $p=.351$	$r(15)=-.09$ $p=.736$
(f) Physical Disability	$r(15)=-.10$ $p=.699$	$r(15)=0$ $p=.990$	$r(15)=-.20$ $p=.442$	$r(15)=-.14$ $p=.601$
(g) Hypokinesia	$r(15)=-.23$ $p=.368$	$r(15)=-.16$ $p=.528$	$r(15)=.01$ $p=.983$	$r(15)=-.14$ $p=.603$

Table 7: Correlation matrix for the 17 HDs between, on the one hand, chorea and various measures of inappropriately suffixed forms, and on the other hand, a variety of measures of disease progression and of behavioral and motor impairments.

Correlations with unmarked forms				
	Unmarked Novel Regular (<i>plag</i>)	Unmarked Existing Regular (<i>look</i>)	Unmarked Existing Irregular (<i>dig</i>)	Unmarked Attracted Regular (<i>glide</i>)
(a) Hypokinesia	r(13)=.66 <i>p</i> =.007	r(15)=.46 <i>p</i> =.064	r(15)=.42 <i>p</i> =.091	r(13)=.32 <i>p</i> =.244
(b) Hypokinesia, with chorea partialed out	r(12)=.69 <i>p</i> =.007	r(14)=.55 <i>p</i> =.028	r(1)=.49 <i>p</i> =.056	r(12)=.32 <i>p</i> =.266
(c) Hypokinesia, with chorea and object naming partialed out	r(11)=-.63 <i>p</i> =.021	r(13)=.44 <i>p</i> =.101	r(13)=.17 <i>p</i> =.544	r(11)=.07 <i>p</i> =.809
(d) Hypokinesia, with chorea and IMC dementia scores partialed out	r(11)=.58 <i>p</i> =.037	r(13)=.46 <i>p</i> =.085	r(13)=.21 <i>p</i> =.453	r(11)=.24 <i>p</i> =.425
(e) Chorea	r(13)=.03 <i>p</i> =.907	r(15)=.25 <i>p</i> =.336	r(15)=.18 <i>p</i> =.491	r(13)=-.04 <i>p</i> =.881
(f) Chorea, with hypokinesia partialed out	r(12)=.24 <i>p</i> =.413	r(14)=.41 <i>p</i> =.113	r(14)=.32 <i>p</i> =.234	r(12)=.03 <i>p</i> =.931

Table 8: Correlation matrix for the 17 HDs between the production of unmarked forms for four verb classes, and measures of hypokinesia or chorea. For novel regulars Spearman's nonparametric rank order correlations are used because of the outlier patient; similar results were obtained with this patient eliminated, using Pearson's parametric correlations (see text).