doi:10.1093/brain/awh387 *Brain* (2005), **128**, 584–596

The basal ganglia and rule-governed language use: evidence from vascular and degenerative conditions

C. E. Longworth,[1] S. E. Keenan,[2] R. A. Barker,[3] W. D. Marslen-Wilson[4] and L. K. Tyler[1]

Departments of [1]Experimental Psychology and [2]Psychiatry, University of Cambridge, [3]Cambridge Centre for Brain Repair, Addenbrooke's Hospital, University of Cambridge, and [4]MRC Cognition and Brain Sciences Unit, Cambridge, UK

Correspondence to: L. K. Tyler, Centre for Speech and Language, Department of Experimental Psychology, University of Cambridge, Cambridge CB2 3EB, UK
E-mail: lktyler@csl.psychol.cam.ac.uk

Summary

The Declarative/Procedural Model of Pinker, Ullman and colleagues claims that the basal ganglia are part of a fronto-striatal procedural memory system which applies grammatical rules to combine morphemes (the smallest meaningful units in language) into complex words (e.g. talk-ed, talk-ing). We tested this claim by investigating whether striatal damage or loss of its dopaminergic innervation is reliably associated with selective regular past tense deficits in patients with subcortical cerebrovascular damage, Parkinson's disease or Huntington's disease. We focused on past tense morphology since this allows us to contrast the regular past tense (jump-jumped), which is rule-based, with the irregular past tense (sleep-slept), which is not. We used elicitation and priming tasks to test patients' ability to comprehend and produce inflected forms. We found no evidence of a consistent association between striatal dysfunction and selective impairment of regular past tense morphology, suggesting that the basal ganglia are not essential for processing the regular past tense as a sequence of morphemes, either in comprehension or production, in contrast to the claims of the Declarative/Procedural Model. All patient groups showed normal activation of semantic and morphological representations in comprehension, despite difficulties suppressing semantically appropriate alternatives when trying to inflect novel verbs. This is consistent with previous reports that striatal dysfunction spares automatic activation of linguistic information, but disrupts later language processes that require inhibition of competing alternatives.

Keywords: striatum; Huntington's disease; Parkinson's disease; cerebrovascular; language

Abbreviations: BDI = Beck Depression Index; CANTAB = Cambridge Neuropsychological Test Automated Battery; CSL = Centre for Speech and Language; CVA = cerebrovascular accident; MMSE = Mini-Mental State Examination; NART = National Adult Reading Test; UHDRS = Unified Huntington's Disease Rating Scale; UPDRS = Unified Parkinson's Disease Rating Scale; WMS = Wechsler Memory Scale

Received April 30, 2004. Revised December 9, 2004. Accepted December 10, 2004. Advance Access publication January 19, 2005

Introduction

Aphasic deficits associated with subcortical damage raise the controversial and unresolved issue of the role of the basal ganglia in language function. Atypical, often transient, aphasic symptoms have been reported following cerebrovascular damage to the basal ganglia (e.g. Brunner *et al*., 1982; Damasio *et al*., 1982; Naeser *et al*., 1982; Wallesch *et al*., 1983; Cappa, 1997). Language impairments have also been reported for neurodegenerative diseases of the basal ganglia, such as Parkinson's disease with its associated loss of the dopaminergic nigrostriatal pathway (e.g. Cummings *et al*., 1988; Illes *et al*., 1988; Illes, 1989) and Huntington's disease, which in the early stages has pathology that targets the striatum (e.g. Gordon and Illes, 1987). The characterization of language disorder accompanying striatal dysfunction and its neural basis, which may reflect either subcortical damage or concomitant cortical dysfunction, constitutes a major challenge for aphasiology.

Several theories propose the involvement of the basal ganglia in what might be termed executive semantic functions, either as part of a neural system regulating the release or

selection of cortically generated lexical items for production after semantic monitoring (Crosson, 1985; Wallesch and Papagno, 1988) or in the selective attentional engagement of the semantic network (Copland, 2003). These theories account for several abnormalities following subcortical cerebrovascular damage, such as the occurrence of semantic paraphasias (e.g. Damasio *et al*., 1982) and the presence of an abnormally extended N400 component in the event-related brain potential, assumed to reflect processes of semantic integration (Kotz *et al*., 2003). They may also explain the inability of Parkinson's disease patients to suppress the infrequent meaning of homophones in semantic priming, despite normal automatic access to semantic information (Copland, 2003).

Theoretical accounts also link the basal ganglia with syntactic processing as part of fronto-striatal circuitry either providing the executive resources required to comprehend complex syntax (Grossman, 1999) or to process syntactic rules (Lieberman *et al*., 1990; Natsopoulos *et al*., 1991, 1993; McNamara *et al*., 1996). However, although patients with Parkinson's disease have difficulties interpreting non-canonical word order in untimed tasks (e.g. Natsopoulos *et al*., 1993; McNamara *et al*., 1996; Kemmerer, 1999; Grossman *et al*., 2000), they show intact automatic access to syntactic information in reaction time experiments (Grossman *et al*., 2002). This is consistent with considerable electrophysiological evidence suggesting that the role of the striatum is restricted to a late stage of syntactic processing. Friederici and colleagues have reported that the event-related brain potential of patients with subcortical lesions or Parkinson's disease shows a normal early left anterior negativity, hypothesized to index automatic syntactic parsing, in response to syntactic violations; however, a late centro-parietal positivity (the P600 component) hypothesized to index late integration of syntactic and semantic information is either absent (Friederici *et al*., 2003; Frisch *et al*., 2003; Kotz *et al*., 2003) or reduced (Friederici *et al*., 1999).

There are similarities between the semantic and syntactic impairments shown by patients with striatal dysfunction. Automatic activation of semantic and syntactic information appears to be intact, but later processes that revise initial linguistic representations are impaired. Although this may indicate separate but parallel deficits in semantic and syntactic processing, it is also possible that both reflect a general impairment of inhibition. Support for this comes from Grossman *et al*. (2002), who found that impaired comprehension of complex syntax in untimed tasks correlated with a measure of processing speed for planning and inhibition in patients with Parkinson's disease. Striatal involvement in inhibition, postulated not only in motor control (Mink, 1996) but also cognition (Lawrence *et al*., 1998), is supported by neuroimaging evidence that the striatum is activated by inhibition in masked priming (Aron *et al*., 2003) and reversal learning (Cools *et al*., 2002). One hypothesis for a restricted, non-language specific role of the striatum in language is therefore that it suppresses competing alternatives in the late integrational processes of language comprehension and, possibly, in the lexicalization stage of language production, given evidence of semantic paraphasias in patients with subcortical lesions.

The Declarative/Procedural Model of Ullman, Pinker and colleagues (Ullman *et al*., 1997; Ullman, 2001; Pinker and Ullman, 2002) presents an important alternative to this non-language specific, inhibitory role for the striatum in language processing. It proposes that the basal ganglia are part of a neural system controlling rule-governed behaviour, including the application of grammatical rules to combine morphemes (the smallest meaningful units in language) into complex words (e.g. jump-ed, jump-ing). This model and its forerunner, the Words and Rules theory (e.g. Pinker, 1991), propose that language processing requires two distinct mechanisms: (i) one that stores arbitrary information in associative memory; and (ii) another that combines stored units to compute larger sequences.

Processing the English past tense has often been used to test this type of dual mechanism account (see Marslen-Wilson and Tyler, 1997, 1998; Tyler *et al*., 2002) because it contrasts the highly predictable –ed suffixation procedure of the regular past tense, with the idiosyncrasy of irregular past tense forms (e.g. bring/brought, but wring/wrung and ring/rang). Dissociations between performance on the regular and irregular past tense in development (e.g. Berko, 1958), in adults (Marslen-Wilson *et al*., 1993) and a range of neuropsychological disorders (e.g. Ullman *et al*., 1997, 2005; Tyler *et al*., 2002) support the case for distinct underlying mechanisms. The Declarative/Procedural Model aligns these mechanisms with the distinction between procedural and declarative memory (Cohen and Squire, 1980), proposing that a fronto-striatal procedural memory system subserves grammatical rule use, whereas a temporo-parietal declarative memory system subserves explicit memory of arbitrary facts. Grammatical rules—such as adding -ed to form the regular past tense in language production, or analysing a regular past tense as a stem plus a suffix in language comprehension—are viewed as cognitive skills, by analogy to the over-learned motor behaviours controlled by procedural memory. In contrast, a temporo-parietal declarative memory system is proposed to subserve the mental lexicon, including stored irregular past tense forms.

In support of their claims Ullman *et al*. (1997) found a correlation between difficulties producing the regular past tense and right-sided (left hemispheric driven) hypokinesia in 28 patients with Parkinson's disease without severe dementia, the majority of whom were receiving levodopa medication. The five most hypokinetic patients showed a trend towards better performance for irregular than regular inflection, and were significantly better at producing irregular past tense forms than they were at adding the regular inflection to nonsense words. This was interpreted as evidence that the suppression of motor activity in Parkinson's disease is associated with the suppression of grammatical rule use. Conversely, Ullman and colleagues suggested that basal

ganglia damage leading to excess motor activity is associated with overactive grammatical rule use. They found that 17 patients diagnosed with Huntington's disease according to family history and symptomatology made significantly more over-regularization errors (e.g. *digged) than controls. A measure of chorea correlated with the number of over-regularization errors and the number of unusual, multiply or syllabically suffixed words produced (e.g. *jumpeded), although performance on regular (80% correct) and irregular (76% correct) verb inflection was similar. It was not reported whether or not these patients were genetically proven to have Huntington's disease.

Although this evidence suggests that damage to the basal ganglia may be associated with difficulties producing regular verb inflections, it remains unclear whether or not the striatum is an essential component of a rule-governed language system and more specifically of a network subserving overt inflectional affixation. Cerebrovascular patients with selective regular past tense deficits typically have damage to the perisylvian cortex in addition to any subcortical lesions (e.g. Ullman *et al*., 1997; Tyler *et al*., 2002). Damage to the basal ganglia in neurodegenerative disorders also has consequences for cortical regions, due not only to their role in cortico-striato-thalamic loops but also the fact that both Parkinson's disease and Huntington's disease have pathology within the neocortex. Thus, it is necessary to test whether the association between basal ganglia pathology and impairments of regular inflectional morphology implies a causal connection or the effects of concomitant cortical dysfunction.

In our current research, we tested whether the basal ganglia are essential components of the network subserving regular inflectional morphology by investigating whether disorders primarily of the basal ganglia are reliably associated with selective regular past tense deficits. We used the past tense elicitation tests of Ullman *et al*. (1997) and Tyler *et al*. (2002) to examine past tense production and the primed lexical decision (word/non-word judgements) experiment of Tyler *et al*. (2002) to assess the comprehension of spoken past tense forms. The primed lexical decision experiment of Tyler *et al*. (2002) has previously proved adequate to demonstrate a dissociation between patients with perisylvian infarcts, who showed impaired comprehension of spoken regular past tense morphology, and patients with damage to the inferior temporal cortex, who showed impairments specific to irregular past tense morphology and semantically related words. If basal ganglia damage is reliably associated with selective regular past tense deficits, this would suggest that the basal ganglia are essential components of a network subserving regular inflectional morphology, supporting a direct role for the striatum in rule-governed language, rather than a restricted, non-language specific role in late inhibitory processes. However, if there is no consistent pattern of regular past tense deficits associated with basal ganglia damage, this would suggest that cortical regions, rather than the basal ganglia, are essential for the processing involved.

Material and methods

This research was approved by the Cambridge Local Research Ethics committee and consent was obtained according to the Declaration of Helsinki. Three groups of patients participated: (i) seven patients with subcortical cerebrovascular damage; (ii) 15 patients with Parkinson's disease (who fulfilled the UK Parkinson's Disease Society Brain Bank criteria for idiopathic Parkinson's disease); and (iii) 10 patients with genetically proven Huntington's disease. These were compared with eight healthy control subjects, who were recruited from the participant panel of the MRC Cognition and Brain Science Unit, Cambridge, to match the patients in terms of age (mean age = 54.5 years, SD = 4.2) and sex ratio (six males, two females). All participants were native speakers of UK English, who reported no premorbid language difficulties.

The cerebrovascular patients were recruited from the Cambridge Cognitive Neuroscience Research Panel on the basis of a CT report of a cerebrovascular accident (CVA) involving the left basal ganglia. In most cases, the cerebrovascular damage extended to cortical regions, but as cortical lesions varied both in location and extent across patients, the only thing they shared in common was damage to the striatum (see Table 1).

The Parkinson's disease patients were diagnosed as having idiopathic Parkinson's disease and assessed using standard rating scales including the Unified Parkinson's Disease Rating Scale (UPDRS) (Fahn *et al*., 1987), Schwab and England (1969) and Hoehn and Yahr (1967) staging (see Table 2A). They were all currently receiving L-dopa medication and some were also taking dopamine receptor agonists (10 patients), tricyclic antidepressants (six patients), selective serotonin re-uptake inhibitors (SSRI) (two patients), selective norepinephrine re-uptake inhibitors (SNRI) (one patient), benzodiazepines (three patients) and/or thyroxine (one patient). All patients were tested in the ON state. None of the patients showed evidence of dementia on the Mini-Mental State Examination (MMSE) (Folstein *et al*., 1975) or depression on the Beck Depression Inventory (BDI) (Beck *et al*., 1961). They were divided into two groups, mild or moderate Parkinson's disease, based on their overall performance on the UPDRS (mild: mean UPDRS = 37, SD = 9.2; moderate: mean UPDRS = 47.9, SD = 18.8) and their cognitive ability on a variety of neuropsychological assessments including the MMSE, National Adult Reading Test (NART) (Nelson, 1982) and subsets of the CANTAB battery (Robbins *et al*., 1994) (see Table 2B). The fact that the moderate Parkinson's disease patients were older and had suffered from Parkinson's disease for less time than the mild Parkinson's disease patients implies that they may have a more aggressive form of the disease.

Genetically proven Huntington's disease patients were assessed using standard protocols such as the Unified Huntington's Disease Rating Scale (UHDRS) (Kieburtz and the Huntington Study Group, 1996), motor evaluation, total functional capacity and independence score. No patient showed evidence of dementia on the MMSE (Folstein *et al*., 1975), but their scores on the BDI were consistent with mild depression (see Table 3). Three patients were unmedicated; of the others, some were receiving dopamine antagonists (four patients), benzodiazepine medication (one patient), SSRI (five patients), SNRI (two patients) and/or tricyclic (one patient) antidepressants. All medicated patients were tested whilst they were taking their medication. The patients were divided into two groups with mild or moderate Huntington's disease on the basis of the UHDRS (see Table 3).

Table 1 *Summary of demographic and neuropsychological characteristics for the cerebrovascular patients*

Patient	Aetiology	Lesion location	Age	Sex	Handedness	Years post-onset	WMS forward digit span	WMS backward digit span	RCPM
MJ	Embolic left hemispheric CVA	Left basal ganglia and subinsula	28	M	R	6	4	3	33/36
SC	Subarachnoid haemorrhage/ anterior communicating artery aneurysm	Left basal ganglia, bilateral frontopolar cortex (BA 10) and cingulate gyri.	32	M	R	3	7	5	33/36
MB	Subarachnoid haemorrhage/ anterior communicating artery aneurysm	Left caudate, internal capsule, orbitofrontal cortex and anterior temporal lobe	39	F	R	4	7	6	34/36
SG	Left intracerebral thalamic haemorrhage with extension into ventricular system	Left globus pallidus and internal capsule	51	M	R	4	6	4	31/36
DF	Left CVA during coronary artery bypass surgery	Left basal ganglia, internal capsule, right parietal and frontal cortices	54	M	L	10	8	6	Not tested
IT	Left anterior and posterior communicating artery aneurysms	Left basal ganglia, temporal and parietal cortices	56	M	R	4	6	3	35/36
CC	Subarachnoid haemorrhage/left MCA aneurysm	Left basal ganglia and anterior inferior frontal cortex (BA 47)	67	M	R	1	4	3	Not tested

BA = Brodmann area; L = left; R = right; RCPM = Raven's Coloured Progressive Matrices (Raven *et al*., 1999); WMS = Wechsler Memory Scale.

Table 2A *Demographic and clinical characteristics of the mild and moderate Parkinson's disease groups*

	n	Sex ratio (M:F)	Age (years)	Duration of disease (years)	NART IQ	UPDRS	Hoehn and Yahr scale	ADL	BDI
Mild	8	6:2	56 (7.0)	11.9 (5.6)	116.1 (8.3)	34.4 (12.7)	2.0 (0.5)	80 (13.1)	7.9 (4.7)
Moderate	7	7:0	66.1 (3.7)	5.7 (5.0)	102.4 (10.8)	47.9 (18.8)	2.2 (0.7)	71.4 (19.5)	7.7 (3.5)

Data represent mean (SD) values. ADL = Schwab and England activities of daily living scale.

Table 2B *Neuropsychological performance of the mild and moderate Parkinson's disease groups*

	MMSE	Verbal fluency	Category fluency	WMS forward digit span	WMS backward digit span	CANTAB pattern recognition	CANTAB spatial recognition
Mild	29.5 (0.8)	49.6 (8.8)	25.0 (6.5)	7.4 (0.5)	5.6 (1.5)	21.5 (1.3)	16.1 (2.1)
Moderate	27.4 (0.8)	25.3 (3.4)	15.1 (3.0)	6.1 (0.7)	3.9 (0.4)	17.9 (2.1)	12.7 (1.6)
P-value	<0.005	<0.001	<0.005	<0.005	<0.005	<0.05	<0.05

Data represent mean (SD) values.

Past tense production

Two untimed past tense elicitation tests (Ullman *et al*., 1997; Tyler *et al*., 2002) were used to investigate whether pathological disorders involving the basal ganglia could be reliably associated with regular past tense deficits in production. Both tests embed regular or irregular verb targets into a two-sentence context. The second sentence of each pair is incomplete and requires a past tense form of the target verb. For example: ''My nose sometimes bleeds. Last night it . . .'' The Ullman elicitation test (Ullman *et al*. 1997) consists of 20 regular, 20 irregular and 20 novel (e.g. *prag) verb targets. The participant reads aloud randomly ordered sentence pairs and is asked to supply the missing word. The Centre for Speech and Language (CSL) elicitation test (Tyler *et al*. 2002) has 26 regular and 27 irregular verb targets, matched for lemma and word form frequencies as verbs using the Celex lexical database (Baayen *et al*., 1995). The procedure is the same as in the Ullman test except that, to avoid confounding morphological deficits with acquired dyslexias, the participant listens to the randomly ordered sentence pairs rather than reading aloud. Healthy controls typically find both tests easy and perform at, or near, ceiling (Ullman *et al*., 1997; Tyler *et al*., 2002).

Table 3A *Demographic and clinical characteristics of the mild and moderate Huntington's disease patients*

	n	Sex ratio (M:F)	Age (years)	Duration of disease (years)	NART IQ	UHDRS	Shoulson and Fahn (1979) independence scale	BDI
Mild	7	5:2	49.7 (6.2)	7.0 (4.2)	113.0 (8.1)	28.0 (13.5)	80.7 (16.9)	11.7 (9.7)
Moderate	AG	M	71	12	93	48	75	9
	AS	M	41	10	103	87	60	3
	SB	F	71	28	118	69	60	12

Data represent mean (SD) values. BDI data were available for three mild Huntington's disease patients.

Table 3B *Neuropsychological performance of the mild and moderate Huntington's disease patients*

Patient	MMSE	Verbal fluency	Category fluency	WMS forward digit span	WMS backward digit span	CANTAB pattern recognition	CANTAB spatial recognition
Mild (n = 7)	26.3 (2.5)	30.8 (9.6)	15.8 (6.8)	6.1 (1.2)	3.9 (0.7)	18.0 (2.6)	13.7 (3.8)
AG	25	5	10	4	3	16	12
AS	16	4	5	4	2	14	10
SB	18	Not tested	Not tested	5	2	Not tested	Not tested

Data represent mean (SD) values.

Past tense comprehension

We used a paired auditory priming and lexical decision experiment previously used to demonstrate selective regular past tense deficits in cortical CVA patients (Marslen-Wilson and Tyler, 1997, 1998; Tyler *et al*., 2002) to investigate whether pathological disorders involving the basal ganglia could be reliably associated with deficits in the automatic processing of spoken regular past tense forms. This paradigm requires participants to make speeded lexical decision responses (i.e. word/non-word judgements) to spoken targets preceded by related or unrelated prime words. Healthy volunteers show response facilitation, or priming, following morphologically (e.g. jumped/jump or taught/teach) and semantically (e.g. skirt/dress) related primes, but not for spoken words related only by form (e.g. gravy/grave) (Marslen-Wilson and Tyler, 1997; Tyler *et al*., 2002). The Words and Rules theory suggests that morphological priming for regular and irregular verbs depends on different mechanisms: spoken regular past tense forms are analysed into stems and inflections (e.g. walk + -ed) so that the stem primes itself, whereas irregular past tenses are associated with their stems (Pinker, 1997). Impaired ability to decompose regular past tense forms into stems and suffixes results in a selective failure of priming (e.g. Marslen-Wilson and Tyler, 1997, 1998; Tyler *et al*., 2002). Recent versions of the Words and Rules theory (e.g. Berent *et al*., 1999) and the related Declarative/Procedural Model (Pinker and Ullman, 2002) suggest that regularly inflected forms that sound similar to existing irregular verbs, or that are frequently used, are likely to be memorized in full and thus do not require decomposition into stems and inflections. For this reason, it is important to examine priming for regular past tense forms that are infrequently encountered, since these are the most likely to be decomposed into stems and inflections.

The experiment consisted of four conditions:

(i) & (ii) 42 regular and 42 irregular past tense forms and their related verb stems (e.g. jumped/jump or taught/teach), excluding no-change verbs and suppletive forms;

(iii) a phonologically related comparison condition consisting of 24 words that share the initial syllable of the target but which are not semantically or morphologically related to it (e.g. gravy/grave);

(iv) a semantically related comparison condition consisting of 24 words related to the target in meaning, but not phonology or morphology (e.g. skirt/dress).

Each prime word was matched to a control word in frequency and number of syllables. The control primes for past tense conditions were unrelated verbs and nouns inflected for third person singular or number (e.g. -s inflection). Details of item composition and matching are given by Tyler *et al*. (2002). As word pairs in the two past tense conditions are related in both form and meaning, the two comparison conditions allow us to test priming for words that are only related by semantics or phonology. A range of filler items was selected to ensure that semantic relationships or verb primes could not be used to predict the lexicality of targets, thus reducing the possibility of strategic processing confounds. To this end there were 80 unrelated prime/target real word fillers, equally divided between simple primes with regularly inflected targets (-ed and -s inflections), -s inflected primes (nouns and verbs) with uninflected targets, and uninflected prime/target pairs. There were 212 phonotactically legal non-word foils.

The materials were divided into two versions of the experiment, balanced so that all targets appeared once in each version, preceded by either a related or unrelated prime. In total all versions had 472 trials: 30 practice trials, 18 warm-up trials, 66 related trials, 66 unrelated trials and 292 filler/foil trials. There were an equal number of word and non-word targets in each version and in each experimental block. Related experimental items made up 31% of the word targets. These were pseudo-randomly distributed throughout the list, with the same order of experimental and filler items in both versions.

All stimuli were recorded by a native speaker of English and digitized for binaural presentation over headphones. There was an interval of 250 ms between the prime and target stimuli and, following a response, the next trial was presented after an interval of 1500 ms. The experiment was administered on a laptop computer

using DMDX experimental software (Forster and Forster, 2003). Word pairs were played over headphones and subjects had 4 s in which to make a lexical decision via a two-button response-box. Subjects were instructed to decide whether the word target was a real word in English and to press the button marked either 'word' or 'non-word' accordingly. Reaction times were measured from target onset. Each version of the experiment took ~45 minutes to complete. Participants carried out both versions in the same order over two testing sessions, with at least 4 weeks between the two testing sessions to minimize the risk of episodic memory confounds.

Results

Past tense production

The accuracy of performance on the two elicitation tasks is presented in Fig. 1. One item of the Ullman elicitation test (the irregular homophone wring/wrung) was removed from analyses because it proved problematic for all groups, including control volunteers. In line with Ullman *et al*. (1997), healthy control volunteers performed at or near ceiling in all conditions. Impairment was defined as performance below, or equal to 2 SD beneath the control mean for each condition. The relationship between incidence of impairments and condition (e.g. regular, irregular and novel past tense elicitation) is shown in Table 4. Although not all the patients show impairment at this standard cut-off, a substantial number do, suggesting that these patients tend to have difficulty with past tense elicitation tasks. However, this impairment is approximately as strong for the irregulars as the regulars, and the following statistical analysis will address this issue.

The Fisher exact test, a statistical test of association suitable for small cell sizes, was used to test whether the incidence of impairment in each group was associated with condition. One-tailed significance was used to test the directional hypothesis that there is higher incidence of impairment for regular and novel past tense than irregular. The results for CVA patients, mild and moderate Parkinson's disease patients, and moderate Huntington's disease patients showed no evidence that inflecting regular or novel verbs was associated with a significantly higher incidence of impairment than inflecting irregular verbs ($P > 0.05$ in all pairwise comparisons for each group; Table 4). The mild Huntington's disease patients were the only group to show any evidence of a higher incidence of impairment for regular inflectional morphology and this was inconsistent across tasks. In the CSL task, they showed a significant association between impairment and condition, with a higher incidence of impairment for regular verbs than irregular ($P = 0.051$). However, in the Ullman task, there was no evidence that the incidence of impairment differed between regular and irregular verb inflections ($P = 0.28$) or between novel and irregular verb inflections ($P = 0.28$). In sum, the results show no evidence for a consistent association between impairment and condition to suggest a selective deficit in applying the –ed inflection to regular or novel verbs in patients with striatal dysfunction.

Right-lateralized hypokinesia was measured for Parkinson's disease patients using the four hand and foot movement sub-tests of the UPDRS, as in Ullman *et al*. (1997). There was no evidence of significant differences in the degree of right-sided hypokinesia between patients with and without impairments in regular past tense elicitation, in either the Ullman test [mean score out of 16 for right-sided hypokinesia = 5.75 (SD = 3.46) with impairment; 3.57 (SD = 2) without impairment; $t(13) = 1.46$, $P = 0.168$] or the CSL test [mean score out of 16 for right-sided hypokinesia = 4.88 (SD = 3.57) with impairment; 4.57 (SD = 2.46) without impairment; $t(13) = 0.19$, $P = 0.85$]. There was a trend towards a small difference in the degree of right-sided hypokinesia between patients with and without impairments in novel verb inflection in the Ullman test [mean score out of 16 for right-sided hypokinesia = 5.89 (SD = 2.88) with impairment; 3 (SD = 2.43) without impairment; $t(13) = 2.018$, $P = 0.065$].

Tables 5 and 6 summarize the average percentage of correct first responses and errors in each group for the Ullman and CSL tests, respectively. The controls made a small number of errors, including repeating, rather than inflecting the cue, substituting another verb instead of the target (both for existing and novel forms) and regularizing irregular past tenses (e.g. slitted).

CVA patients also made a small number of repetition, substitution and regularization errors overall. However, one patient accounted for all unmarked errors (e.g. jump instead of jumped) in the CSL test, 60% of regularization errors (e.g. digged instead of dug) for the irregular condition of the CSL test and 95% of lexical intrusion errors (e.g. stroked instead of prapped) for the novel condition of the Ullman test. This patient performed at floor for novel verb inflection as he substituted an existing past tense form for every novel target. This may indicate an impairment in applying the regular past tense 'rule' to novel verbs. However, the patient cannot be said to have a selective deficit in regular inflectional morphology since he performed below control norms on all conditions (Ullman test: regular verbs: 80%; irregular verbs: 73.7%; novel verbs: 0%; CSL test: regular verbs: 96%; irregular verbs: 70%) and produced correctly inflected forms of semantically appropriate verbs for 90% of novel verbs (40% irregular, 50% regular), including four low frequency regular past tense forms.

Mild Parkinson's disease patients made relatively few errors. Some of these were similar to those made by controls, for example, repeating the cue or regularizing irregular verbs, but there were a few errors unlike those of controls, where the phonology of the verb was distorted (e.g. statched instead of satched). Moderate Parkinson's disease patients were much more clearly impaired across all conditions. In contrast to mild Parkinson's disease patients, they showed an increased tendency to repeat real word cues (e.g. 3.57% and 2.26% for regular and irregular verbs presented as stem cues on the Ullman test) or to inflect semantically related verbs (e.g. 4.4% for regular verbs on the CSL test and 7.52% for irregulars on the Ullman test). They also showed an increased

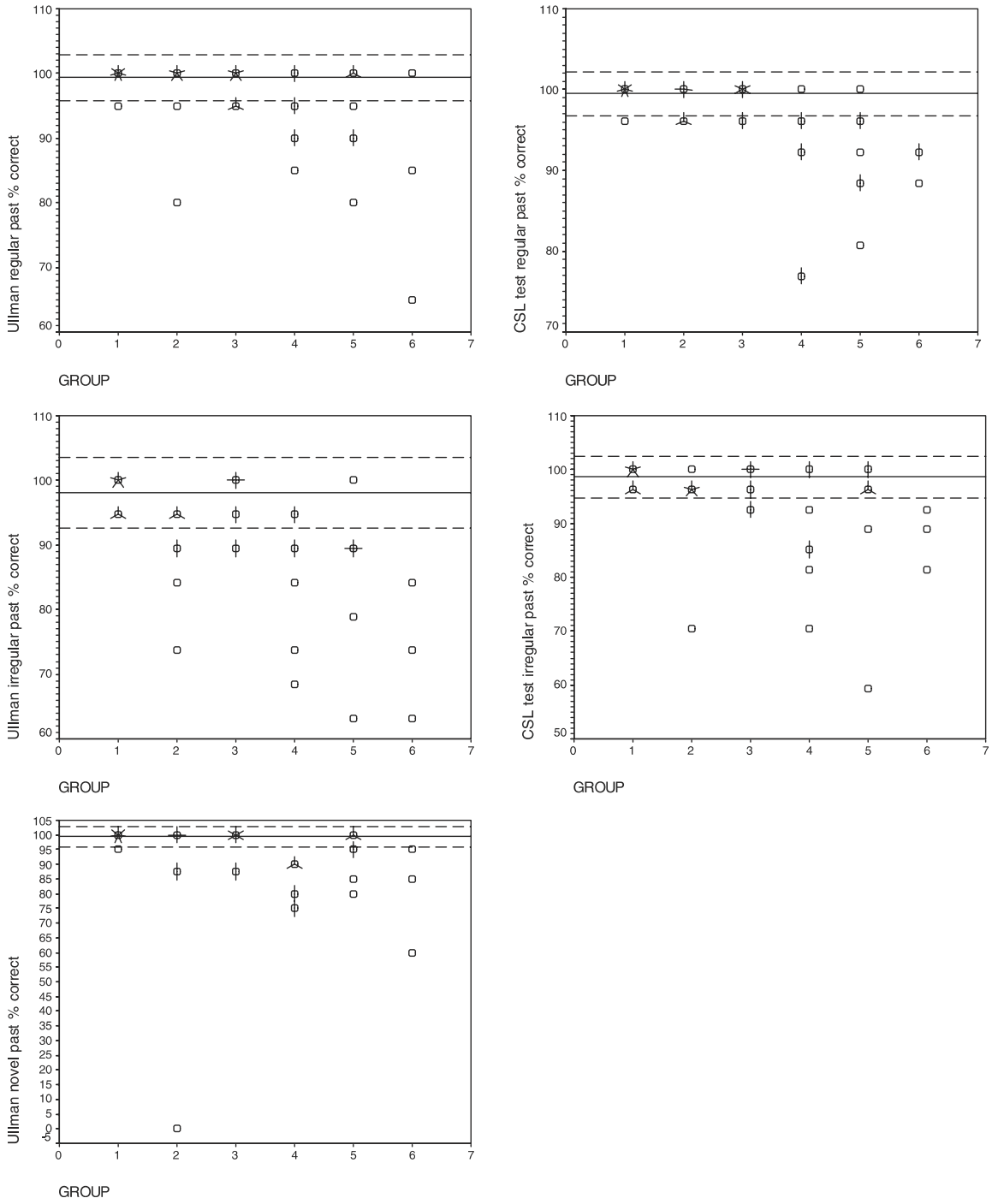

Fig. 1 Percentage correct on first responses to past tense elicitation (Left = Ullman past tense elicitation test and Right = CSL past tense elicitation test). Group: 1 = healthy controls; 2 = CVA; 3 = mild Parkinson's disease; 4 = moderate Parkinson's disease; 5 = mild Huntington's disease; 6 = moderate Huntington's disease. A circle represents each data point and the number of petals represents the number of cases at each data point. Solid lines indicate mean accuracy of the control group and dashed lines indicate two standard deviations above and below the control mean [Ullman test: regular: 99.38% (SD = 1.77%); irregular: 98.03% (SD = 2.72%); novel 99.38% (SD = 1.77%); CSL test: regular: 99.52% (SD = 1.36%); irregular: 98.61% (SD = 1.92%)].

tendency to make correctly inflected, phonological distortion and lexical intrusion errors for novel verbs. These may be signs of a general phonological output disorder in Parkinson's disease that increases with disease progression and which is independent of morphological processing.

Mild Huntington's disease patients showed a tendency to repeat cues in all conditions (e.g. 2.14%, 3.76% and 1.43% for regular, irregular and novel verbs presented as stem cues in the Ullman test, respectively) and to substitute correctly inflected, semantically appropriate verbs for targets in all

Table 4 *Incidence of impairment on past tense production as a function of condition*

Group	Condition	Ullman test			CSL test		
		Impairment		Fisher's exact test 1-tailed *P* value	Impairment		Fisher's exact test 1-tailed *P* value
		Present	Absent		Present	Absent	
CVA	Regular	2	5	Regular v irregular past 0.296	3	4	Regular v Irregular
	Irregular	4	3	Irregular v novel past 0.5	1	6	past 0.28
	Novel	3	4	Regular v novel past 0.5			
Mild Parkinson's	Regular	3	5	Regular v irregular past 0.5	2	6	Regular v Irregular
disease	Irregular	2	6	Irregular v novel past 0.715	2	6	past 0.715
	Novel	2	6	Regular v novel past 0.5			
Moderate Parkinson's	Regular	5	2	Regular v irregular past 0.72	6	1	Regular v Irregular
disease	Irregular	5	2	Irregular v novel past 0.231	5	2	past 0.5
	Novel	7	0	Regular v novel past 0.231			
Mild Huntington's	Regular	4	3	Regular v irregular past 0.28	6	1	Regular v Irregular[1]
disease	Irregular	6	1	Irregular v novel past 0.28	2	5	past 0.051
	Novel	4	3	Regular v novel past 0.704			
Moderate Huntington's	Regular	2	1	Regular v irregular past 0.5	3	0	Regular v Irregular
disease	Irregular	3	0	Irregular v novel past[1]	3	0	past[1]
	Novel	3	0	Regular v novel past 0.5			

[1]Impairment is constant in this comparison so Fisher's exact test has not been computed.

Table 5 *Ullman test: mean percentage responses*

Condition	Example	Control	CVA	Mild Parkinson's disease	Moderate Parkinson's disease	Mild Huntington's disease	Moderate Huntington's disease
Regular: correct	slammed	99.38 (1.77)	96.43 (7.48)	98.13 (2.59)	93.57 (5.56)	93.57 (7.48)	83.33 (17.56)
Cue repetition (stem)	slam	0	0.71 (0.83)	0	3.57 (4.76)	2.14 (3.93)	0
Cue repetition (S suffixed)	slams	0	1.43 (1.67)	0	1.43 (2.44)	2.14 (2.67)	1.67 (2.89)
ING suffixed	slamming	0	0	0	0	0	1.67 (2.89)
Related past tense	banged	0.63 (1.77)	1.43 (2.44)	1.88 (2.59)	0.71 (1.89)	1.43 (2.44)	13.33 (15.28)
Related uninflected form	bang	0	0	0	0.71 (1.89)	0	0
Phonological distortion	slommed	0	0	0	0	0.71 (1.89)	0
Irregular: correct	dug	98.03 (2.72)	88.72 (7.7)	96.05 (4.67)	84.96 (10.27)	85.71 (11.66)	73.68 (10.53)
Cue repetition (stem)	dig	1.32 (2.44)	6.02 (1.99)	1.97 (3.92)	2.26 (2.81)	3.76 (2.57)	7.02 (3.04)
Cue repetition (S suffixed)	digs	0	3.01 (4.14)	1.32 (2.44)	2.26 (4.14)	0.75 (1.99)	0
Regularization	digged	0.66 (1.86)	0.75 (1.99)	0	0.75 (1.99)	6.77 (11.25)	1.75 (3.04)
S suffixed	digs	0	0	0	0	0.75 (1.99)	0
Unmarked	dig	0	0	0	0	0.75 (1.99)	0
Related past tense	ploughed	0	1.5 (3.98)	0.66 (1.86)	7.52 (4.14)	1.5 (2.57)	14.04 (13.25)
Related uninflected form	ploughing	0	0	0	0.75 (1.99)	0	1.75 (3.04)
Phonological distortion	deg	0	0	0	1.5 (2.57)	0	1.75 (3.04)
Novel: correct	scashed	99.38 (1.77)	82.14 (36.73)	96.88 (5.94)	82.86 (6.99)	93.57 (8.02)	80 (18.03)
Cue repetition (stem)	scash	0	0	0	0.71 (1.89)	1.43 (3.78)	0
Cue repetition (S suffixed)	scashes	0	0.71 (1.89)	0	0	1.43 (3.78)	1.67 (2.89)
S suffixed	scashes	0	0	0	0	0	3.33 (5.77)
ING suffixed	scashing	0	0.71 (1.89)	0	0	0	0
Existing past tense	scratched	0.63 (1.77)	13.57 (33.75)	1.88 (3.72)	5.71 (6.07)	1.43 (2.44)	13.33 (10.41)
Existing S/ING form	scratches	0	0	0	1.43 (2.44)	0.71 (1.89)	0
Existing stem	scratch	0	1.43 (3.78)	0	2.14 (2.67)	1.43 (2.44)	1.67 (2.89)
Phonological distortion	scosh	0	1.43 (2.44)	1.25 (2.31)	7.14 (4.88)	0	0

SD is shown in brackets.

conditions (e.g. 1.43%, 1.5% and 1.43% for regular, irregular and novel verbs in the Ullman test, respectively). Their dominant error for irregular verbs may appear to be over-application of the -ed inflection, which would be consistent with the Declarative/Procedural Model. However, the majority of patients made either no regularization errors or a single regularization error (almost always for verbs that also elicited regularization errors in healthy controls) while a single patient accounted for 10 out of 13 regularization errors on the CSL test and six out of nine on the Ullman test. The errors of this patient are consistent with the Declarative/Procedural Model, but she is atypical of the

Table 6 *CSL test: mean percentage responses*

Condition	Example	Control	CVA	Mild Parkinson's disease	Moderate Parkinson's disease	Mild Huntington's disease	Moderate Huntington's disease
Regular: correct performance	slammed	99.52 (1.36)	98.35 (2.06)	99.04 (1.78)	90.11 (9.38)	91.76 (6.45)	91.03 (2.22)
Cue repetition (stem)	slam	0	0	0.48 (1.36)	2.2 (3.03)	1.1 (1.88)	0
Cue repetition (S suffixed)	slams	0.48 (1.36)	1.1 (1.88)	0.48 (1.36)	1.1 (1.88)	2.75 (4.28)	2.56 (4.44)
Unmarked	slam	0	0.55 (1.45)	0	1.65 (3.03)	0	0
Related past tense	banged	0	0	0	4.4 (6.45)	3.85 (4.97)	6.41 (2.22)
Related S/ING form	bangs	0	0	0	0	0.55 (1.45)	0
Related uninflected form	bang	0	0	0	0.55 (1.45)	0	0
Irregular: Correct performance	dug	98.61 (1.92)	93.12 (10.13)	97.22 (3.28)	87.83 (10.63)	91.01 (14.48)	87.65 (5.66)
Cue repetition (S suffixed)	digs	0	0	0	0.53 (1.4)	1.59 (4.2)	2.47 (2.14)
Regularization	digged	1.39 (1.92)	5.29 (7.67)	2.31 (2.76)	6.88 (7.23)	6.88 (13.43)	2.47 (4.28)
Unmarked	dig	0	0.53 (1.4)	0	0	0.53 (1.4)	0
Related past tense	ploughed	0	0.53 (1.4)	0	2.65 (4.64)	0	4.94 (4.28)
Related S/ING form	ploughs	0	0.53 (1.4)	0	1.06 (2.8)	0	0
Phonological distortion	deg	0	0	0.46 (1.31)	1.06 (1.81)	0	2.47 (4.28)

SD is shown in brackets.

group as a whole and cannot be said to have a selective impairment of regular inflectional morphology since she was impaired on almost all conditions tested with the exception of novel verbs.

Unlike the patients in Ullman *et al*. (1997), the moderate Huntington's disease group made relatively few regularization errors and no multiply suffixed errors in their first responses. In contrast to mild Huntington's disease patients, they showed an increased tendency to substitute correctly inflected, semantically related words and lexical intrusions for targets. The fact that substitution errors occur across all conditions in patients with moderate Parkinson's disease and Huntington's disease suggests a general difficulty with the task, rather than a selective deficit with applying the regular past tense rule. Since their substitution errors were correctly inflected, it is possible that rather than reflecting an impairment of morphological processing *per se*, they indicate a secondary consequence of a failure to inhibit prepotent responses, which increases with disease progression.

Past tense comprehension

The reaction time data were prepared and analysed separately for each group, with the exception of the moderate Huntington's disease patients, who proved unable to carry out the task and for whom data collection had to be abandoned. Items with high error rates (e.g. $\geqslant$30%) were removed (one from control data, three from mild Parkinson's disease data and seven from moderate Parkinson's disease data) and the remaining data were inverse-transformed (1000/reaction time) to correct a positively skewed distribution (Ratcliff, 1993). Mean reaction times were calculated over participants and items and entered into two analyses of variance (ANOVA) on participant (F1) and item (F2) means for each subject group. In the F1 analysis, prime type (related versus unrelated) and condition (regular past tense, irregular past tense, phonologically related, semantically related) were treated as within-participants factors. In the F2 analysis, prime type was treated as a within-item factor and condition was treated as a between-items factor. In all analyses, version (1–2) was included as an independent, dummy variable to stabilize variance due to the rotation of items over test lists. No main effects or interactions involving version are reported.

Figure 2 shows the amount of priming per condition for each group and Table 7 summarizes the analysis of variance results for each group. The main effects of prime type and condition were significant across all groups. The interaction between prime type and condition was significant for all groups with the exception of the moderate Parkinson's disease patients, for whom the interaction was significant by items, but only marginal by subjects. All groups showed significant facilitatory priming for regular and irregular verbs, and semantically related words. For most groups, phonologically related primes provided either no significant facilitation (controls, CVA and mild Huntington's disease groups) or significant inhibition (mild Parkinson's disease group). The only evidence of abnormality in any group was significant facilitation for phonologically related words in moderate Parkinson's disease patients.

Overall, there appears to be no evidence for a selective deficit in regular past tense comprehension, as indexed by morphological priming, in any of the patient groups tested. We used *post hoc* tests to examine the amount of priming for regular past tense forms that are infrequently encountered and which are, according to the Words and Rules theory and the related Declarative/Procedural Model, most likely to be decomposed into stems and inflections. We defined low frequency regular past tense primes as those with a word form frequency $\leqslant$6 ($n = 6$), following research suggesting that such words are recognized in terms of their component stems and inflections (e.g. hack + ed; shove + d) in comprehension (Alegre and Gordon, 1999). Despite the very small number of items, significant priming was confirmed for low frequency regular past tense primes in all groups by ANOVAs testing the

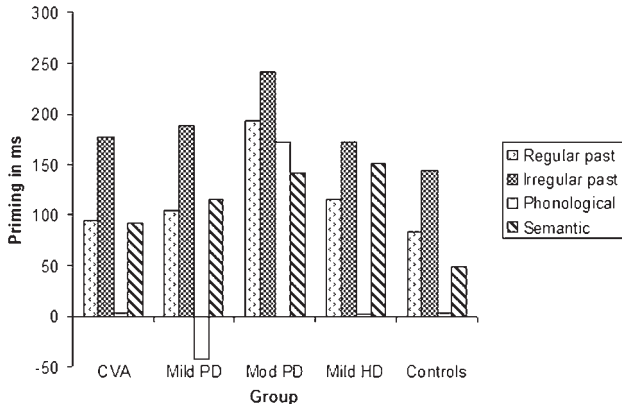

Fig. 2 Amount of priming (the difference in reaction times following related and unrelated words) in ms.

effects of prime type (within item factor) and version (between item factor) [controls: $F(1,5) = 56.047$, $P = 0.001$; CVA: $F(1,5) = 267.53$, $P < 0.001$; mild Parkinson's disease: $F(1,5) = 18.057$, $P = 0.008$; moderate Parkinson's disease: $F(1,5) = 68.439$, $P < 0.001$; mild Huntington's disease: $F(1,5) = 21.42$, $P < 0.001$]. This confirms that there is intact automatic comprehension even for low frequency regular past tense forms, which are postulated by the Words and Rules theory to require decomposition (leading to identity priming from the repeated stem).

Discussion

This research investigated the role of the basal ganglia in the language system by testing the claim that a fronto-striatal network subserves grammatical rule use, including adding the inflection -ed to verbs to produce the regular past tense in English and decomposing regular past tense forms into stems and suffixes in comprehension (Ullman *et al*., 1997, 2005; Ullman, 2001; Pinker and Ullman, 2002). This claim provides an important alternative to the hypothesis that the role of the striatum in language is restricted to the inhibition of competing alternatives in late integrational processes of language comprehension and, possibly, in the lexicalization stage of language production.

Our results suggest that basal ganglia disorders are not reliably associated with difficulties in comprehending regular past tense forms, implying that the basal ganglia are not essential components of the system involved. Patients with cerebrovascular damage or neurodegenerative disease of the basal ganglia showed a normal pattern of significant morphological and semantic priming. There was no evidence of an association between basal ganglia disorders and impaired interpretation of spoken regular past tenses, despite the fact that the experimental paradigm has sufficient sensitivity to demonstrate a double dissociation between regular and irregular past tense priming in different groups of brain-damaged patients (Marslen-Wilson and Tyler, 1997, 1998; Tyler *et al*., 2002). These results extend the finding that patients with basal ganglia dysfunction have intact automatic processing of

Table 7 *Analyses of variance on primed lexical decision data for each group*

	Controls	CVA	Mild Parkinson's disease	Moderate Parkinson's disease	Mild Huntington's disease
Range of mean reaction times (ms)	770–1285	802–1449	795–1494	977–2190	914–1475
Prime type	$F1(1,14) = 31.02$, $P < 0.001$	$F1(1,12) = 7.54$, $P = 0.018$	$F1(1,14) = 52.44$, < 0.001	$F1(1,12) = 19.37$, $P = 0.001$	$F1(1,12) = 16.05$, $P = 0.002$
	$F2(1,123) = 106.16$, < 0.001	$F2(1,124) = 111.95$, $P < 0.001$	$F2(1,121) = 161.49$, $P < 0.001$	$F2(1,117) = 196.42$, $P < 0.001$	$F2(1,124) = 80.46$, $P < 0.001$
Condition	$F1(3,42) = 37.05$, $P < 0.001$	$F1(3,36) = 13.74$, $P <.001$	$F1(3,42) = 35.5$, $P < 0.001$	$F1(3,36) = 6.48$, $P = 0.001$	$F1(3,36) = 11.06$, $P < 0.001$
	$F2(3,123) = 4.54$, $P = 0.005$	$F2(3,124) = 3.04$, $P = 0.032$	$F2(3,121) = 5.61$, $P = 0.001$	$F2(3,117) = 3.41$, $P = 0.02$	$F2(3,124) = 4.62$, $P = 0.004$
Interaction	$F1(3,42) = 21.44$, $P = 0.001$	$F1(3,36) = 15.17$, $P <.001$	$F1(3,42) = 37.89$, $P < 0.001$	$F1(3,36) = 2.43$, $P = 0.081$	$F1(3,36) = 10.09$, $P < 0.001$
	$F2(3,123) = 13.23$, $P < 0.001$	$F2(3\ 124) = 11.18$, $P < 0.001$	$F2(3,121) = 26.98$, $P < 0.001$	$F2(3,117) = 3.17$, $P = 0.027$	$F2(3,124) = 7.14$, $P < 0.001$
Regular past	$F1(1,14) = 25.86$, $P = 0.001$	$F1(1,12) = 7.08$, $P = 0.02$	$F1(1,14) = 43.54$, $P = 0.001$	$F1(1,12) = 15.09$, $P = 0.002$	$F1(1,12) = 6.04$, $P = 0.03$
	$F2(1,40) = 49.63$, $P < 0.001$	$F2(1,40) = 37.52$, $P < 0.001$	$F2(1,39) = 41.21$ $P < 0.001$	$F2(1,38) = 46.74$ $P < 0.001$	$F2(1,40) = 22.71$, $P < 0.001$
Irregular past	$F1(1,14) = 31.63$, $P = 0.001$	$F1(1,12) = 27.29$, $P < 0.001$	$F1(1,14) = 62.28$, $P = 0.001$	$F1(1,12) = 21.37$, $P = 0.001$	$F1(1,12) = 32.95$, $P < 0.001$
	$F2(1,40) = 97.45$, $P < 0.001$	$F2(1,40) = 85.57$, $P < 0.001$	$F2(1,40) = 150.04$, $P < 0.001$	$F2(1,40) = 107.1$, $P < 0.001$	$F2(1,40) = 45.8$, $P < 0.001$
Phonologically related	Both $Fs < 1$	Both $Fs < 1$	$F1(1,14) = 3.57$, $P = 0.08$	$F1(1,12) = 9.79$, $P = 0.009$	Both $Fs < 1$
			$F2(1,20) = 4.70$ $P = 0.042$	$F2(1,19) = 26.27$ $P < 0.001$	
Semantically related	$F1(1,14) = 14.07$, $P = 0.002$	$F1(1,12) = 9.46$, $P = 0.01$	$F1(1,14) = 44.42$, $P < 0.001$	$F1(1,12) = 10.09$, $P = 0.008$	$F1(1,12) = 17.6$, $P = 0.001$
	$F2(1,22) = 3.97$, $P = 0.059$	$F2(1,22) = 15.65$, $P = 0.01$	$F2(1,22) = 28.49$, $P < 0.001$	$F2(1,20) = 24.61$, $P <.001$	$F2(1,22) = 43.7$, $P < 0.001$

F1 = Analysis by subjects. *F2* = analysis by items. The degrees of freedom for each analysis are shown in brackets.

syntax in language comprehension (Friederici *et al*., 1999, 2003) into the morpho-syntactic domain. They also replicate previous findings of semantic priming in Parkinson's disease and subcortical cerebrovascular patients at short stimulus onset intervals (Copland, 2003), indicating that patients with basal ganglia dysfunction have intact automatic semantic processing.

Production deficits are among the most commonly reported aphasic symptoms of basal ganglia damage and tests of theoretical claims concerning the role of the basal ganglia in past tense morphology have focused predominantly on past tense production (Ullman *et al*., 1997; Ullman, 2001; Pinker and Ullman, 2002). The Declarative/Procedural Model proposes that hypokinesia in Parkinson's disease is associated with suppression of rule use, causing patients to produce unmarked forms of regular verbs, whereas hyperkinesia in Huntington's disease is associated with excess rule use, causing patients to produce over-regularization errors and unusual multiply or syllabically suffixed words (Ullman *et al*., 1997). In contrast to these claims, we found no evidence that inflecting regular and novel verbs was consistently associated with a higher incidence of impairment than irregular verbs in patients with striatal dysfunction. We also found no evidence for significant differences in right-sided hypokinesia between Parkinson's disease patients with and without regular past tense impairments.

Impairments were common for moderate Huntington's disease and Parkinson's disease patients across all conditions of the past tense elicitation tasks. This suggests a general problem with producing the correct morphological features in a syntactic sequence, irrespective of verb regularity, in contrast to the selective deficit for regular inflectional morphology predicted by the Declarative/Procedural Model. The errors produced also appear to be inconsistent with the Declarative/Procedural Model. There were no multiply suffixed errors in the first responses of Huntington's disease patients and only a single patient showed a substantial tendency to over-regularize irregular verbs. In the CSL test, patients with moderate Parkinson's disease were more likely to produce semantically related verbs with the correct inflection (e.g. banged rather than slammed) than not to inflect the verb (e.g. slam). In the Ullman test, they showed a tendency to repeat cues rather than to produce a past tense, but at similar rates for both regular and irregular verbs. They were least likely to do this for novel verbs, which instead elicited phonological distortion and lexical intrusion errors, despite correct application of the -ed inflection.

Our findings do not support an essential role for the basal ganglia in applying grammatical rules to combine morphemes in language production or to decompose words into morphemes in language comprehension. This is consistent with a previous report that patients with Parkinson's disease did not show a significant difference between untimed past tense elicitation for regular and irregular verbs, matched for frequency and were faster and less error-prone in speeded regular past tense production than irregular (Almor *et al*., 2002). Several aspects of our results are compatible, however, with the hypothesis that the striatum plays a restricted, non-language specific, inhibitory role in the late stages of language processing. First, the automatic activation of semantic and morphological representations in language comprehension was intact in all groups as discussed above. Secondly, patients with moderate Parkinson's disease showed an abnormal pattern of response facilitation for phonologically related words in language comprehension. This suggests that despite normal activation of lexical representations initially consistent with incoming speech, there is a failure to suppress activation for those representations that subsequently become incompatible with the speech signal. Thus, for example, the possibility that the word 'captive' is being heard would not be inhibited by hearing the second syllable in the word 'captain'. Thirdly, patients across all groups showed evidence of an inability to suppress semantically appropriate alternatives when trying to inflect novel verbs and this was the dominant error made by patients with moderate Huntington's disease across all conditions of both tasks. This appears to be consistent with reports of semantic paraphasias following subcortical cerebrovascular damage (Damasio *et al*., 1982) and the failure of patients with striatal dysfunction to suppress the infrequent meaning of homophones in semantic priming at long stimulus onset intervals (Copland, 2003).

In conclusion, the data reported here suggest that subcortical cerebrovascular accidents or neurodegenerative disorders of the basal ganglia are not reliably associated with selective regular past tense deficits, contrary to the predictions of the Declarative/Procedural Model. Thus, the basal ganglia do not appear to be essential for processing the regular past tense as a sequence of morphemes, either in comprehension or production. It seems more likely that neocortical regions are critical for this processing rather than the basal ganglia. Such a conclusion would be consistent with our recent finding that healthy volunteers show increased activation of the left inferior frontal gyrus and the left superior temporal gyrus when processing the regular past tense than irregular forms or words matched to past tense phonology (Marslen-Wilson *et al*., 2003). Our results add to the increasing evidence that the role of the striatum in the language system is restricted to late integrational processes requiring inhibition of competing alternatives.

Acknowledgements

We wish to give sincere thanks to all our participants. We also wish to thank Professor Barbara Sahakian, Professor Trevor Robbins and Dr Raymond Knott for their help with this research. We also thank our reviewers for their helpful comments on earlier drafts. This research was supported by MRC post-graduate research studentships to S.E.K. and C.E.L., a University of Cambridge Pinsent-Darwin Research Fellowship to C.E.L. and a MRC programme grant to L.K.T.

References

Alegre M, Gordon P. Frequency effects and the representational status of regular inflections. J Mem Lang 1999; 40: 41–61.

Almor A, Kempler D, Andersen ES, McDonald MC, Hayes UL, Hintiryan H. The production of regularly and irregularly inflected nouns and verbs in Alzheimer's and Parkinson's patients. Brain Lang 2002; 83: 149–51.

Aron AR, Schlaghecken F, Fletcher PC, Bullmore ET, Eimer M, Barker R, et al. Inhibition of subliminally primed responses is mediated by the caudate and thalamus: evidence from functional MRI and Huntington's disease. Brain 2003; 126: 713–23.

Baayen RH, Piepenbrock R, Gulikers L. The CELEX Lexical Database (CD-ROM). Philadelphia: Linguistic Data Consortium, University of Pennsylvania; 1995.

Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry 1961; 4: 561–71.

Berent I, Pinker S, Shimron J. Default nominal inflection in Hebrew: evidence for mental variables. Cognition 1999; 72: 1–44.

Berko J. The child's learning of English morphology. Word 1958; 14: 150–77.

Brunner RJ, Kornhuber HH, Seemuller E, Suger G, Wallesch CW. Basal ganglia participation in language pathology. Brain Lang 1982; 16: 281–99.

Cappa SF. Subcortical aphasia: still a useful concept? Brain Lang 1997; 58: 424–6 [discussion 436–58].

Cohen NJ, Squire LR. Preserved learning and retention of pattern-analysing skill in amnesia: dissociation of knowing how and knowing that. Science 1980; 210: 207–10.

Cools R, Clark L, Owen AM, Robbins TW. Defining the neural mechanisms of probabilistic reversal learning using event-related functional magnetic resonance imaging. J Neurosci 2002; 22: 4563–7.

Copland D. The basal ganglia and semantic engagement: potential insights from semantic priming in individuals with subcortical vascular lesions, Parkinson's disease, and cortical lesions. J Int Neuropsychol Soc 2003; 9: 1041–52.

Crosson B. Subcortical functions in language: a working model. Brain Lang 1985; 25: 257–92.

Cummings JL, Darkins A, Mendez M, Hill MA, Benson DF. Alzheimer's disease and Parkinson's disease: comparison of speech and language alterations. Neurology 1988; 38: 680–4.

Damasio AR, Damasio H, Rizzo M, Varney N, Gersh F. Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. Arch Neurol 1982; 39: 15–24.

Fahn S, Elton RL, Members of the UPDRS Development Committee. Unified Parkinson's Disease Rating Scale. In: Fahn S, Marsden CD, Calne DB, Goldstein M, editors. Recent developments in Parkinson's disease. Florham Park (NJ): Macmillan; 1987. p. 153–63.

Folstein MF, Folstein SE, McHugh PR. 'Mini-mental state'. A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975; 12: 189–98.

Forster KI, Forster JC. DMDX: A Windows display program with millisecond accuracy. Behav Res Methods Instrum Comput 2003; 35: 116–24.

Friederici AD, von Cramon DY, Kotz SA. Language related brain potentials in patients with cortical and subcortical left hemisphere lesions. Brain 1999; 122: 1033–47.

Friederici AD, Kotz SA, Werheid K, Hein G, von Cramon DY. Syntactic comprehension in Parkinson's disease: investigating early automatic and late integrational processes using event-related brain potentials. Neuropsychology 2003; 17: 133–42.

Frisch S, Kotz SA, von Cramon DY, Friederici AD. Why the P600 is not just a P300: the role of the basal ganglia. Clin Neurophysiol 2003; 114: 336–40.

Gordon WP, Illes J. Neurolinguistic characteristics of language production in Huntington's disease: a preliminary report. Brain Lang 1987; 31: 1–10.

Grossman M. Sentence processing in Parkinson's disease. Brain Cogn 1999; 40: 387–413.

Grossman M, Kalmanson J, Bernhardt N, Morris J, Stern MB, Hurtig HI. Cognitive resource limitations during sentence comprehension in Parkinson's disease. Brain Lang 2000; 73: 1–16.

Grossman M, Lee C, Morris J, Stern MB, Hurtig HI. Assessing resource demands during sentence processing in Parkinson's disease. Brain Lang 2002; 80: 603–16.

Hoehn MM and Yahr MD (1967) Parkinsonism: onset, progression and mortality. Neurology 17, 427–442.

Illes J. Neurolinguistic features of spontaneous language production dissociate three forms of neurodegenerative disease: Alzheimer's, Huntington's, and Parkinson's. Brain Lang 1989; 37: 628–42.

Illes J, Metter EJ, Hanson WR, Iritani S. Language production in Parkinson's disease: acoustic and linguistic considerations. Brain Lang 1988; 33: 146–60.

Kemmerer D. Impaired comprehension of raising-to-subject constructions in Parkinson's disease. Brain Lang 1999; 66: 311–28.

Kieburtz K, The Huntington Study Group. The unified Huntington's disease rating scale: reliability and consistency. Mov Disord 1996; 11: 136–42.

Kotz SA, Frisch S, von Cramon DY, Friederici AD. Syntactic language processing: ERP lesion data on the role of the basal ganglia. J Int Neuropsychol Soc 2003; 9: 1053–60.

Lawrence AD, Sahakian BJ, Robbins TW. Cognitive functions and corticostriatal circuits: insights from Huntington's disease. Trends Cogn Sci 1998; 2: 379–88.

Lieberman P, Friedman J, Feldman LS. Syntax comprehension deficits in Parkinson's disease. J Nerv Ment Dis 1990; 178: 360–5.

Marslen-Wilson WD, Hare M, Older L. Inflectional morphology and phonological regularity in the English mental lexicon. 15th Annual Meeting of the Cognitive Science Society; Boulder (CO), USA. Hillsdale (NJ): Erlbaum; 1993.

Marslen-Wilson WD, Tyler LK. Dissociating types of mental computation. Nature 1997; 387: 592–4.

Marslen-Wilson WD, Tyler LK (1998). Rules, representations and the English past tense. Trends in Cognitive Science 2, 428–436.

Marslen-Wilson WD, Stamatakis EA, Post B, Randall B, Tyler LK. Neural differentiation within the language system: the past tense. Neuroimage 2003; 19: S1321.

McNamara P, Krueger M, O'Quin K, Clark J, Durso R. Grammaticality judgments and sentence comprehension in Parkinson's disease: a comparison with Broca's aphasia. Int J Neurosci 1996; 86: 151–66.

Mink JW. The basal ganglia: focused selection and inhibition of competing motor programs. Prog Neurobiol 1996; 50: 381–425.

Naeser MA, Alexander MP, Helm-Estabrooks N, Levine HL, Laughlin SA, Geschwind N. Aphasia with predominantly subcortical lesion sites: description of three capsular/putaminal aphasia syndromes. Arch Neurol 1982; 39: 2–14.

Natsopoulos D, Katsarou Z, Bostantzopoulou S, Grouios G, Mentenopoulos G, Logothetis J. Strategies in comprehension of relative clauses by parkinsonian patients. Cortex 1991; 27: 255–68.

Natsopoulos D, Grouios G, Bostantzopoulou S, Mentenopoulos G, Katsarou Z, Logothetis J. Algorithmic and heuristic strategies in comprehension of complement clauses by patients with Parkinson's disease. Neuropsychologia 1993; 31: 951–64.

Nelson HE. National Adult Reading Test (NART) test manual. Windsor (UK): NFER-Nelson; 1982.

Pinker S. Rules of language. Science 1991; 253: 530–5.

Pinker S, Ullman MT. The past and future of the past tense. Trends Cogn Sci 2002; 6: 456–63.

Ratcliff R. Methods for dealing with reaction-time outliers. Psychol Bull 1993; 114: 510–32.

Robbins TW, James M, Owen AM, Sahakian BJ, McInnes L, Rabbitt P. Cambridge Neuropsychological Test Automated Battery (CANTAB): a factor analytic study of a large sample of normal elderly volunteers. Dementia 1994; 5: 266–81.

Schwab RS, England AC. Projection technique for evaluating surgery in Parkinson's disease. In: Gillingham FJ, Donaldson IML, editors. 3rd Symposium on Parkinson's disease, Royal College of Surgeons of Edinburgh; 1968 May 20–22; Edinburgh. Edinburgh: E & S Livingstone; 1969. p. 152–7.

Tyler LK, de Mornay-Davies P, Anokhina R, Longworth C, Randall B, Marslen-Wilson WD. Dissociations in processing past tense morphology: neuropathology and behavioural studies. J Cogn Neurosci 2002; 14: 79–94.

Ullman MT. A neurocognitive perspective on language: the Declarative/Procedural Model. Nat Rev Neurosci 2001; 2: 717–26.

Ullman MT, Corkin S, Coppola M, Hickok G, Growdon JH, Koroshetz WJ, et al. A neural dissociation within language: evidence that the mental dictionary is part of declarative memory, and that grammatical rules are processed by the procedural system. J Cogn Neurosci 1997; 9: 266–76.

Ullman MT, Izvorski R, Love T, Yee E, Swinney D, Hickok G. Neural correlates of lexicon and grammar: evidence from the production, reading and judgment of inflection in aphasia. Brain Lang. In press, 2005.

Wallesch C-W, Papagno C. Subcortical aphasia. In: Rose FC, Whurr R, Wyke M, editors. Aphasia. London: Whurr Publishers; 1988. p. 256–87.

Wallesch CW, Kornhuber HH, Brunner RJ, Kunz T, Hollerbach B, Suger G. Lesions of the basal ganglia, thalamus, and deep white matter: differential effects on language functions. Brain Lang 1983; 20: 286–304.