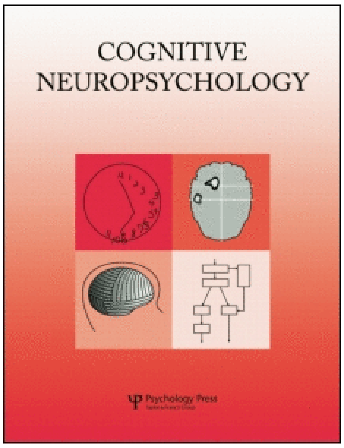

Cognitive Neuropsychology
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t713659042

"Deep" language disorders in nonfluent progressive Aphasia: an evaluation of the "summation" account of semantic errors across language production tasks

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Online Publication Date: 01 September 2005

To cite this Article Tree, Jeremy J. Dr, Kay, Janice and Perfect, Timothy J.(2005)'"Deep" language disorders in nonfluent progressive Aphasia: an evaluation of the "summation" account of semantic errors across language production tasks',Cognitive Neuropsychology,22:6,643 — 659

To link to this Article: DOI: 10.1080/02643290442000220

URL: http://dx.doi.org/10.1080/02643290442000220

COGNITIVE NEUROPSYCHOLOGY, 2005, 22 (6), 643–659

Psychology Press
Taylor & Francis Group

"DEEP" LANGUAGE DISORDERS IN NONFLUENT PROGRESSIVE APHASIA: AN EVALUATION OF THE "SUMMATION" ACCOUNT OF SEMANTIC ERRORS ACROSS LANGUAGE PRODUCTION TASKS

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This study focuses on the pattern of impairments seen in a new case KT, diagnosed with nonfluent progressive aphasia (NFPA), a degenerative disorder of language production. A systematic examination of KT's performance on a wide range of language production tasks (i.e., repetition, reading, spelling, spoken and written naming) determined that both written naming and repetition were better preserved than reading, spelling-to-dictation, and spoken naming. Closer examination of error performance in both reading aloud and written production revealed evidence of "deep dyslexia" and "deep dysgraphia" that has not been documented in previous cases of NFPA, and as such the present case represents the first detailed case study of this pattern of impairment in the context of progressive aphasia. An evaluation and discussion of such deep language impairment disorders in the context of other cases of NFPA has been undertaken with reference to the summation hypothesis proposed by Hillis and Caramazza (1991, 1995). It is suggested that as a principle that holds across all language production tasks, this account can encompass patterns of deep disorders thus far reported in NFPA, although other theoretical hypotheses cannot be excluded.

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We are most grateful to both KT and his wife for consenting to participate in this research, and to the many pleasant sunny summer afternoons enjoying their company. We are also grateful to Dr Paul Hogbin for initially bringing this case to our attention, and for his helpful insights concerning KT's medical history. We would like to thank both Max Coltheart and Rick Hanley for their comments concerning the summation hypothesis. We would also like to thank Karen Croot and Karalyn Patterson for providing unpublished test results concerning their cases. Finally, we would like to thank Nadine Martin for providing the items for the auditory discrimination tests that she devised. This research was supported by a research grant provided by the University of Plymouth.

http://www.tandf.co.uk/journals/pp/02643294.html
DOI:10.1080/02643290442000220

INTRODUCTION

In this paper we examine the language performance of KT, diagnosed with nonfluent progressive aphasia (NFPA).[1] Spontaneous speech in NFPA is typically classified as nonfluent as it is effortful, halting, agrammatic, and often contains phonemic paraphasias (e.g., Kertesz, Davidson, McCabe, Takagi, & Munoz, 2003; Snowden, Neary, & Mann, 1996). Although such cases present with severe expressive language impairments, they typically perform well on tests of comprehension of single words and demonstrate no evidence of generalised dementia with normal activities of daily living (Mesulam, 1982, 1987). The focus of the majority of published cases of NFPA has been clinical in nature (e.g., Caseli & Jack, 1992; Kartsounis, Crellin, Crewes, & Toone, 1991; McDaniel, Wagner, & Greenspan, 1991; Thompson, Ballard, Tait, Weintraub, & Mesulam, 1997) and therefore there is a need to conduct more detailed investigations of language deterioration in such patients. Little has been done to examine performance of cases of NFPA across language production tasks (i.e., repetition, reading, writing to dictation, spoken naming, and written naming).

In an attempt to address this oversight, we examined in a previous paper the performance of an NFPA patient, PW, across naming, reading, and repetition tasks (Tree, Perfect, Hirsh, & Copstick, 2001). Overall, PW demonstrated superior reading performance relative to naming and repetition, but equivalent performance on naming and repetition, consistent with the profile of two other previously reported NFPA cases: CB (Croot, Patterson, & Hodges, 1999) and CO (Majerus, Lekeu, Van der Linden, & Salmon, 2001). In particular, and consistent again with both these cases, PW demonstrated imageability effects and semantic errors when repeating single words. These three NFPA cases (CB, CO, and PW) therefore provide evidence for the emergence of a repetition impairment similar to that observed in cases of "deep dysphasia" (e.g., Butterworth & Warrington, 1995; Coslett, 1991; Katz & Goodglass, 1990; Marshall & Newcombe, 1988; Martin & Saffran, 1992; Michel & Andreewsky, 1983). Deep dysphasic patients demonstrate an inability to repeat nonwords, indicating severe disruption to sublexical repetition processes. Of critical theoretical relevance in the NFPA cases, therefore, is whether they were able to repeat nonwords. Although we can draw no clear conclusions for CB, as nonword repetition performance was not reported, it is clear that neither PW nor CO were able to repeat nonwords, and this profile is therefore consistent with stable cases of deep dysphasia in the literature.

How might the pattern of deep dysphasia be explained? Used as an account for semantic errors in the context of deep dyslexia, an acquired disorder of reading, the summation hypothesis (e.g., Hillis & Caramazza, 1991, 1995; Miceli, Capasso, & Caramazza, 1994) claims that an intact sublexical reading route "blocks" the production of semantic errors by constraining the phonology of potential targets, thereby largely excluding semantically related words. Generalising across disorders of production, this hypothesis suggests that severe impairment or abolition of sublexical phonology will allow the production of semantic errors. The performance of PW and CO, and possibly CB, is therefore consistent with a summation account of semantic errors in auditory repetition. A critical indicator of whether semantic errors will be produced in output is an ability to produce nonwords on the same test. Interestingly, neither PW, CO, nor CB produced semantic errors in tasks of reading aloud and written spelling, and we note that nonword production on these tasks was relatively well preserved for at least two of the patients (nonword data are again unavailable for CB). Unfortunately, other reports of NFPA have generally failed to examine systematically word and nonword language production, and therefore the presence (or otherwise) of semantic errors in such cases cannot be evaluated.

[1] We note that such cases have been alternatively referred to as nonfluent primary progressive aphasia, primary progressive nonfluent aphasia, and progressive nonfluent aphasia. In our previous study of a patient with a similar disorder we adopted the term nonfluent progressive aphasia, and in the interests of consistency have done the same for this case.

To address this issue, the present study sought to determine whether semantic errors are present across language production tasks (repetition, reading aloud, and writing) given to KT, a new case of NFPA, and at the same time to examine his word and nonword production. We also compared KT's performance across tasks with that of our former case, PW, to identify how much their cognitive profiles overlap and in what ways they differ. Only a single report of primary progressive aphasia (CO—Majerus et al., 2001) has systematically examined spoken and written language performance across language production tasks (i.e., oral/written naming, reading, repeating and spelling) and the present study therefore provides a comparison with the patterns of impairment reported in that case also.

CASE DESCRIPTION

KT is a 61-year-old, right-handed male who left full-time education at 18. At the time of testing he had recently retired from a managerial post. KT's medical history involved the presence of tonic-clonic seizures from the age of 19, until approximately 28, when there was a spontaneous remission. In November 1997, KT presented to clinical services complaining of some mild word-finding difficulties, although an MRI scan in January 1998 detected no abnormalities. However, a SPECT scan conducted in April 1998 determined symmetrical reduction in blood flow to the frontal lobes. A subsequent SPECT scan conducted in February 2001 revealed a somewhat similar pattern, with global reduction in cerebral perfusion and significantly reduced perfusion to the cortex and deep white matter, although there was some indication of greater reduction in left relative to right hemisphere activation. Throughout this period, neurological examination of KT was unremarkable and there was no evidence of limb or speech apraxia, although his anomic pattern worsened. There was also no evidence of hearing loss or visual impairment. After examination in February 2001, he was given a diagnosis of primary expressive aphasia (Maudsley Hospital, London), consistent with an insidious deterioration in speech and language performance, with no evidence of equivalent nonlinguistic cognitive deficits (Mesulam, 2001). Also consistent with such a diagnosis, there was no evidence of a deterioration of personality or behavioural change in the period from initial presentation up to the date of our final session (September 2001).[2]

Two initial clinical testing sessions (01/06/2001 and 08/06/2001) were conducted, during which KT's performance on a variety of verbal and nonverbal tasks was examined. In the Introduction we established that classification of NFPA is characterised by nonfluent speech, phonemic paraphasias, and agrammatism (e.g., Kertesz et al., 2003; Snowden et al., 1996). All these hallmarks were apparent in KT's performance (with the latter being most evident in comprehension, as his speech was limited to single words). His spontaneous speech was marked by long pauses with clear word-finding difficulties. In many instances phonological errors were apparent (both in

[2] NFPA cases tend to show asymmetrical anterior atrophy (typically left worse than right) and this is a relatively good neurodiagnostic feature, but it is noteworthy that such a pattern is not found in every patient (Mesulam, Grossman, Hillis, Kertesz, & Weintraub, 2003), with some cases being reported having bifrontal atrophy (e.g., Mimura et al., 2001) or bilateral symmetrically reduced perfusion in the dosolateral regions of the frontal lobes (e.g., Kartsounis et al., 1991; Nagy, Jelencsik, & Szirmai, 1999). In a recent review of 59 PET and SPECT studies of PPA, 69% of scans revealed abnormal results in the left hemisphere only, whilst 31% revealed bilateral abnormalities (Westbury & Bub, 1997). It is noteworthy that in some cases PPA patients can develop personality changes and develop stereotyped phrases/behavioural patterns characteristic of frontal lobe dementia (Snowden et al., 1996). This typically occurs soon after the first 2 years of presentation and such patients can be described as PPA-plus syndrome or frontal lobe dementia with aphasia (Mesulam et al., 2003). However, no such behavioural changes were ever present during the 3-year period of language degeneration preceding the commencement of our testing, and this remained the case throughout the conduction of our study. Overall, although KT's first SPECT scan is somewhat atypical of cases of NFPA, it is by no mean unique. Additionally, although bilateral, symmetrical frontal changes are also potentially indicative of a frontal lobe dementia, KT's overall clinical picture was not consistent with such cases. This is why a diagnosis of NFPA was considered appropriate.

conversation speech and spoken repetition performance) with examples of "articulatory groping" during which repeated attempts were made to produce the appropriate output (e.g., Repeat: 'BREAD'—KT: "Bled . . . erm . . . gred . . . gr . . . err . . . gr . . . erm . . . gred . . . no"). Single word comprehension was good, while comprehension of complex grammatical structures was impaired.

Despite progressively impaired speech production difficulties, throughout the testing period KT demonstrated normal performance on routine domestic day-to-day activities such as cooking, cleaning, shopping, etc. (e.g., Bristol Activities of Daily Living scale, in which a high score indicates impairment in everyday activities, he scored 4/60). He remained a keen singer, capable of holding a tune with good melody and rhythm. However, during the time we conducted our testing, KT had withdrawn from his local choral group after communication difficulties with other group members and an inability to learn new lyrics. For similar reasons (e.g., frustration with a marked decline in his communicative abilities) KT eventually decided not to continue with the study.

SUMMARY OF INITIAL TESTING RESULTS

We compared KT's performance across an initial range of tests with that of our previous case, PW; see Table 1. KT performs in a number of similar ways to PW (e.g., see BORB, WAIS-III), with the following notable exceptions: (1) retention of nonverbal material for delayed recall is better for KT than for PW; (2) KT's performance on verbal fluency is severely impaired and worse than that of PW; (3) KT's repetition performance was impaired but superior to that of PW; (4) consistent with better repetition abilities, KT's auditory verbal short-term memory, though impaired (both with and without spoken output), was better preserved than that of PW. KT's performance on measures of general IQ, executive function (see WCST; he completed all six categories), and object and face recognition (neither KT nor PW showed evidence of visuospatial or perceptual deficits) was normal (but KT and PW both have poor verbal IQ scores on the WAIS-III).[3] Grammatical and syntactic processing (TROG), and initial measures of reading ability were equivalent in both cases (although subsequent examination of KT's performance revealed important differences that we set out below).

EXPERIMENTAL INVESTIGATIONS

After an initial clinical assessment, we examined KT's language deficits in greater detail over a 4-month period (June–September 2001). We conducted the testing series in a concentrated period in order to reduce confounds between our interpretation of KT's pattern of impairment and any progressive worsening of his aphasic symptoms (Majerus et al., 2001). However, at points within the text we make reference to longitudinal data demonstrating KT's progressive language decline.

Comprehension

Although KT's day-to-day comprehension performance was good, examination of comprehension of single words showed modality-specific impairments (see Table 2), with normal performance on word/picture matching for spoken words (PALPA 47: KT = 39/40, controls = 39.29, *SD* = 1.07; PPT: KT = 49/52, controls = 51.25, *SD* = 0.74), but below normal range when responding to written

[3] Impairments of executive function are poorly understood in NFPA, and on formal tests of executive function a mixed pattern of preserved (on nonverbal measures such as the WCST) and impaired performance (on measures such as verbal fluency) is typically reported (Westbury & Bub, 1997). What is of critical importance is that cases of NFPA do not show the more qualitative features of frontal impairment, (i.e., inflexibility of responses, perseverations, and concreteness: Snowden et al., 1996). Consistent with such an evaluation of NFPA, KT does show variable performance on measures of executive function, but no qualitative features of frontal impairment. Thus we would argue that his overall performance is not consistent with a more generalised frontal impairment.

Table 1. *Basic neuropsychological data*

	KT score	*PW score*	*Norms*
Mini-Mental State	22/30	(20)	(29)
Raven's Progressive Matrices	10/12		(10)
WCST	6/6		(6)
WAIS–III			
Verbal IQ	68	(62)	
Performance IQ	97	(94)	
BORB			
Object Decision Test	115/128	(117)	(115)
Item Match Test	32/32	(31)	(30)
Foreshortened Match Test	24/25	(24)	(22)
WMS-III			
Visual Reproduction			
Copy	104/104	(100)	(79)
Recall Immediate	78/104	(58)	(77)
Recall Delayed	76/104	(56)	(51)
Face Recognition (Immediate)	36/48	(36)	(36)
Face Recognition (Delayed)	42/48	(34)	(35)
Information & Orientation	14/14	(14)	N/A
WRMT			
Faces	40/50	(50)	(42)
Words	40/50	(48)	(43)
Fluency			
Spoken initial letter (FAS)	0	(8)	(36)
Spoken animals	0	(16)	(17)
Written initial letter (A)	4	(6)	N/A
Written animals	3	(8)	N/A
Grammatical/syntactic processing			
TROG	64/80	(55)	(78)
Reading			
NART	7/50	(5)	(18)
Repetition			
PALPA 9	63/80	(42)	(79)
Auditory–verbal short-term memory			
WMS–III: Digit span – Forward/Backward	4/1	(2/2)	
PALPA 13: Digit matching span	5	(2)	
Word matching span	5	(2)	

BORB = Birmingham Object Recognition Battery, NART = National Adult Reading Test, PALPA = Psycholinguistic Assessments of Language Processing in Aphasia, TROG = Test for the Reception of Grammar, WAIS–III = Wechsler Adult Intelligence Scale Third Edition, WCST = Wisconsin Card Sorting Test, WMS–III = Wechsler Memory Scale Third Edition, WRMT = Warrington Recognition Memory Test.

words (PALPA 48: KT = 34/40, controls = 39.47, *SD* = 1.01; PPT: KT = 38/52, controls = 51.25, *SD* = 0.74). Synonym judgment performance across modalities was also examined (ADA; Franklin, Turner, & Ellis, 1992). Performance was equally poor regardless of test modality (no significant difference). Both tests manipulated imageability of stimulus pairs and KT made

Table 2. *Test data relating to language processing*

	KT score	*PW score*
Semantic matching		
PALPA 47 – Spoken word/Picture matching	39/40	(40)
PALPA 48 – Written word/Picture matching	34/40	(40)
Association Match Test	29/30	(30)
PPT – Picture/Picture matching	49/52	
PPT – Written word/Picture matching	38/52	
Synonym judgment		
ADA Battery (Auditory) – High vs. low imageability	67/80	(62)
ADA Battery (Written) – High vs. low imageability	61/80	(70)
Lexical decision		
PALPA 27 – Auditory	57/60	(56)
PALPA 4 – Auditory	156/160	(143)
PALPA – 27 – Visual	48/60	(58)
PALPA – 25 – Visual	134/160	(153)
Letter processing		
PALPA 21 – Letter discrimination	59/60	
PALPA 22 – Letter naming	0/26	
Reading		
PALPA 35 – Regularity	20/40	
PALPA 36 – Nonword reading	0/24	(24)
Repetition		
PALPA 9 – Frequency vs. imageability	63/80	(42)
Howard and Franklin (1988) – Regularity vs. imageability	186/240	(118)
PALPA 8 – Nonwords	26/40	(0)
Picture naming		
BNT	16/60	
PALPA 53 – Spoken	14/40	(27)
PALPA 53 – (Written)	35/40	
Writing to dictation		
PALPA 39 – Letter length	14/24	
PALPA 40 – Imageability vs. frequency	14/40	
PALPA 44 – Regularity	28/40	
PALPA 30 – Syllable length	7/24	
PALPA 45 – Nonwords	0/24	
Rhyme judgement (auditory presentation)		
PALPA 14	37/40	(33)
PALPA 15	60/60	(55)
Rhyme judgment (written presentation)		
PALPA 14	30/40	(20)
PALPA 15	29/60	(37)
Homophone judgment		
Regular homophones	41/50	(38)
Irregular homophones	31/50	(38)
Nonwords	39/50	

ADA = Action for Dysphasic Adults battery, BNT = Boston Naming Test, PALPA = Psycholinguistic Assessments of Language Processing in Aphasia, PPT = Pyramids & Palm Trees.

significantly more errors on low imageability relative to high imageability words on written (5 high vs. 14 low imageability errors), Fisher's exact $\chi^2(1) = 5.59$, $p < .017$, and auditory versions (3 high vs. 10 low imageability errors), Fisher's exact $\chi^2(1) = 4.50$, $p < .03$.

Picture naming and repetition

Consistent with other reported cases of NPFA, KT was severely impaired at spoken picture naming (see Table 2). The majority of KT's errors consisted of "no response" errors. In many of these cases, he would attempt to spell out his responses on his hand or leg, though this strategy did not benefit his performance in any reliable way. It is should be noted that his naming performance indicated a substantial decline from his previous ability when tested by a speech and language therapist over a year earlier (06/1999; PALPA 53, 31/40 correct).

We initially examined KT's repetition performance in a test taken from the PALPA battery, which manipulates performance on items that vary in both frequency and imageability (see PALPA 9: Table 2), together with a further test, devised by Howard and Franklin (1988), which manipulates regularity and imageability. Although KT was clearly impaired in single word repetition, none of the manipulated variables had any significant impact on his error performance. Unlike PW (and CB and CO), he did not make semantic errors, all errors being phonological in nature (PALPA 9: formal errors, 18%, target-related neologisms, 82%; Howard & Franklin, 1988: formal errors, 31.5%, target-related neologisms, 68.5%). A final test of non-word repetition also indicated substantial impairment.

Reading, single letter processing and visual lexical decision

KT demonstrated severely impaired word reading (see, for example, NART: Table 1). However, there was no evidence that regularity had an impact on his reading ability (PALPA 35: Errors, regular 10/20, irregular 10/20) and there was therefore no indication of "surface dyslexia" (see McCarthy & Warrington, 1984). On the contrary, he shows a "deep dyslexic" reading pattern, with semantic errors in reading aloud (e.g., three errors on this test: knife–"fork", swing–"see-saw", cow–"moo"), and an inability to read aloud nonwords (e.g., Coltheart, 1980; Lambon Ralph & Graham, 2000, for a recent review).

KT was also impaired in visual lexical decision, with all errors being false positives (i.e., a bias towards "yes" responses). While KT was able to determine whether pairs of words (one in upper and the other in lower case) were the same or different, he was unable to name presented letters, consistent also with cases of deep dyslexia (Coltheart, 1980). It is worth noting that his performance on this task had declined substantially from that of over a year earlier when tested by a speech and language therapist (06/1999; PALPA 22, Letter Naming, 23/26 correct).

Rhyme and homophone judgment

Given that KT was unable to read aloud nonwords, we examined whether he had difficulties in accessing phonology from print, or whether he had a more general phonological impairment. Two sets of rhyme judgment tests (PALPA 14/15) were used. When pairs of items were presented aurally, KT performed relatively well. However, KT's written word rhyme judgment was poor, which reflected his problems in accessing appropriate phonology from orthographic input. When performance on each test was compared, KT was significantly more impaired on visual word rhyme judgment relative to auditory word rhyme judgment: McNemar change test, PALPA 14, $\chi^2(1) = 4.50$, $p < .03$; PALPA 15, $\chi^2(1) = 41.80$, $p < .0001$. The majority of his errors on both tests were false positives (PALPA 14, 3 false negative and 7 false positive errors; PALPA 15, 8 false negative and 23 false positive errors), and this reflected the fact that he was performing at chance with written rhyme judgments and that his guessing strategy revealed a bias to say that two strings rhymed. Consistent with chance performance, closer examination of his false positive errors showed that KT selected equivalent numbers of visually similar and visually dissimilar nonrhymes.

KT was also impaired with homophone judgment, alongside poor performance at rhyme judgment. Homophone judgment differs from rhyme judgment in that no phonological segmentation is needed. Thus, poor performance on this task demonstrates that KT has deficits in access and short-term storage of appropriate lexical phonology from orthographic input, above and beyond possible impairments in phonological segmentation.[4] As can be seen from Table 2, KT was impaired on all forms of homophone judgment. However, he performed worse with irregular homophones compared with regular homophones, Fisher's exact $\chi^2(1) = 4.96$, $p < .022$. Consistent with rhyme judgment performance, the majority of his errors on these tasks were false positives (regular homophones, 7 false positives/ 2 false negatives; irregular homophones, 14 false positives/ 5 false negatives; Pseudohomophones, 8 false positives/ 3 false negatives), and thus KT was biased toward making "yes" responses.[5]

Writing to dictation

As we noted in the Introduction, there has been little investigation of written spelling performance in NFPA. KT's writing to dictation ability was therefore examined in a variety of tests (taken from the PALPA). In all cases, performance was impaired with a total inability to spell nonwords (see Table 2). Spelling ability was not affected by letter length, syllable length, frequency or regularity. It was influenced significantly by imageability: 7 high vs. 19 low imageability errors, Fisher's exact $\chi^2(1) = 15.82$, $p < .0001$. (It is important to note that KT had made few errors on these several tests when they had been administered by a speech and language therapist over a year previously: 06/1999; PALPA 39, 24/24 correct; PALPA 40, 35/40.) As in reading aloud, KT made semantic errors in writing to dictation; across all the tests KT made eight such errors: tongue–*mouth,* priest–*chaplain,* pill–*tablet,* wheat–*sheaf,* pupil–*student,* rat–*mouse,* child–*boy,* seat–*chair*.

Error performance across language tasks

As a direct comparison of his abilities across language production tasks, we compared KT's performance on a single set of items in spoken naming, written naming, repetition, spelling, and reading aloud (using the 40 stimuli that make up the PALPA 53 test). In each session, the 40 items were divided evenly across the five different language tasks: KT was asked to read 8 items, spell 8 items, etc., and all stimuli were administered across five testing sessions, with 2 weeks between each one.

KT's errors were classified into nine types, based on those proposed by Martin and Saffran (1992). We have employed this classification before with PW (Tree et al., 2001) and it was also used with CB and CO (Croot, Patterson, & Hodges, 1998; Majerus et al., 2001). The categories were: (1) formal paraphasias in which responses are phonologically but not semantically related to the target (e.g., thimble–*"nimble"*); (2) semantically related errors, which are subdivided into (a) semantic paraphasias that consist of a semantically-related word (e.g., glove–*"hand"*) and (b) semantic descriptions or circumlocutions that are semantically related to the target (e.g., anchor–*"In the boat and go in the water"*); (3) formal and semantically related paraphasias or mixed

[4] However, KT is impaired in visual lexical decision and therefore a word recognition impairment cannot be excluded, though this would not explain, by itself, a difference between regular and irregular items.

[5] It is interesting that on both rhyme and homophone judgment testing, KT produces a large number of false positive errors. This is a pattern that is typically associated with "frontal" or executive impairments, and this may well underline the examination of executive impairments in NFPA as a focus for future research. However, we would argue that such an impairment could not explain regularity effects in KT's homophone judgment accuracy. We would add that KT understood the tasks he was asked to do and despite the fact he found it extremely hard and time-consuming, he diligently endeavoured to complete the task.

errors (e.g., ladder–*"stir"* possibly via "stairs"); (4) unrelated lexical errors, which are lexical responses that bear neither a semantic or a phonological relation to the target (e.g., trumpet–*"boy"*); (5) target-related neologisms, which are nonwords that sound similar to the target-related response (e.g., dummy–*"dunny"*); (6) neologism on a semantic paraphasia, which is a nonword response that sounds similar to a semantic neighbour of the target (e.g., glasses–*"spektl"* via "spectacles"); (7) abstruse neologism, which is a response that is a nonword that is unrelated to the target (e.g., equal–*"wuk"*); (8) no-response errors.

Across all production tasks, written spelling to dictation, reading, and spoken naming were all significantly more impaired than written naming and repetition, with equivalent levels of correct performance on the latter two tasks: written naming and repetition versus spelling, Fisher's exact $\chi^2(1) = 3.66$, $p < .05$; written naming and repetition versus reading, McNemar change test, $\chi^2(1) = 13.09$, $p < .004$; written naming and repetition versus naming, McNemar change test, $\chi^2(1) = 13.09$, $p < .0001$.

Inspection of the error pattern across production tasks reveals that KT made a small number of semantic paraphasias in written naming, spelling, and reading, consistent with our earlier findings (see Table 3). The majority of other errors on those production tasks were failures to respond. The pattern of errors in word repetition is somewhat different, with the few errors being phonological in nature (i.e., formal paraphasias or target-related neologisms). Finally, in the case of spoken naming, almost all the errors consisted of failures to respond.

GENERAL DISCUSSION

One of the aims of the present study was to compare KT's language abilities with those of our previously reported case of NFPA, PW (Tree et al., 2001). Examination of KT's pattern of language impairment indicates the following.

1. He was impaired across modalities on semantic matching, association tasks, and auditory and written synonym judgment; and showed imageability effects across modalities on the latter task (PW demonstrated a similar pattern, showing imageability effects across modalities on synonym judgment, but her performance was significantly more impaired in the auditory modality).
2. KT was impaired on visual lexical decision, but otherwise normal on auditory lexical decision tasks, while the reverse was true of PW.
3. KT and PW were both equivalently impaired on rhyme judgment and homophone judgment, with worse performance in written relative to auditory modality presentation; they also both made large numbers of false positive errors on these tasks.
4. KT was impaired in reading words and was entirely unable to read single letters or nonwords.

Table 3. *Error response types across production tasks (PALPA 53—% responses for 40-item set)*

		KT					
Response type	*PW spoken naming*	*Spoken naming*	*Written naming*	*Repeat words*	*Spell words*	*Read words*	*Total*
Target Correct	68	35	87	87	70	50	66
Formal Paraphasia	5	8	0	7	0	5	4
Semantic Paraphasia	5	2	5	0	5	8	4
Semantic Description	5	0	0	0	0	0	0
Formal & Semantic Paraphasia	0	0	0	0	0	0	0
Target-related Neologism	0	0	3	3	0	2	1
Abstruse Neologism	7	0	0	3	0	0	1
Unrelated Lexical	0	3	0	0	0	0	1
No Response	10	52	5	0	25	35	23

He also made several semantic errors in reading aloud. Thus, his reading ability matched that of reported cases of deep dyslexia. PW was also impaired at reading, but was able to read nonwords, did not make semantic errors, and showed regularity effects.

5. Consistent with previous cases of NFPA, confrontational naming was severely impaired in both KT and PW, but naming impairment was more severe for KT with a greater number of no-response errors in his case.

6. KT's ability to repeat words and nonwords was impaired, but relatively well preserved compared to performance on other spoken language production tasks. Imageability and frequency did not affect his repetition; PW was far more impaired on repetition tasks, totally unable to repeat nonwords, made semantic errors and performed similarly to reported cases of deep dysphasia.

7. Letter length, syllable length, regularity and frequency had no impact on KT's spelling performance. However, imageability effects were present in spelling, as were semantic errors. Thus, KT's spelling performance matched that of reported cases of deep dysgraphia; PW was not tested on spelling ability.

8. An examination of KT's performance across production tasks determined that spelling, reading, and spoken naming performance were significantly more impaired than written naming and repetition. KT also made semantic errors in all production tasks with the exception of repetition; PW differed from KT in that reading performance was the most preserved, with equivalently severe impairment for repetition and naming.

Overall, KT demonstrates differential patterns of impairment across language production tasks: reading, spelling and repetition. In particular, KT makes semantic errors in both reading (deep dyslexia) and writing (deep dysgraphia). Given this striking pattern has not been reported before in the context of NFPA, we examine KT's performance on both reading and writing tasks individually, with specific reference to several other reported cases of NFPA discussed earlier (see Table 4).

Broadly speaking, two kinds of model have been used to address patterns of breakdown of language production. Single route interactive activation models (e.g., Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Foygel & Dell, 2000; Martin, Dell, Saffran, & Schwartz, 1994; Martin & Saffran, 1992; Martin, Saffran, & Dell, 1996) explain language production impairments in terms of either pathological decay or weakening of links between semantic and phonological representations. Dual-route modular models propose that tasks like reading, repetition, and spelling can take place via specific lexical and sublexical processing routes (see Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001; Hanley, Kay, & Edwards, 2002; Howard & Franklin, 1988; McCarthy & Warrington, 1984,

Table 4. *Language performance across nonfluent progressive aphasic cases*

	Reading performance			*Repetition performance*		*Spelling performance*		
Patient	*Regularity*	*Nonwords (% correct)*	*Semantic errors*	*Nonwords (% correct)*	*Semantic errors*	*Regularity*	*Nonwords (% correct)*	*Semantic errors*
PG	Yes[a]	50%[a]	No	N/A	No	N/A	N/A	N/A
LM	Yes[a]	32.5%[a]	No	N/A	No	N/A	N/A	N/A
RB	Yes[a]	15%[a]	No	77%[a]	No	N/A	N/A	N/A
CB	Yes[a]	90%[a]	No	N/A	Yes	N/A	N/A	N/A
CO	Yes	84%	No	4%	Yes	N/A	0%	Yes
PW	Yes	60%	No	0%	Yes	N/A	N/A	N/A
KT	No	0%	Yes	65%	No	No effect	0%	Yes

PG, LM: Croot et al. (1998); RB, CB: Croot et al. (1999); CO: Majerus et al. (2001); PW: Tree et al. (2001).

[a]Confirmed with test results provided to us that were not published in the original work (Croot, personal communication, 2004).

for detailed accounts). Currently, single route interactive-activation models have only focused on particular production tasks (i.e., reading, repetition or spoken naming). Thus it remains an open question whether a single route interactive activation model can adequately account for cases in which differential patterns of impairment are present across the entire range of language production tasks, and this is a challenge for the future. However, given that the present paper attempts to focus on the issue of the summation hypothesis (e.g., Hillis & Caramazza, 1991, 1995) as an adequate theoretical explanation for the pattern of semantic errors seen across different language production tasks in NFPA, we have chosen to couch KT's impairments with reference to a dual-route modular language production model (e.g., Howard & Franklin, 1988). This is because dual-route modular models can claim to account for impairments across production tasks through the principle of summation.

Nonfluent progressive aphasic performance in reading

Examination of KT's reading performance revealed a pattern of impairment consistent with reported cases of deep dyslexia (e.g., Allport & Funnell, 1981; Coltheart, 1980; Marshall & Newcombe, 1966; Newton & Barry, 1997). It is also apparent that KT's pattern of reading impairment is unlike any of the six other cases of NFPA discussed (see Table 4). Lambon Ralph and Graham (2000) claim that there have been no reported cases of primary progressive aphasia that present with a deep dyslexic reading performance. Since this comprehensive review, we are aware of only one progressive aphasic deep dyslexic case (described as a progressive anomic in the context of Pick's disease); BF (Cipolotti, 2000) made semantic errors in reading coupled with a complete inability to read nonwords. Unfortunately, little is made of BF's pattern of reading impairment, as this was not the major focus of the study. Nonetheless, although there was no systematic examination of both word and nonword reading performance in this case, it still provides tentative evidence in support of the principle of "summation." However, this particular case differs substantially from any of the previously discussed cases of NFPA, since BF's speech production was fluent and although anomia was present early in disease progression, it was modality specific. Therefore, to the best of our knowledge, this report of deep dyslexic performance in the context of NFPA is the first of its kind.

Within the context of a dual-route language model, we would explain KT's pattern of reading impairment as reflecting a severely damaged sublexical reading route, and a disrupted lexical reading route. KT therefore appears to depend heavily on lexical-semantic reading when reading single words. In line with such a proposal, Shallice (1988) and Newton and Barry (1997) propose that cases of deep dyslexia fall within three major types: (1) patients with impaired orthographic input/access to the semantic system; (2) patients with impairments of the semantic system itself; and (3) patients with impairments of accessing appropriate phonological output from the semantic system. Given that KT is impaired at written word lexical decision and written word semantic processing (on the basis of poor performance at both written word semantic matching and synonym judgement), it is possible that KT fits the performance of an "input" type deep dyslexic. However, in this paper we did not seek to examine KT's pattern of reading impairment in sufficient detail to discriminate between these possible selective levels of impairment in the lexical-semantic reading route. Thus for the present purposes, our study of KT's reading performance has demonstrated that his pattern of impairment is strikingly similar to previously reported cases of deep dyslexia (even though it results from a very different underlying aetiology) and that such a pattern of impairment can occur in cases of NFPA.

Although KT is totally unable to read aloud nonwords, we have demonstrated that he is able to carry out pseudohomophone judgment and that he shows regularity effects in homophone judgment. These data would suggest that the sublexical phonology is not entirely abolished in the case of KT (although we cannot rule out the possibility that correct pseudohomophone judgment was achieved by a visual matching strategy, and thus our findings must be accepted with some caution). Nonetheless, our findings are consistent with other studies of deep dyslexia suggesting that implicit activation of nonword pronunciation remains in such patients, despite the fact that nonword reading may no longer be possible (Buchanan, Hildebrandt, & MacKinnon, 1996; Veeken, Titov, Buchanan, Hildebrandt, & MacKinnon, 1996). Katz and Lanzoni (1992) also report that their deep dyslexic showed faster reaction times with rhyming, similarly spelled words (e.g., bribe–tribe) and slower reaction times on trials with nonrhyming, similarly spelled words (e.g., couch–touch) a pattern entirely consistent with controls. Katz and Lanzoni argue that this demonstrates that automatic phonological activation of printed words remains in deep dyslexia, despite severely impaired single word reading performance. A similar proposal was made by Patterson (1978), who demonstrated above-chance performance on spoken-written nonword matching in her reported cases of deep dyslexia. Thus, our findings are consistent with converging evidence suggesting that some implicit resources for the manipulation of phonology can still remain to a limited extent in a case of deep dyslexic reading performance, even when the ability to read aloud nonwords is abolished.

Nonfluent progressive aphasic performance in spelling and written naming

Overall, KT's pattern of performance on spelling tasks is consistent with cases of "deep dysgraphia" (e.g., Hillis, Rapp, & Caramazza, 1999; Newcombe & Marshall, 1980; Nolan & Caramazza, 1983). As far as we are aware, KT is the first reported case of NFPA who demonstrates a co-occurring deep dyslexic and deep dysgraphic performance, although (as we have stressed earlier) in most studies reading and spelling are not commonly tested together (and as we established earlier, writing performance in cases of NFPA has been largely overlooked). Such a co-occurrence of impairments has, however, been reported in other neuropsychological cases of different aetiology (Nolan & Caramazza, 1983).

In Table 4 we see that only one of the identified cases, CO (Majerus et al., 2001), was tested on written spelling performance and on written naming. Like KT, CO showed imageability effects and semantic errors in writing to dictation and a total inability to write nonwords. However, CO was equivalently impaired on spelling and written naming tasks. Consistent with our earlier discussion of KT's deep dyslexia, we would argue that his performance reflects a severely impaired sublexical phonological to graphemic route and a somewhat impaired semantic to orthographic lexical route (for further details on multiple routes of spelling, see Howard & Franklin, 1988). In agreement with the proposals of differential underlying impairments in cases of deep dyslexia, we would argue that deep dysgraphic performance may similarly reflect impairment at either, (1) auditory input/access to the semantic system or (2) the semantic system, or (3) an impairment of accessing appropriate orthographic output from the semantic system. Previous studies of deep dysgraphic performance have explained their cases in terms of damage at one of these different potential levels (for an example of an "input" type deep dysgraphic, see Nolan & Caramazza, 1983, and for an "output" type deep dysgraphic, see Hillis et al., 1999). Given that KT is relatively unimpaired at auditory lexical decision, discrimination, and rhyme judgment, but more impaired in spelling than written naming, we would argue that his pattern of impairment on spelling does not reflect a semantic-level impairment (at least not for high imageability words), but is consistent with a difficulty in phonological retrieval (that is also evident in impaired nonword repetition, and an inability to spell nonwords to dictation). However, as was the case in our discussion of KT's reading impairments, we did not seek to examine KT's spelling impairment in sufficient detail to discriminate between these possible selective levels of impairment in the lexical-semantic spelling route. Rather, we sought to demonstrate that his pattern of impairment is consistent with previously reported cases of deep dysgraphia, which is unusual in the context of cases of NFPA.

It is worth noting that KT was significantly less impaired on written naming relative to spoken naming. Such a pattern of impairment was not found in the only other study of primary progressive aphasia of both written and spoken naming (CO—Majerus et al., 2001). In this case there was no difference in written naming and spoken naming performance. On the basis of these two studies there is tentative evidence that primary progressive aphasia can result in differential degeneration in naming performance across modalities. Nonetheless, this type of pattern (i.e., superior written naming relative to spoken naming) has been reported in a case of primary progressive anomia (Snowden & Neary, 2003), and has also been reported in other nondegenerative neuropsychological cases (e.g., Hanley & Kay, 1997; Hanley et al., 2002; Marangolo & Basso, 1994). Rapp, Benzing, and Caramazza (1997) point out that such a pattern of impairment indicates orthographic lexical forms can be independently accessed for production without the need for mediating phonological processes. We would argue that KT's differential performance on spoken and written naming provides further evidence for this conclusion.

Semantic errors in nonfluent progressive aphasic production performance and the summation hypothesis

Earlier in the introduction we pointed out that, taken as a universal principle, the summation hypothesis (Hillis & Caramazza, 1991) predicts that the presence of semantic errors in language production is heavily dependent on the integrity of sublexical processing mechanisms, such that as long as such mechanisms exist for a particular output modality, semantic errors will not occur in that modality of output. Consistent with this general principle, from our sample of NFPA cases, semantic errors in repetition only occurred when coupled with abolition of nonword repetition (see Table 4). In the case of KT, the critical prediction that semantic errors will occur in a language production task when the sublexical route is abolished can also be applied to both reading and spelling (see Table 4).[6] Thus, we would suggest that the reports of "deep" language production performance across three cases of NFPA (CO, PW, and KT) provide further converging evidence in favour of the principle of summation in potentially explaining impairments in reading, repetition, and spelling.

One final observation is that KT differs from cases PW and CO in that he does not make semantic errors in repetition (see Table 4). However, KT's overall repetition performance shows a striking similarity to a further NFPA case, RB (see Table 4). Additionally, two recent papers, Hanley and Kay (1997) and Hanley et al. (2002), report two stroke cases with a somewhat similar pattern of impairment. Both cases showed impaired word and nonword repetition performance, with no semantic errors. However, unlike KT, both cases also demonstrated imageability effects in repetition. Hanley, Kay and colleagues argue (in line with the summation principle) that their patients' impairments reflected partial damage to both lexical and sublexical repetition routes and suggest that these routes were able to interact when repeating words.[7]

[6] This leads to the question of whether such a pattern of impairment occurs relatively frequently in cases of primary progressive aphasia. It is clear from a recent review of a large sample of primary progressive aphasic cases that little research has been done in terms of examining patient performance on spelling and written naming (Westbury & Bub, 1997). Thus it still remains an open question as to what proportion of cases of primary progressive aphasia present with a similar pattern of impairment to KT and CO.

[7] Note that, consistent with the overall principle of summation, KT's nonword repetition performance is better (at 65%) than either of the patients reported in their paper. MF was able to repeat 54% of nonwords and PS was able to repeat 10% of nonwords. Poorer performance on nonword repetition could explain why imageability effects were present in the repetition performance of both PS and MF but not present for KT. This finding would suggest that KT's relatively spared sublexical repetition route results in less dependence on the lexical-semantic repetition route, which ameliorates the effects of imageability. We should point out that a trend toward imageability effects was present in one of the repetition tests administered, and we would predict that as KT's repetition performance deteriorates, and the sublexical repetition route reaches a degree of damage equivalent to that seen in PS and MF, imageability effects will appear in KT's repetition.

The summation hypothesis and primary progressive aphasia

Although the focus of this report has centred on cases of NFPA, it is worthwhile considering the implications of the summation hypothesis for the pattern of degeneration seen in other cases of primary progressive aphasia. Of critical relevance is the fact that although we have demonstrated that deep disorders of several types can manifest themselves in the pattern of degeneration seen in cases of NFPA, such disorders are *not* seen in cases of semantic dementia (a fluent form of progressive aphasia). In the context of the summation hypothesis the reason for this is very straightforward; the language production impairments seen in cases of semantic dementia primarily implicate the *semantic* component of such tasks, otherwise relatively sparing sublexical processes involved in converting sound input to speech (in repetition), orthography to phonology (in reading), or phonology to graphemes (in writing to dictation). Therefore, as predicted by the summation hypothesis, the *preservation* of these mechanisms in cases of semantic dementia prevent the potential production of semantic errors in such tasks. Over-dependence on a mildly disrupted lexical route with no sublexical support for the three cases of NFPA that we have discussed (CO, PW, and KT) results in the production of semantic errors across a majority of (though not all) language production tasks; the opposing pattern of over-dependence on the sublexical route in cases of semantic dementia results in phonological errors in reading and spelling.[8] It is an intriguing point that this principle could imply that semantic dementia and NFPA constitute focal damage to alternative routes to successful production; namely semantic/lexical processes in cases of semantic dementia and sublexical/ phonological processes in cases of NFPA. However, it is important to point out that not *all* published cases of these two types of progressive aphasia fall into this proposal. Funnell (1996) provides some evidence that the surface dyslexic reading performance seen in the semantic dementia case EP runs contrary to the predictions of the summation hypothesis.[9]

Watt, Jokel, and Behrmann (1997) report a case of NFPA (JH) who demonstrates a surface dyslexic reading pattern, which suggests that it is not universally true that disease progression in case of NFPA focally implicates sublexical processing. Nevertheless, even this case supports the principle of summation in that nonword reading performance was entirely preserved and no semantic errors occurred. The authors explain JH's reading performance as not being due to a disruption of semantic processing in reading, but rather occurring as a result of insufficient access to phonological representations from semantics. This may imply that JH was not a usual case of NFPA at all, and exhibited performance more consistent with a case of progressive anomia (FM—Graham, Hodges, & Patterson, 1995). As a final point, it is worth observing that surface dyslexic type performance was also seen in cases PW and CO (see Table 4), although in the latter this was only demonstrated in reading irregular nonwords. In our discussion of PW's pattern of reading

[8] It is well documented that surface dyslexic reading often co-occurs with the disorder of comprehension seen in semantic dementia cases (Funnell, 1996; Hodges et al., 1992; Parkin, 1993; Patterson & Hodges, 1992), and surface dysgraphic performance has also been documented in such cases (Saffran, Coslett, Martin, & Boronat, 2003). In fact both Graham, Hodges, and Patterson (1994), and Ward, Scott, and Parkin (2000), provide evidence that the surface dyslexic patterns seen in their cases was consistently predicted by their semantic memory impairment, whilst Caramazza, Papagno, and Ruml (2000), present a fluent progressive aphasic who made exclusively phonological errors across language production tasks with disease progression.

[9] In this case it was suggested that, given that this hypothesis claims picture naming involves the same semantic-phonological pathway to output as irregular word reading, then if a phonological cue is given with a picture (which acts as the "summated" phonological support seen when reading irregular words), naming a picture with an irregular name or reading the same item should result in equivalent performance. This was found not to be the case with EP; however the author does point out that the cues used did not necessarily provide the full specification of the irregular pronunciation.

impairment we provided evidence that it could be explained using an account that involved the combination of partially damaged sublexical and lexical reading routes (see Tree et al., 2001, p. 485), and therefore we would argue that her performance is also not inconsistent with the principle of summation. Overall, with these cases in mind, it may be too extreme a position to believe all cases of NFPA focally implicate sublexical processing (a principle that implicitly seems consistent with the notion that nonfluent production reflects a disruption in accessing phonology). Nonetheless, it is striking that the pattern of error disorders seen in reading, spelling, and repetition for a number of patients with one or other type of primary progressive aphasia generally falls along theoretically predicted lines.

Conclusion

Our study has shown that KT demonstrates varied patterns of performance across language production tasks; distinct language processing routes appear to be differentially impaired in NFPA. Our detailed investigations have revealed both deep dyslexia and deep dysgraphia. Although deep dysgraphia has been reported in the context of primary progressive aphasia (Majerus et al., 2001), our report of a co-occurring presentation of deep dyslexia in the context of NFPA is the first of its kind. This begs the question whether such impairments are more frequent in relation to NFPA. Clearly, more detailed investigations of cases of NFPA must be conducted, and this demonstrates the importance of such research. Overall, the pattern of deep disorders seen in KT and in other reported cases (PW & CO) can be successfully accounted for with reference to the principle of summation, and moreover such results provide further evidence in support of dual (both lexical and sublexical) route models of language production.

Manuscript received 22 May 2002
Revised manuscript received 17 May 2004
Revised manuscript accepted 24 June 2004
PrEview proof published online 07 February 2005

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