



Lexical–semantic deficits in two patients with dominant thalamic infarction

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Abstract—Two patients with dominant thalamic infarction, one in the tuberothalamic artery territory, the other in the paramedian artery territory, demonstrated language impairment limited to word retrieval difficulties in spontaneous language and structured naming tasks. Using a cognitive neuropsychological model of lexical processing developed in the study of patients with cortical lesions, we carried out a detailed investigation of their lexical abilities. Both patients demonstrated impairment restricted to oral and written picture naming and oral naming to definition, and spared performance on tasks of lexical comprehension, oral word reading, and writing to dictation, as well as syntactic comprehension and production. Naming impairment disproportionately affected lower frequency words, and word substitutions often corresponded to objects that were semantically-related to target words. We propose that our patients' word retrieval impairments reflect a failure of thalamic input to effectively engage the cortical networks subserving lexical–semantic processing, leading to degraded levels of activation as the semantic system interfaces with subsequent stages of lexical processing. Copyright © 1996 Elsevier Science Ltd.

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Introduction

A number of lines of clinical investigation support the involvement of the dominant thalamus in language processing. These have included studies demonstrating language impairment in patients with surgical and vascular lesions of certain thalamic nuclei [2, 19, 44, 47] and during electrical stimulation of the thalamus [38, 39, 48], as well as changes in thalamic electrical activity during performance of language tasks [1].

Thalamic aphasia occurs most commonly following infarction in the distribution of the tuberothalamic artery, but it also develops occasionally with infarcts in the paramedian artery distribution [3, 19]. The aphasia is typically characterized by problems with lexical retrieval including semantic paraphasias in connected speech and difficulty in picture confrontation naming. Some inves-

tigators have described deficits in auditory or reading comprehension and in writing in these patients. Although the patients may speak with a reduced amount of verbal output, the syntactic form of responses is usually adequate. The patients typically can repeat words and sentences [3, 4, 17, 18, 32, 44]. These findings suggest that the thalamus is primarily involved in lexical–semantic aspects of language processing and is implicated less in syntactic and phonologic processing. These observations have provided much of the impetus for recent theories of the role of the thalamus and other subcortical structures in language [6, 8, 9, 51].

In general, the thalamic aphasia literature has focused on clinico-anatomic correlations. However, recent cognitive neuropsychological models of lexical processing [12, 45] (Fig. 1) may be useful in the study of language deficits resulting from dominant thalamic lesions. Dissociations in performance across lexical tasks in various brain-damaged patients have provided evidence for a lexical system composed of separate orthographic and phonologic input and output lexicons representing memory stores of familiar spoken and written words. Like-

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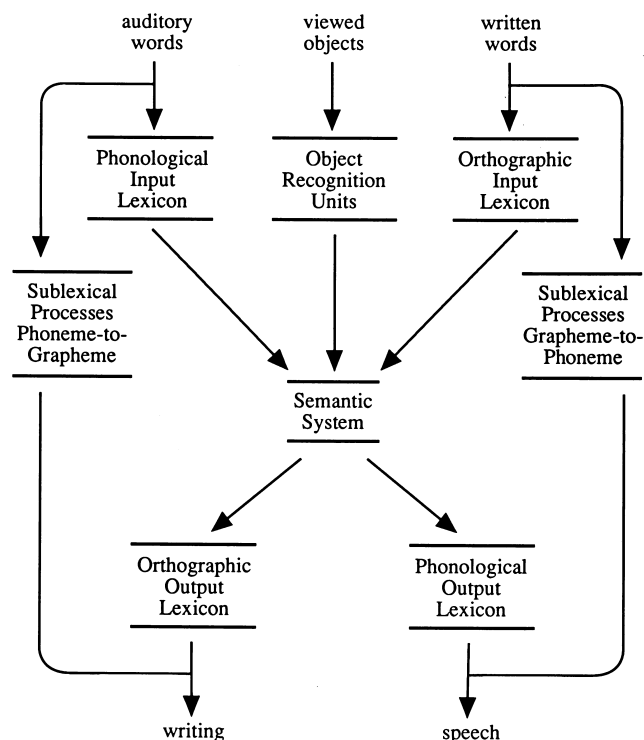


Fig. 1. Cognitive neuropsychological model of lexical processing [12, 45].

wise, an object recognition system provides a memory store for recognition of familiar viewed objects. The lexical and object recognition systems provide a processing advantage for previously experienced stimuli and allow one to acknowledge the familiarity of words or pictures. Subsequently, through activation of the semantic system, meaning relevant to all modalities of information is applied to words or pictures. Processing of novel spoken and written forms (i.e. nonwords such as 'voce' or 'quam') is accomplished with sublexical processes for converting graphemes to phonemes (in reading) and phonemes to graphemes (in spelling). These lexical and sublexical mechanisms presumably are highly interactive and operate in cascade fashion [26, 49]. For the sake of brevity, additional mechanisms involved in processing for other modalities of input, (e.g. tactile, auditory nonverbal) and modes of output (e.g. gestural) have been omitted from the current depiction of the lexical model. One way cognitive neuropsychologists evaluate the function of the various mechanisms is by examining patient performance (accuracy and error patterns) in single-word tasks such as oral and written naming, auditory and reading comprehension, oral reading, and writing to dictation [45].

Most evidence supporting this lexical model (Fig. 1) comes from the evaluation of deficits in patients with primarily cortical lesions [12]. However, because problems with lexical retrieval are a hallmark of the language dissolution observed in many patients with dominant thalamic lesions, the influence of the thalamus should also be considered in attempts to elaborate functional models of lexical processing. This paper presents data

from extensive language testing of two patients with thalamic infarctions. We will consider our results in the context of both the lexical model and theories of the role of subcortical structures in language processing [8, 9, 35, 51].

Methods

Subjects

Case 1. BD was a 45-year-old, right-handed woman with a high school education who worked as a bookkeeper. On the day of her initial symptoms, she awoke with evidence of mild confusion and slight dysarthria, and she misnamed her daughter. She drove to work, but required an hour to do so instead of the usual 15 min; afterward she had no recollection of the drive. Later that day, she called home and sounded so confused that her daughter brought her to the hospital.

On admission, she was slightly lethargic but easily arousable, fully oriented, and able to complete detailed bedside clinical testing. She recalled three of three objects after 5 min of distraction. Language was fluent but moderately anomnic with occasional verbal paraphasic errors. She had difficulty naming parts of common objects. She failed to comprehend only compound commands, whether presented orally or in written form. Her sentence repetition was normal. Her finger naming was good, and there was no right-left confusion. She could do simple additions but not subtraction of serial sevens. She produced only four words beginning with the letter S in 1 min. She performed contrasting motor programs quite well without evidence of echopraxia. She was neither perseverative nor distractible. Her constructions were well performed. She exhibited no hemispatial neglect or extinction. Her detailed neurologic examination was normal except for some unsteadiness during tandem walking and absent ankle jerks. A computed tomographic (CT) scan of BD the following day demonstrated a focal area of low density in the left anterior thalamus with ill-defined margins consistent with an acute infarction.

Although BD initially had significant cognitive deficits, she improved rapidly over the ensuing months. Standardized testing was administered 5 months after her stroke in conjunction with experimental testing to be described later. On the Western Aphasia Battery [30], she had an Aphasia Quotient of 92.2 and a pattern that was consistent with anomnic aphasia. She scored 48/60 on the Boston Naming Test [28], which is slightly below normal for her age and education [36, 37]. On the Wechsler Memory Scale—Revised [53], her recall for short narratives on the Logical Memory subtest was below average (16th percentile for immediate recall; 29th percentile for delayed recall), while her figure recall on the Visual Reproduction subtest was above average in both immediate and delayed conditions (98th and 97th percentile, respectively). Finger tapping and grooved peg-board performance were within normal limits for both hands. Results of most tests of frontal functions (Wisconsin Card Sorting Test [20], six categories and 14 perseverations; go-no go paradigm, 1 error; Trail Making Test, part A, 50th percentile, part B, 60th percentile) were normal. However, BD made several perseverative errors on recursive figures.

Case 2. WT was a 59-year-old, right-handed retired salesman with a college education. On the day of his neurological event, he was found by the police standing by his car on the roadside in a confused state. Seven days later he was referred to the Gainesville Veterans Affairs Medical Center. He had no recall of events over the prior week. He felt he was having some trouble getting words out. He denied weakness, sensory impair-

ment, or visual difficulty. His past medical history was significant for a myelodysplastic syndrome with refractory anemia.

On clinical neurological examination WT was alert and cooperative but disoriented to time and place. In his spontaneous language, the quantity of verbal output was significantly reduced because of marked word-finding difficulty; he was only occasionally successful in producing nouns. His naming of objects to confrontation was severely impaired, but he could name colors. Examiners judged his phonology and grammar to be intact; he produced a number of sentences with embedded clauses. They noted neologisms and paraphasic errors. He had excellent comprehension of low frequency object names and grammatically complex commands. His ability to repeat was normal, even for nonwords and sentences with embedded clauses. His reading was normal and his writing was severely anomic. His ability to calculate was reduced and he demonstrated right-left confusion. His praxis was normal and he exhibited no neglect. His affect was flat. He repeated six digits forward. He recalled zero of three objects after several minutes of distraction. His language impairment impeded assessment of remote memory. When asked to sort pictures of famous people, he correctly placed all the American presidents into one category but he sorted other pictures without any consistent pattern. The remainder of his neurologic examination was normal.

WT's CT scan, obtained 5 days after the stroke, demonstrated only an old lacunar infarction in the right putamen. A magnetic resonance imaging (MRI) scan obtained 10 days after the stroke demonstrated a new ischemic lesion in the left thalamus.

Between the time of his stroke and his death one and a half years later from complications of the myelodysplastic syndrome, WT exhibited considerable recovery of language function. However, he demonstrated a persistent and profound change in personality. Once a strongly self-motivated and gregarious salesman, he became passive and taciturn. Even though he agreed to follow-up language and imaging studies, in practice it proved very difficult to complete these studies as he constantly complained of either fatigue or lack of time. For this reason we obtained little standardized neuropsychological data beyond the aphasia assessment. Four months after his stroke, WT's performance on the Western Aphasia Battery [30] yielded an aphasia quotient of 91.2 and a pattern consistent with anomic aphasia. He named only 22/60 items on the Boston Naming Test [28], producing both semantic and phonemic paraphasic errors and indicating a significant word retrieval impairment for his age and education [36, 37]. On the Wisconsin Card Sorting Test [20], WT attained only three categories and made 26 perseverative responses. We were unable to obtain any other standardized neuropsychological testing.

Lesion analysis

The lesions in these two patients as defined on MR scans were mapped by a modified camera lucida technique onto plates from a stereotactic atlas [35, 46]. The thalamic infarct for BD was in the polar (tuberthalamic) artery distribution and appeared to involve portions of ventral anterior (VA) and ventral lateral (VL) nuclei, posteriorly affecting fibers in the internal medullary lamina (IML), and extending inferiorly to include the inferior thalamic peduncle (ITP) (Fig. 2). The infarct for WT was in the paramedian (interpeduncular profundus) artery distribution, involving the VL, dorsomedial (DM), lateral posterior (LP), centromedian (CM), and ventral posterior lateral (VPL) nuclei (Fig. 3). Detailed mapping of each patient's lesion can be viewed elsewhere [35].

Standardized aphasia testing 4–5 months after stroke revealed a pattern consistent with anomic aphasia in both cases. The nature of these naming difficulties was the focus of our experimental testing. Both patients provided informed consent to participate in research testing.

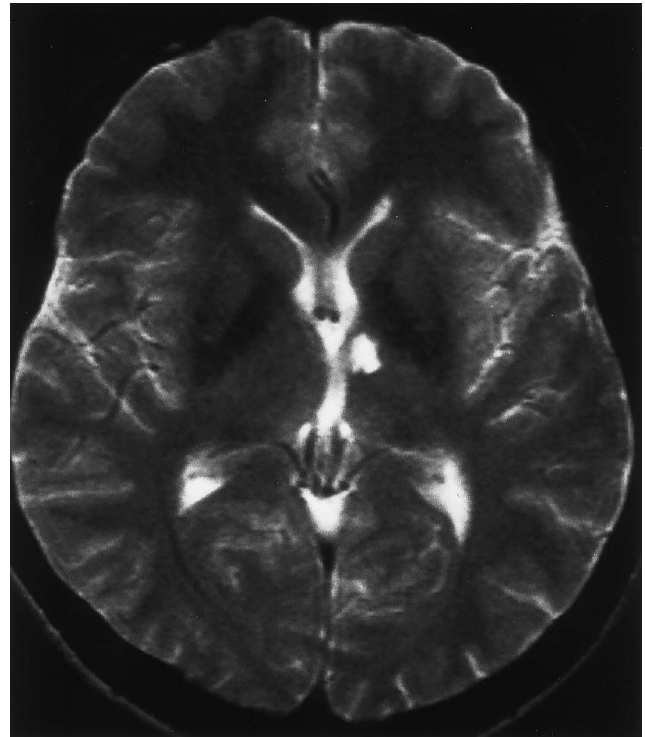


Fig. 2. MRI scan of BD showing area of thalamic infarction in the distribution of the tuberthalamic artery on the left.

Experimental tasks

We administered to both patients a lexical-semantic test battery (Florida Semantics Battery, unpublished test) [42] developed to assist in localizing the source of lexical deficits with respect to the cognitive neuropsychological model described earlier. The battery included seven tasks of lexical processing: oral picture naming, written picture naming, naming to auditory definition, oral word reading, writing to dictation, auditory word/picture matching, and written word/picture matching. In the two matching tasks, subjects matched a target word to one of four picture choices: the target and three semantic coordinate distractors (e.g. APPLE–cherry–pear–orange). In naming to definitions, the patients named objects corresponding to descriptions that included information regarding semantic category, function, context, and associated knowledge (e.g. “This is a vegetable that can make you cry” described the target word onion). These descriptions avoided clues regarding the visual qualities of the targets such as color or distinctive features. Each of the seven tasks incorporated the same 120 items from 12 different semantic categories (e.g. fruits, transportation, and tools). There were 40 high, 40 medium, and 40 low frequency words in the English language [14].

Examiners coded responses in the three naming tasks as correct if the patients provided the target word or an acceptable synonym. Errors were coded as semantic (including coordinate, associate, or superordinate word substitutions or circumlocutions), visual (incorrect responses that shared basic physical features with the target object and not semantically related to the target object), semantic+visual (semantic coordinate responses that were also visually similar to the target), phonologic (incorrect responses sharing more than 50% of the phonologic information with the target), unrelated, neologistic, or no response.

Patient performance on each lexical task was compared to that of control groups comprised of three women 40–45 years who had completed high school (i.e. subjects demographically similar to BD) and three men 61–65 years of age who had

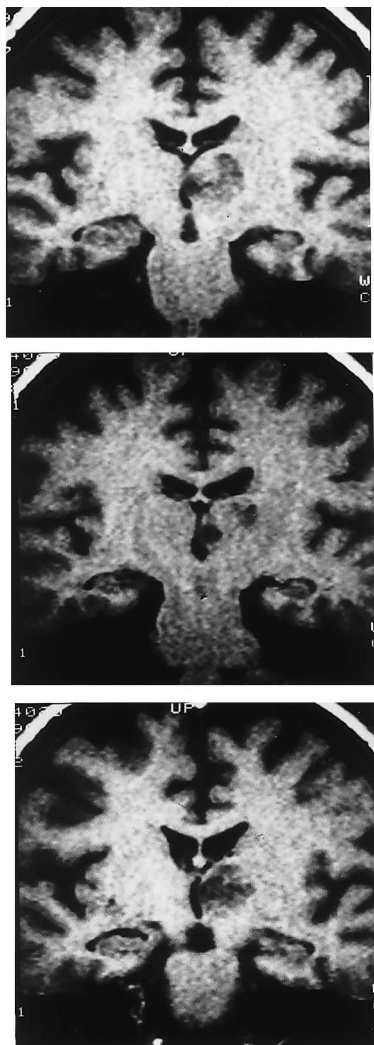


Fig. 3. MRI scan of WT showing area of thalamic infarction in the distribution of the paramedian artery on the left.

completed high school (i.e. subjects demographically similar to WT) [50]. Because of similar high levels of performance by both groups, the control data were collapsed into one group to which both thalamic patients' performances were compared.

In addition to the lexical processing tasks, patients completed tests of syntactic processing to evaluate abilities in an alternate language area that has typically been regarded as intact in patients with thalamic lesions. These included a test of sentence comprehension incorporating direct and indirect object relations (e.g. He showed the parents the boy's pants. vs He showed the parents' boys the pants.) [21]. The patient pointed to one of four pictures that corresponded to the spoken target sentence. One distractor picture was a syntactic foil and two distractor pictures were semantic foils.

To assess syntactic production, both patients completed a Story Completion task [16] that elicits 14 different syntactic structures across 28 trials. Productions incorporating the target syntactic structures were scored as correct.

Results

As shown in Table 1, normal control subjects made no errors and both thalamic patients made only occasional errors in four of the lexical tasks: oral word reading,

writing to dictation, and the word/picture matching tasks. In the writing to dictation task, each patient produced occasional simple misspellings of the correct target words.

In the three naming tasks, however, both patients had moderate naming difficulties compared to the overall 0–5.8% error rate observed in normal control subjects. Overall, BD produced 10–15.8% errors and WT produced 15.8–24.2% errors across the three naming tasks. In all three naming tasks, both patients produced significantly more errors when naming low frequency items compared to naming high frequency items. BD made 25–27.5% errors on low frequency items and only 0–10% errors on high frequency items. WT made 27.5–35% errors naming low frequency items and 7.5–12.5% errors on high frequency items. Statistical analysis (Fisher's exact test comparing high and low frequency items) indicated that frequency was a significant factor for both BD and WT in two of three naming tasks each (Table 1).

Of the 45 total errors BD produced across the three naming tasks, 73% were semantic+visual errors, 11% were semantic (and not visual) errors, and 16% were omissions. WT produced a total of 76 errors in the three naming tasks, including 33% semantic+visual errors, 30% semantic errors and 27% omissions, with an occasional neologistic, phonemically-related, or unrelated response. The few errors produced by normal control subjects included occasional perceptual, semantic+visual, or omission responses.

We also analyzed our patients' naming impairments for semantic category effects across the 12 semantic categories included in the test stimuli. For each patient, we calculated the mean number of errors per semantic category collapsed across the three naming tasks (i.e. a total of 30 trials per category). BD made a mean of $3.8 (\pm 2.2)$ errors per category. For no category did her number of errors differ from the mean by more than two standard deviations, suggesting no significant effect of semantic category. WT made a mean of $6.3 (\pm 3.9)$ errors per category for the 12 categories. His performance for the category musical instruments (16 total errors) was greater than two standard deviations from his error mean. However, this finding is confounded by the fact that the musical instrument category consists of many lower frequency items. Moreover, given that we used a cut-off of two standard deviations and tested twelve semantic categories in each of two patients, this may represent a chance finding.

In contrast to their naming performance, both patients performed at high levels on the syntactic tasks. BD produced no error in either the syntactic comprehension or production task. WT made two errors on 20 sentence comprehension items, and one error in 28 items testing production of 14 syntactic structures.

In summary, both of our patients, tested at 4–5 months after thalamic infarction, demonstrated language deficits largely restricted to naming tasks, with preserved abilities in lexical comprehension, oral word reading, writing

Table 1. Percent error rates across lexical tasks

Lexical Task	BD	WT	Controls <i>n</i> =6	
			Mean	S.D.
Oral picture naming				
Overall (<i>n</i> = 120)	15.8	23.3	1.82	(0.97)
High frequency (<i>n</i> = 40)	10.0	12.5	0.83	(1.29)
Mid frequency (<i>n</i> = 40)	12.5	22.5	0.83	(1.29)
Low frequency (<i>n</i> = 40)	25.0	35.0	3.75	(2.62)
Fisher's exact test*	<i>P</i> = 0.139	<i>P</i> = 0.034		
Written picture naming				
Overall (<i>n</i> = 120)	10.0	15.8	1.67	(1.90)
High frequency (<i>n</i> = 40)	0	7.5	1.25	(3.06)
Mid frequency (<i>n</i> = 40)	2.5	12.5	1.67	(2.04)
Low frequency (<i>n</i> = 40)	27.5	27.5	2.50	(3.16)
Fisher's exact test	<i>P</i> < 0.0005	<i>P</i> = 0.037		
Name to auditory definition				
Overall (<i>n</i> = 120)	14.2	24.2	3.07	(1.70)
High frequency (<i>n</i> = 40)	7.5	12.5	0.83	(2.04)
Mid frequency (<i>n</i> = 40)	7.5	32.5	3.33	(2.04)
Low frequency (<i>n</i> = 40)	27.5	27.5	5.00	(2.24)
Fisher's exact test	<i>P</i> = 0.037	<i>P</i> = 0.161		
Oral word reading (<i>n</i> = 120)	0	0	0	
Write to dictation (<i>n</i> = 120)	1.7	3.3	0	
Auditory wd/pic match (<i>n</i> = 120)	0	0.8	0.42	(0.71)
Written wd/pic match (<i>n</i> = 120)	0	†		†

*Fisher's exact test for high frequency versus low frequency items.

†Not administered.

words to dictation, and syntactic comprehension and production. Naming failure was greatest for lower frequency items and semantic + visual paraphasias were the predominant naming error type.

Discussion

We report the results of language testing in two patients with infarcts of the dominant thalamus whose aphasia, like that of previously described patients [3, 19, 44], was characterized by significant word retrieval impairment in both oral and written naming. In addition, we showed that the degree of naming impairment was related to word frequency, with low frequency items disproportionately affected. In characterizing our patients' cross-modal naming impairments with respect to the proposed lexical model (Fig. 1), their impairments could be due to: (1) a defect in the function of the semantic system; (2) a defect in the phonological and orthographic output lexicons concurrently; or (3) a defect in the interface between the semantic system and the output lexicons. We will consider each of these possibilities in turn.

Crossmodal naming failure could stem from an impairment in the function of the semantic system which is involved in processing for both verbal and written modes of lexical output. However, both WT and BD demonstrated excellent comprehension abilities in word/picture matching tasks using semantic distractors which place

demands on semantic processing and are typically impaired in patients with semantic system impairments [23, 25]. Moreover, WT demonstrated intact semantic processing in category sorting tasks for pictures and words contrasting both distant (e.g. transportation vs furniture) and close (e.g. farm animals vs jungle animals) semantic category distinctions (116/120 pictures; 119/120 words) and he performed at ceiling (23/24 correct) in an Odd Man Out task in which he selected two semantically associated pictures from a choice of three (e.g. cigarette–ashtray–*sandwich*). Thus, semantic processing in our patients appeared to be intact, at least insofar as they were capable of making some fine conceptual distinctions.

Concurrent impairments in the phonological and orthographic output lexicons could result in crossmodal word retrieval failure with intact word comprehension as has been described by Miceli and his colleagues [34] for their patient SF with a cortical lesion. Our thalamic patients' preponderance of semantic errors in oral and written naming differs from those of SF who produced mostly omission errors, however. Likewise the oral naming impairment in our thalamic patients differs from other patients with selective impairment at the level of the phonological output lexicon. For example, Kay and Ellis [29] reported that their anomic patient EST demonstrated phonological errors in naming as well as significant difficulties in oral reading, and attributed the impairment to difficulty in retrieving lexical phonological output. In contrast, Caramazza and Hillis [5] described two patients

who exhibited semantic errors in oral naming and oral reading and not in written naming, and ascribed the impairment to the phonological output lexicon.

The oral naming errors of our thalamic patients are like those of Caramazza and Hillis' [5] patients and may reflect a similar functional impairment of phonological lexical retrieval. However, the thalamic lesions in our patients also affected performance in written picture naming. Our thalamic patients' accurate oral reading performance may relate to preserved abilities in sublexical processing which when combined with moderate degradation of lexical-semantic function are able to circumvent the production of semantic errors in the reading domain [22, 34, 40]. Hence, different sorts of difficulties may emerge with impairment at the same level in the lexical system with the differences attributable to details of the nature and extent of malfunction of the lexical system.

If the lexical retrieval impairments of our patients BD and WT relate to concurrent impairments of the output lexicons, they differ substantially from other patients with impairments at this level of the lexical system. However, we might consider the alternative possibility that our patients' lexical retrieval impairments stem from a defect in the interface between the semantic system and the output lexicons resulting in intact performance in comprehension tasks and cross-modal naming deficits. This type of lexical impairment has been proposed previously to account for naming deficits in patients with primarily cortical lesions [43, 56]. As we discuss shortly, these analyses of the mechanisms of our patients' naming impairments may not be sufficient.

The lexical deficits described in our two patients were qualitatively similar despite the rather different anatomic loci of their thalamic lesions, suggesting that the deficit reflects a functional impairment that occurs with lesions of multiple thalamic nuclei and pathways or a structure affected in both patients' lesions, such as the internal medullary lamina. Thus, the pattern of deficit may provide a clue to a fundamental role of thalamo-cortical systems in language.

In a preliminary theory that we have described in detail elsewhere [35], we have suggested that thalamic aphasia is caused by disruption of a mechanism incorporating the frontal cortex, the nucleus reticularis thalami, and the centromedian nucleus which functions in the selective engagement of cerebral cortex. Nucleus reticularis consists of a thin shell of GABAergic neurons surrounding the thalamus that regulates thalamocortical transmission via projections to both thalamocortical neurons and thalamic interneurons. It has previously been implicated in mechanisms of attention and intention [52].

In BD, the thalamic lesion extended inferiorly to involve the portion of the nucleus reticularis that receives fibers from the frontal cortex via the inferior thalamic peduncle, and posteriorly to affect the internal medullary lamina that carries fibers between frontal cortex and the centromedian nucleus. In WT, the lesion involved the

centromedian nucleus of the thalamus which is interconnected with the frontal lobes through fibers travelling in the internal medullary lamina of the thalamus, and comprises another part of the system, including nucleus reticularis, whereby the frontal lobes regulate the relay of thalamic input to the cerebral cortex [35]. Thus, whereas the lesions in the two patients were in different thalamic nuclei, they may have affected the internal medullary lamina and implicated the same fundamental system in both patients. The further anatomic, physiologic, and behavioral data supporting this hypothesis are beyond the scope of this discussion and are explained in a separate paper [35].

According to the selective engagement theory, the cortico-thalamo-cortical system acts to selectively engage cortical networks instantiating lexical and semantic information. Defective selective engagement by the thalamus degrades the function of networks supporting lexical-semantic function, yielding concepts that are somewhat less sharply defined but not grossly distorted. In the semantic system, these less sharply defined concepts may be adequate to support nearly perfect performance on word/picture matching tasks, category sorting tasks, and odd man out tasks that we used to test comprehension and semantic function. Errors would occur in these tasks only with substantial misdefinition or lack of definition of concepts, and not with minor distortions that may occur in association with a thalamic lesion.

However, correct naming requires unambiguous activation and selection of one and only one item from an output lexicon. The less efficient operation of the semantic system due to reduced thalamic input may lead to less differentiation of activation levels for output to phonological and orthographic lexical systems [7]. The semantic system's activation of multiple lexical representations that share many visual and/or semantic features may result in misnaming errors that are close visual+semantic paraphasias or, alternatively, failure to achieve the minimal threshold of activation required to produce any one word, resulting in a complete failure to respond (anomia). Semantic and visual+semantic paraphasias and no response errors were common in both of our patients. Thus, we could revise the notion mentioned above that our two patients had adequate semantic processing and propose a third possibility that the thalamic lesion may have produced a disorder of the semantic system itself that *functionally* led to a disconnection between the semantic system and the output lexicons and affected word retrieval across naming tasks. This disconnection is not absolute, but represents a loosening of connections between semantic and phonological mechanisms to the degree that word retrieval at times is impeded.

Likewise it is possible that the critical effect of defective selective engagement is in thalamic projections to networks subserving the phonological and orthographic output lexicons. In this scheme, thalamic dysfunction would also produce a functional disconnection between sem-

antic and lexical output systems by failing to optimize communication between the systems. If this lexical output system proposal is correct, one may wonder about the frequent occurrence of semantic errors in our patients. Caramazza and Hillis [5] provided an explanation for semantic errors arising at the level of the output lexicon. They proposed that semantic representations activate multiple semantically-related lexical output representations and the most highly activated lexical representation is ultimately produced. When the target representation is less accessible, semantic errors will result. In our thalamic lesion patients, defective thalamic engagement of lexical output networks may have reduced the accessibility of target words and resulted in semantic errors.

Defective selective engagement can also account for the lexical effects we observed. High frequency words, which tend to be more familiar and earlier learned words [13, 24], are easier to activate than are low frequency words which require greater activation to reach threshold. Therefore, low frequency words are likely to be more vulnerable to the effect of lesions that degrade the operation of the lexical-semantic network, such as thalamic infarcts.

The cortico-thalamo-cortical network involved in selective engagement includes the frontal cortex, a region that is critical for lexical retrieval in verb naming tasks [10, 33, 57]. Therefore, one may predict that lexical impairments following thalamic lesions may also affect verb naming. We did not test this hypothesis in WT and BD, and now await the rare opportunity to examine other patients with discrete thalamic infarcts to replicate our findings and to test these hypotheses further.

An alternative hypothesis to the selective engagement theory is that our patients' naming impairments resulted from disruption of a cortico-striato-pallido-thalamo-cortical loop, incorporating the VA and VL nuclei, that is involved in lexical processing. Wallesch and Papagno [51] suggested this loop chooses the best of several cortically generated lexical alternatives. Crosson [9], on the other hand, believed the same loop activates the release of language segments into an output stream after a semantic verification process. In BD, the involvement of VA could account for deficient lexical selection with either of these theories [9, 51]. For WT, deficient lexical selection could be an indirect result of disrupting the activity of the cortico-striato-pallido-thalamo-cortical loop through VA and VL because the intralaminar nuclei project strongly to the striatum [9]. At this time, we do not favor either the Wallesch and Papagno [51] or Crosson [9] hypotheses because lesions that are relatively confined to the striatum (e.g. striatocapsular infarcts) often produce minimal or no language deficits [11, 27, 31, 39, 41, 54]. When aphasia does occur with such lesions, it may be related to decreased circulation in the territory of the middle cerebral artery. A detailed accounting of the evidence supporting this contention is beyond the scope of this paper and the interested reader is referred to detailed discussion

of this matter elsewhere [35, 55]. However, it must be acknowledged that patients with lesions confined to the striatum have not received the thorough assessment of lexical and semantic function conducted in the present study.

The competing hypotheses regarding the mechanism of thalamic aphasia discussed above deserve further testing. One approach would be to give the lexical-semantic tasks used in this study to patients with lacunar infarcts confined to the striatum. If they show deficits similar to those of our patients, then the cortico-striato-pallido-thalamo-cortical loop would be implicated. If they do not show similar deficits, then the frontal lobe-nucleus reticularis-intralaminar selective engagement system would be favored. The adequacy of the latter hypothesis can be further tested through better definition of the locus and the extent of thalamic lesions and further definition of the effect of thalamic lesions on lexical-semantic function, other dominant hemisphere processes such as praxis, and nondominant functions such as visuospatial function, facial recognition, and emotional processes.

Finally, it is possible that the effect of the thalamic lesion in these patients was neither to disrupt selective engagement nor to interfere with the function of cortico-striato-pallido-thalamocortical systems, but rather to reduce the total cognitive resources these patients were able to bring to the tasks we used. In this conceptualization, our subjects performed poorly on naming tasks simply because these were the most difficult tasks we employed. We think this is unlikely because, on face value, it is not obvious that our naming tasks were more difficult than, for example, a category sorting task. Our colleagues recently reported a patient with predominantly frontal lobe deficits who performed well on these same naming tasks but was impaired in semantic category sorting [15]. Furthermore, on this account we might expect that the three naming tasks incorporated in this study would require differing levels of processing resources and result in different error rates across tasks. However, both subjects made comparable rates of errors in oral and written naming to pictures and naming to definitions. Definitive testing of the reduced total resource hypothesis would require redesign of the experimental tasks so that ceiling effects were no longer present in the control groups.

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