THALAMIC THOUGHT DISORDER: ON BEING "A BIT ADDLED"*

Anjan Chatterjee[1], Robert Yapundich[1], Mark Mennemeier[2], James M. Mountz[3], Chakri Inampudi[3], Jullie W. Pan[1,4], Galen W. Mitchell[1]

(Departments of [1]Neurology, [2]Rehabilitation Medicine, [3]Radiology and [4]the Center for Nuclear Imaging Research, The University of Alabama in Birmingham, Birmingham, Alabama, USA)

Abstract

Humans can generate and maintain relatively coherent trains of thought in natural discourse. The neural mediation of this ability and the phenomenology of its breakdown are not well understood. We report a case of a woman with paramedian thalamic strokes involving the mammillothalamic tract, intralaminar nuclei, parts of the dorsomedial and ventral lateral nuclei bilaterally. She presented with a dense amnesia and confusion typical of the syndrome of bilateral paramedian thalamic infarcts. Her Tc-99m HMPAO brain SPECT scan showed decreased thalamic and basal ganglia blood flow. General diminution of cerebral blood flow and areas of further diminution in the right frontal, left temporal and left temporoparietal regions were also observed. Although her amnesia was characteristic of diencephalic amnesia, her most striking clinical feature was a bizarre, disconnected and at times incoherent speech output. Analysis of her speech revealed relatively preserved lexical and morpho-syntactic linguistic production. By contrast, analysis of the macrostructure of her discourse revealed frequent unpredictable topic shifts that were completely unconstrained by contextual factors. Many of her shifts were intrusions from previous topics. We interpret her severely disordered speech output as representing the surface manifestations of a thought disorder (rather than as a language disorder per se) characterized by an inability to maintain and appropriately shift themes that normally guide discourse. Median and intralaminar thalamic nuclei appear to be critical for the neurophysiologic regulation of thalamocortical and striatocortical circuits, which in turn may be critical for the functional regulation of contextually appropriate transitions of thought.

Introduction

Humans can generate and maintain relatively coherent trains of thought. Themes can be traced across sentences in conversation as ideas are developed and expressed. Humans can also shift from one set of ideas to others. These shifts may vary from gradual transitions to abrupt changes; the nature of these shifts being dictated by pragmatic and contextual settings in which ideas are conveyed. William James (1890) referred to these stable and dynamic aspects of thought as "substantive" and "transitive" states of consciousness, respectively.

The current interest in the neuropsychology of consciousness has focused

* Portions of this paper were presented as a poster at the 24th Annual Meeting of the International Neuropsychological Society at Chicago in February, 1996.

Cortex, (1997) 33, 419-440

primarily on what James might have considered substantive aspects of consciousness. Amnesia, blindsight, unilateral neglect, prosopagnosia, and anosognosia provide important data for our investigations into the neurobiology of consciousness (Milner and Rugg, 1992). By contrast, little is known of transitive aspects of consciousness. How are transitions of thought neurally regulated? How would the breakdown of this regulation be manifested? How are such questions best pursued empirically? If transitive aspects of thought are distinct from substantive ones, then these functions might be mediated and controlled by different neural systems. Brain damage, under some circumstances, might then impair thought transitions selectively. James (1890) suggested that dynamic aspects of thought are revealed in the "rhythm of language". Thus, an individual's discourse in relatively unstructured settings might offer insight into the structure of thought transitions. Impairment of the ability to regulate thought transitions would be evident in speech, but would not be explained by disruptions of local features of language (phonology, syntax, lexical-semantics). Rather, the disordered speech would be the surface consequence of a dynamic disorder not captured adequately by our understanding of traditional aphasic disorders.

With these considerations in mind, we report observations in a patient with bilateral paramedian thalamic infarcts. Patients with bi-thalamic lesions are amnestic, frequently appear confused, and may produce bizarre confabulations. This amnesia has been well studied (Bogousslavsky, Regli and Uske, 1988b; Castaigne, Lhermitte, Buge et al., 1981; Crosson, 1992; Graff-Radford, Tranel, Van Hoesen et al., 1990; Von Cramon, Hebel and Schuri, 1985). By contrast, the confusional state in this syndrome has been examined only rarely (Luria, 1977). In this case study we focus on the nature of our patient's confusion. We examine the structure of her discourse and consider its implications for the neuropsychology and neurobiology of transitions in thought.

Case History

LB, a 70 year old right-handed woman with a history of hypertension and coronary artery disease, was found lying on the floor of her home in a confusional state. Despite being able to answer questions, she could not offer a cogent explanation for why she was lying on the floor. In the first few days after admission to the hospital, she was somnolent but arousable. Her speech was dysarthric initially. She was severely amnestic. Her level of arousal improved after a few days. Despite this improvement, LB remained disoriented to place and time, and her responses to questions were often bizarre. For example, when asked where she was she replied South America. When asked how old she was she replied that she was 17. A few moments later she said she was in her 30s. Her digit span was five digits forward. She was unable to repeat two digits backwards. When asked to count backwards from 20, she did so till 11 before talking about something else. She could follow simple one and two step commands and repeat phrases without difficulty. Her spontaneous speech was fluent. On verbal fluency she named 11 words beginning with the letter F in 60 seconds. Some of these were low frequency words such as fearsome, folly and forlorn. She was unable to generate words for the semantic categories of animals, tools or fruits. This poor performance seemed to be a result of her quickly switching topics rather than an inability to generate words. She performed gestures to command of transitive movements (such as hammer) well. When asked to follow simple simultaneous motor commands, such as "open your mouth and raise your hand" she performed the movements accurately but would

perseverate, repeating the movements five or six times till she was asked to stop. By contrast, when asked to repeat motor sequences such as the Luria gestural sequence of "fist-edge-palm", she was able to follow and mimic these sequences, but on her own could not produce the sequence more than once. She could repeat 10 bits of information in a short story, but was unable to recall the story after five minutes. She could recite the days of the week, the letters of the alphabet and a short jingle (row, row, row your boat...). Her conversational discourse was disconnected, confabulatory and at time bizarre (examples in the Appendix). One week after her admission her visiting daughter woke her and LB immediately began drilling her on math problems. The daughter (in her 40s) later explained that her mother used to test her similarly over 35 years before.

LB's visual acuity was poor because of a prior history of macular degeneration. Her cranial nerve exam was notable for restricted up-gaze. She did not have motor or somatosensory deficits. Her clinical evaluation included normal electrolytes, thyroid function tests, vitamin B12 level, negative syphilis serologies, ANA titres and rheumatoid factor. Cerebrospinal fluid analysis was normal. The electroencephalogram revealed mild background slowing. Her hospital course was notable for gradual improvement in her level of arousal, without improvement in her amnesia or confusion. At no time during her hospital stay was LB aggressive or hostile towards the hospital staff. She, however, did demonstrate a kind of inertia and did not herself initiate activities. She required considerable supervision and constant encouragement to persist in physical therapy tasks. She continued to be severely amnestic and confused for at least a year after her initial presentation.

Neuroimaging

Magnetic Resonance Imaging (MRI)

A clinical 1.5 Tesla MRI scans revealed bilateral medial thalamic infarcts in the paramedian artery distribution. To better delineate her diencephalic lesions, a 4.1 Tesla 256×256 T1 weighted MRI scan was obtained using an inversion recovery gradient echo sequence (Figure 1). The two axial MRI images in which lesions were evident were matched to relevant images in Haines' neuroanatomic atlas (Haines, 1995). This atlas allows correlation of axial brain imaging slices with neuropathologic sections. The figures relevant to our case, Figures 6-1 to 6-4 from Haines (1995) show thalamic nuclei in relation to basal ganglia structures at these levels. Based on comparison with these sections, LB's lesions involved the mammillothalamic tract, the internal medullary lamina and parts of the dorsomedial nucleus and ventral lateral nuclei bilaterally. Small portions of the ventral anterior nuclei may also have been damaged.

Single Photon Emission Computed Topographic (SPECT)

LB was scanned twice, one week and two months after her stroke using the same protocol (Mountz, Zhang, Liu et al., 1994b). Regional cerebral blood flow (rCBF) SPECT brain scans were done after intravenous injection of 25 mCi of Technetium-99m Hexamethylpropyleneamine oxime (Tc-99 HMPAO). The patient was injected in a quiet environment with eyes closed in a dimly lit room. After five minutes of tracer uptake the patient was scanned on the ADAC dual-head Genesys Anger Gamma Camera (ADAC laboratories, MILPITAL, CA). Scanning parameters were 128 stops (64 stops per head), 30 seconds per stop using a 25 cm field of view resulting in a pixel dimension size of 0.196 cm on edge. Images were reconstructed using a Butterfield filter back projection

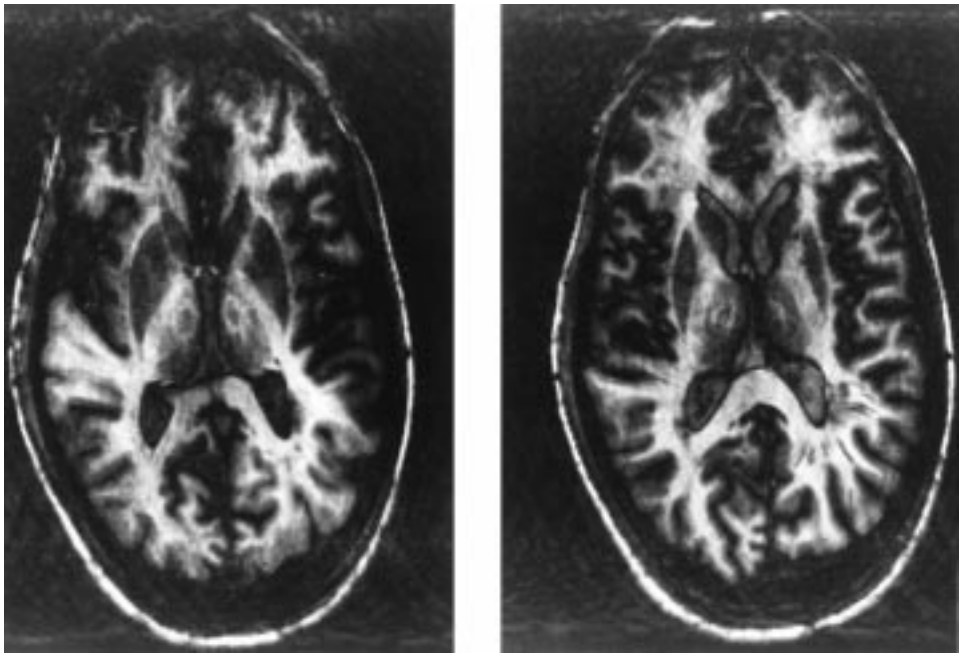

Fig. 1 – *4.1 Tesla 256 × 256 T1 weighted MRI scan (TR/TE = 2500/17.5 msec) obtained one week after her stroke. The bilateral paramedian infarcts are evident in both panels.*

algorithm (frequency cut-off = 0.22 Nyquest, order 6) (Liu, Harris, Inampudi et al., 1995). Attenuation correction was performed using the Chang (1978) algorithm. A reference system was employed during the scan such that the brain could be sectioned parallel to and sequentially above the canthomeatal line (Mountz et al., 1994b). Analysis of the basal ganglia and thalami were performed by co-registration of the brain SPECT scan with the MRI scan using a reference system technique developed at this institution (Mountz, Wilson, Wolff et al., 1994a). Counts were characterized as the average counts in the regions of interest (ROI) normalized to cerebellar and whole brain counts (Mountz, 1991). Cortical ROIs were generated using a circumferential ROI technique standard at this institution (Mountz, 1994). Briefly, an outer boundary is computer drawn at the 50% counts per pixel threshold, and an inner boundary is drawn at a distance eight pixels radially inward. The annulus is subdivided into twelve equal angular sections for cortical count semi-quantification. The ratios of maximum and average cortical counts and cerebellar whole brain counts were calculated identically for both scans.

The first scan showed increased rCBF in the thalamus (left thalamus = 1.05, right thalamus = 1.1; normal thalamic uptake = 0.96 + 0.04) interpreted as representing luxury perfusion. The follow-up scan (Figure 2) showed diminished bi-thalamic perfusion (left thalamus = 0.70, right thalamus = 0.74) and diminished basal ganglia flow (left = 0.76, right = 0.73; normal 0.94 + 0.05). Basal ganglia hypoperfusion is compatible with the fact that intralaminar nuclei have major efferents to the striatum (Groenewegen and Berendse, 1994; Jones, 1985). She also

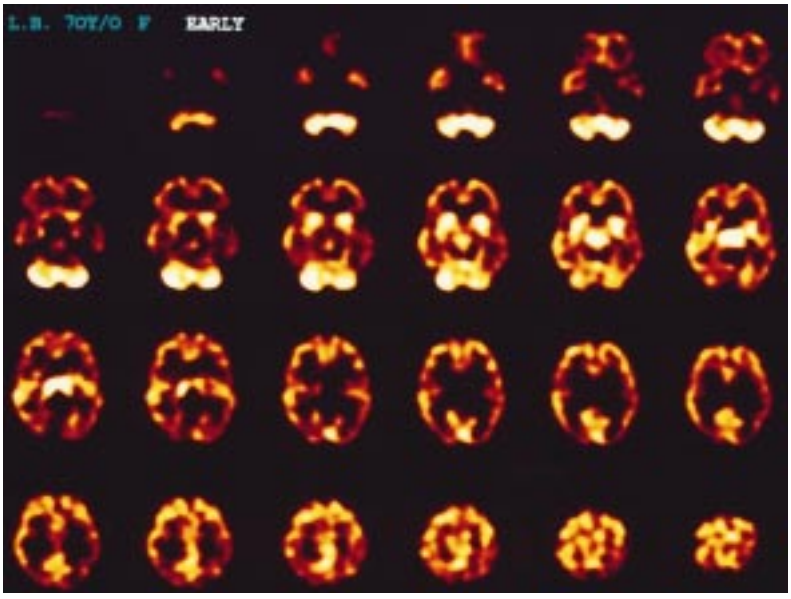

Fig. 2 – *(a) Sequential Tc-99m HMPAO brain SPECT scan sections (approximately 5 mm thick) obtained one week post-stroke. The scans progress from the ventral (top left) to the dorsal part of the brain (bottom right). High tracer uptake were noted in the thalamic regions bilaterally, which when integrated with the abnormalities on the MRI scan were interpreted as the "luxury perfusion" phase of infarction.*

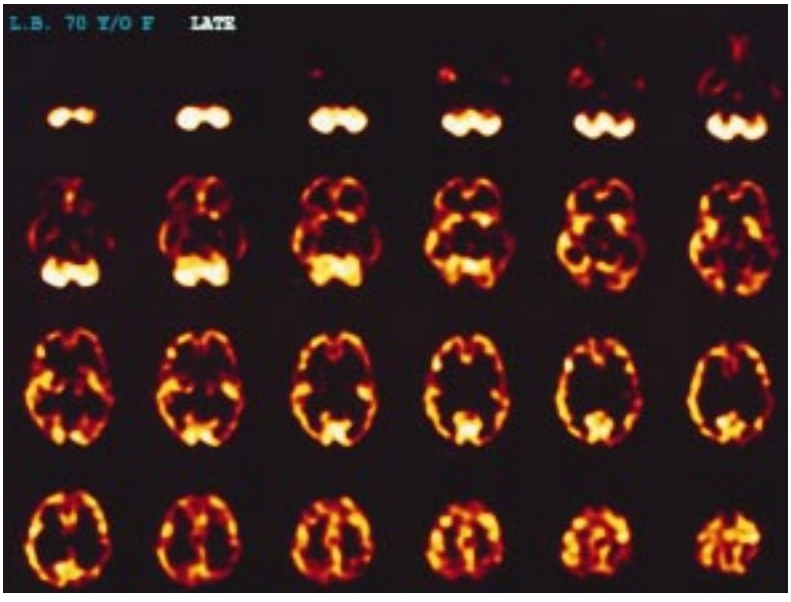

Fig. 2 – *(b) Sequential transverse Tc-99m HMPAO brain SPECT sections (approximately 5 mm thick) from the patient 2 months post-stroke. Marked reduction of regional cerebral blood flow to the thalamus is indicative of bilaretal thalamic infarction.*

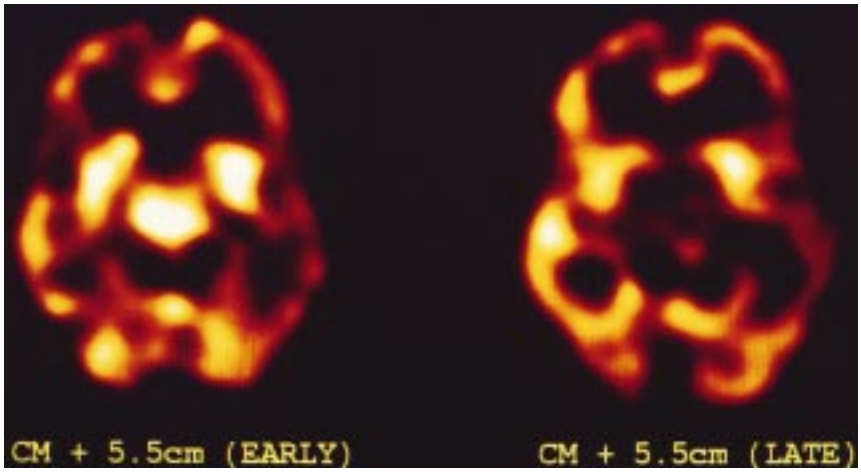

Fig. 2 – *(c) Tc-99m HMPAO brain SPECT scan sections oriented parallel to and 5.5 cm above the canthomeatal line. The changes in blood flow to the thalamus from the one week post-stroke scan (left) are contrasted with the marked reduction of blood flow typically associated with infarction on the 2 month blood flow scan section (right).*

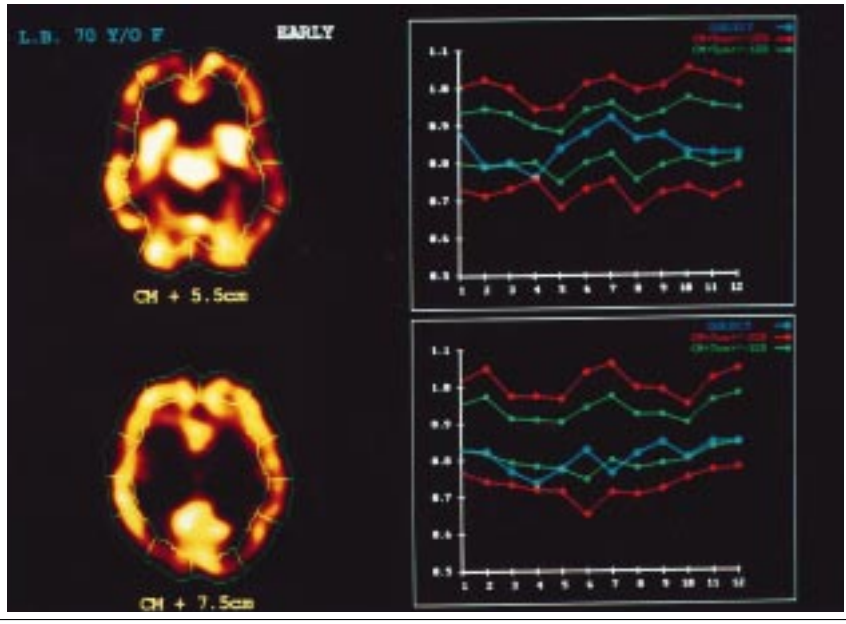

Fig. 2 – *(d) Tc-99m HMPAO brain SPECT scan sections from the one week post-stroke scan positioned parallel to and 5.5 cm (top left) and 7.5 cm (bottom left) above the canthomeatal line. To the right of the images are shown the semi-quantitative cortical circumferential profile of the cortical to cerebellar indices as described in the text. The blue line represents the patient, the green line represents one standard deviation from age matched normal controls (n = 9), and the red line indicates two standard deviations from the mean of age-matched normal controls. The x axis represents cortical regions progressing from the twelve to one o'clock position (cortical region #1) to the eleven to twelve o'clock position (cortical region #12). The y axis has the units of cortical region counts divided by cerebellar counts. The semi-quantitative curves show reduction of regional cerebral blood flow in the left temporal region (top curve region 4 at canthomeatal + 5.5 cm). The bottom curve shows reduction of left posterior parietal cerebral blood flow.*

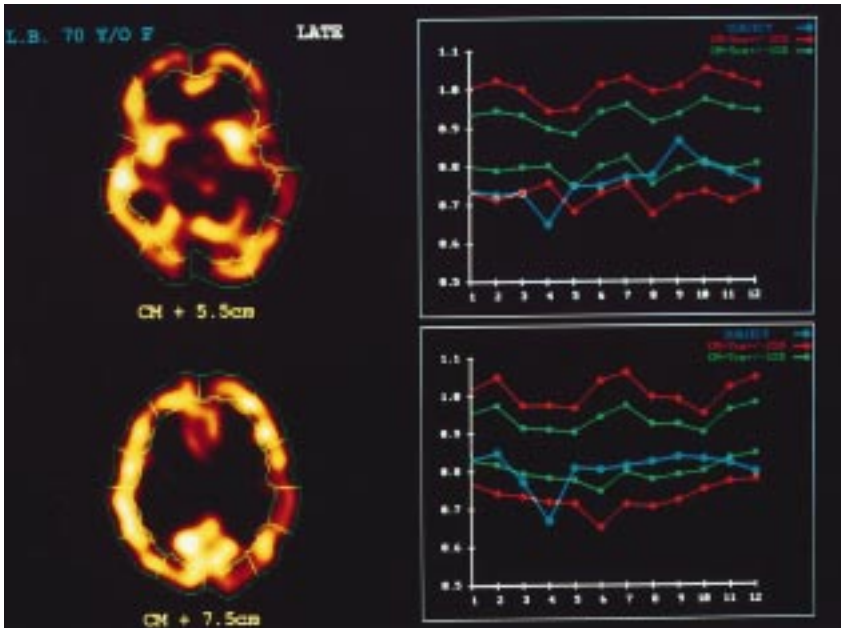

Fig. 2 – *(e) Tc-99m HMPAO brain SPECT scan sections from the two month post-stroke scan presented in the format described for figure 2d. The semi-quantitative profile for the level canthomeatal + 5.5 cm shows further reduction of left temporal blood flow (region 4) and the right frontal blood flow (region 12). At canthomeatal + 7.5 there is also further reduction of regional blood flow at the left parietal lobe (region 4).*

had global reductions of cerebral blood flow and further diminution (greater than two standard deviations from normal age matched sample) in the left temporal, posterior parietal and right anterior prefrontal cortices.

Behavioral and Neuropsychological Testing

Attempts at formal psychometric testing were compromised by LB's inability to persist on tasks. On the Dementia Rating Scale (Mattis, 1976) she scored 44/144, with the following sub-scores: attention 14/37, initiation and perseveration 16/37, construction 0/6, conceptualization 14/39, memory 0/25. We have doubts about the validity of these scores since she frequently seemed not to be trying to perform the required task. Further testing of general intelligence was not pursued since these assessments seemed unable to capture the peculiarities of her deficit.

LB's performances on the Wechsler Memory Scale-Revised (Russell, 1975) and the California Verbal Learning Test (CVLT) (Delis, Kramer, Kaplan et al., 1987) were characteristic of a dense amnesia. She was unable to recall any information after a 20 minute delay and did not display any learning on the CVLT. Responses to the logical memory stories were confabulated and there were more intrusions than correct responses on the CVLT.

Language

LB followed simple commands and repeated phrases easily. Confrontation naming could not be tested adequately because of her poor visual acuity. Two extended interviews conducted by AC were videotaped. The first (lasting 56 minutes and 43 seconds) was done 11 days after her stroke and the second (43 minutes and 25 seconds) three weeks after her stroke. These interviews followed the general format of a neurological history and mental status exam, however, the content drifted from the traditional confines of a clinical interview, as described below. The taped interviews were transcribed independently by two raters. Point-to-point agreement by each word was 0.77 for the first interview (when she was dysarthric) and 0.86 for the second interview. Differences were decided by AC who conducted the interview, after a review of the transcriptions and the videotapes. These transcripts served as the corpus from which subsequent analyses were performed.

Analysis of Syntax

In focusing on the nature of LB's discourse, we wished to examine the structure of her language production. To assess her syntactic abilities we adapted the method described by Saffran et al. to analyze narrative production (Saffran, Berndt and Schwartz, 1989). Subjects are asked to narrate well known fairy tales, such as the Cinderella story. From 150 words of these narrations, grammatical properties of language output can be quantified. Attempts to get LB to generate fairy tale narratives were unsuccessful (see Appendix). However, to confirm the general impression of preservation of lexical selection and structure, two portions of LB's discourse were selected (one from each session) in which she talked without interruption for over 150 consecutive words. In addition, the beginning of the first interview was analyzed similarly despite the disconnected nature of this part of her discourse. Since this corpus was not derived from the general domain of telling fairy tales we recognize that the comparison of LB's production to these normative data has limitations. However, our focus was to learn if the microstructure of her production more closely resembled normal production than agrammatic production. If so, one would not ascribe her disordered output to fragmentation of local features of language. As shown in Table I, her performance for proportion of closed class words, noun/pronoun ratio, presence of obligatory determiners, noun/verb ratio and frequency of verb inflections was close to normal. In addition, the proportion of words in sentences, the proportion of well formed sentences, indices of sentence structure elaboration and embeddings also resembled normal performance where she spoke without prompts for extended periods. In sections of her discourse comprised of smaller segments, indices of structural sentence elaborations were reduced. It is of interest that that impoverished structural elaboration may reflect a conceptual rather than a syntactic disorder (Berndt, 1991).

Analysis of Discourse Macrostructure

Description (also see Appendix). LB did not violate conventions of turn

taking in conversation and generally maintained prosodic contours of speech appropriate to the context of what was being discussed. She made appropriate eye contact and seemed to behaviorally adopt the interactional style one would expect of conversation as a joint activity. When not required to produce a verbal response to simple commands (touch your nose), she performed well. She also performed simple constrained verbal commands, such as repeating digits or phrases, accurately. Her replies to simple questions in which her verbal response was unconstrained fit the form of the question but were often bizarre. For example, when asked where she was, she replied "South America". With open-ended questions her discourse became disorganized. At times she seemed to start appropriately but quickly drifted off to new topics. The reason for the content of her speech and topic transitions was usually indecipherable from the context. At times she seemed 'stuck in set'. At other times previous topics would intrude upon her discourse. Fragments of themes would surface unpredictably and occasionally more than one theme would be fused within single statements. To better characterize these dynamic features of her disordered speech we analyzed her topic transitions formally.

Topic Shifting. Garcia and Joanette (1994), following Crow (1983), point out the insensitivity of traditional aphasia assessment tools to adequately describing disorders of discourse. Rather, they develop an approach predicated on the notion that an overarching theme or topic guides conversational discourse. Utterances cohere to each other by their relevance to the topic. To confirm the clinical impression that LB's transitions were severely disordered, we adapted their method of topic shift analysis. Garcia and Joannette (1994) distinguish interpretative from sequential issues in such analysis. Interpretative issues are concerned with the intended meaning of an utterance. Sequential issues are concerned with connections between utterances and the dynamics of topic shifts. While the bizarreness of LB's responses (Appendix) raise interesting questions of interpretation, we will only touch on these tangentially. We focus mainly on sequential issues in LB's discourse.

The text from LB's conversational discourse was coded by utterances. A noun phrase or sentence was considered an utterance. Some portions of her

TABLE I

Microstructural analysis adapted from Saffran et al. (1989). Their data from controls and agrammatic aphasics are included to show LB's general pattern across both interviews LB's first column reports analyses sections of from uninterrupted speech. The second column reports analyses from the beginning of the first conversation, which includes interrupted discourse.

	Ag	Cntrl	LB	LB
Proportion closed class words	0.33 (0.08)	0.56 (0.04)	0.67	0.46
Noun/pronoun ratio	8.93 (0.27)	1.60 (0.85)	1.39	1.43
Noun/verb ratio	2.93 (0.92)	1.18 (0.30)	1.18	0.94
Frequency of verb inflection	0.34 (0.29)	0.98 (0.05)	0.85	0.92
Determiner/noun ratio	0.37 (0.24)	1.00 (0)	0.90	1.00
Proportion of words in sentence	0.43 (0.14)	1.00 (0)	0.88	0.95
Proportion sentences well formed	0.33 (0.22)	0.91 (0.05)	0.92	0.90
Sentence structural elaboration	1.07 (0.22)	3.76 (6.00)	2.88	2.20
Embedding index	0.28 (0.04)	0.53 (0.33)	0.47	0.25

discourse were considered fragments and not included in the analyses. These fragments were false starts, unconnected prepositional phrases or isolated words from which a theme could not be deciphered. Fragments were excluded from the analysis to be conservative in our estimations of topical shifts. The specific points at which a topic shift were identified was then coded for the characteristics of these shifts. These topic shifts were categorized for 1) type of shift, 2) reason for shift and 3) context of shift. Operational definitions for these categories are given in Table II. The types of shift were either topic initiation (TI), topic shading (TS), topic drift (TD), renewal of topic (R), unexpected (U) or conflation (C). Only TI and TS are considered normal shifts. Reasons for the shifts were coded as the end of a topic (ET), answering previous question (AQ), reacting to an outside stimulus (OS) or unknown (U). The context of the shift refers to whether the topic shift related to information or knowledge shared by both participants. These were categorized as referring to topics previously initiated externally by the examiner (ET), topics previously initiated internally by the patient (IT) and unknown (U).

AC and RY coded topic shifts independently. Differences in coding were resolved by consensus. The results for both interviews are depicted in Table III and Figure 3. In summary, in the first interview she produced 1854 words, 405 utterances, and 111 topic shifts. In the second interview she produced 2250 words, 403 utterances and 113 shifts. The average length of her utterances increased slightly from 4.6 words to 5.6 words per utterance, but

TABLE II

Operational definitions for classification of topic shifts, adapted from Garcia and Joannette (1994) following Crow (1983). Only classifications applicable to this case are included. We introduced classifications marked by (*) to better capture LB's specific topic transitions

1. Type of Shift
1.1 Topic Initiation (TI). This shift represents an attempt to introduce a new topic at the beginning of a conversation, or after a period of silence.
1.2 Topic Shading (TS). The introduction of a new topic by first establishing its connection with a topic already on the floor.
1.3 Topic Drift (TD).* The tangential shifting of a topic from the topic on the floor.
1.4 Topic Renewal (TR). A shift back to a previous topic after one or more topics have since intervened.
1.5 Unexpected (U). The shift to a new topic in a way that is unexpected and is difficult to understand from the context in which it occurred.
1.6 Conflation (C).* The new topic forms a hybrid combining at least two previously mentioned distinct topics.
2. Reason for Shift
2.1 End of Topic (ET). When the topic shifts because it is clear that the speaker has nothing more to say about the previous topic.
2.2 Answering an earlier question (AQ).* The shift can be traced to answering or responding to a previous question or point despite intervening topics.
2.3 Outside stimulus (OS). The shift in topic is triggered by some environmental stimulus.
2.4 Undetermined (U). The reason for the shift could not be determined.
3. Relation to Context
3.1 Externally Initiated Topic (ET).* A shift to a topic previously introduced by the interviewer.
3.2 Internally Initiated Topic (IT).* A shift to a topic previously introduced by the patient.
3.3 Unknown (U). The context within which the shift occurred could not be determined.

utterances per shift remained unchanged at 3.6 utterances per shift. Topic shifts only refer to shifts made by LB and not the interviewer. Most of her shifts were either completely unexpected or inappropriate renewals of previously mentioned topics. Very few (less than 5%) of her topics shifts were either the initiation of or shading into new topics, both considered appropriate and normal types of shifts. In the vast majority of instances (> 90%) the reason for her shift was not evident from the context of the discourse. Almost half of these unexpected shifts introduced novel topics with no previously shared referent, and the rest were intrusions. The intrusions were about evenly divided between topics the patient had previously introduced, and topics introduced by the interviewer.

A Control Group

Although it seemed self-evident that LB's topic shifting was strikingly abnormal, we compared her discourse to that of three patients with right hemisphere strokes. Right brain damage is also associated with disorders of discourse (Bloom, 1994). These patients were video taped for extended interviews regarding the awareness of their left sided weakness. Detailed discussion of these patients are published elsewhere (Chatterjee and Mennemeier, 1996). MR had a discrete right cortical stroke confined primarily to motor cortex. EM had a large right basal ganglia-internal capsule hemorrhagic stroke. HS had a large right hemispheric stroke involving most of the distribution of the middle cerebral artery. Their shifting characteristics are outlined in Table III. In brief, they initiated far fewer shifts than LB. Aggregating their data, 90% (19 of 21) of the shifts they produced were appropriate as compared to 1 or 2% of LB's shifts. These appropriate shifts were invariably considered topic shadings, in which they introduced personal anecdotal information related to the topic at hand. Of the two inappropriate shifts, one was a topic drift which also introduced personal anecdotal information, but was not germane to the topic. The second was unexpected and was the single shift produced by these patients which was similar to the shifts LB produced commonly. In summary, these patients' discourse throws into relief the severe abnormality of LB's topic shifting behavior.

TABLE III

General discourse and shifting characteristics of LB and the three right brain damaged control subjects

	Words	Utterances	Words/ utterance	Shifts	Utterances/ shift	% normal shifts
LB 1	1854	405	4.6	111	3.6	2
LB 2	2250	403	5.6	113	3.6	1
MR	1218	159	7.6	7	22.7	86
EM	803	134	6.0	4	33.0	100
HS	1275	192	6.6	10	19.2	90

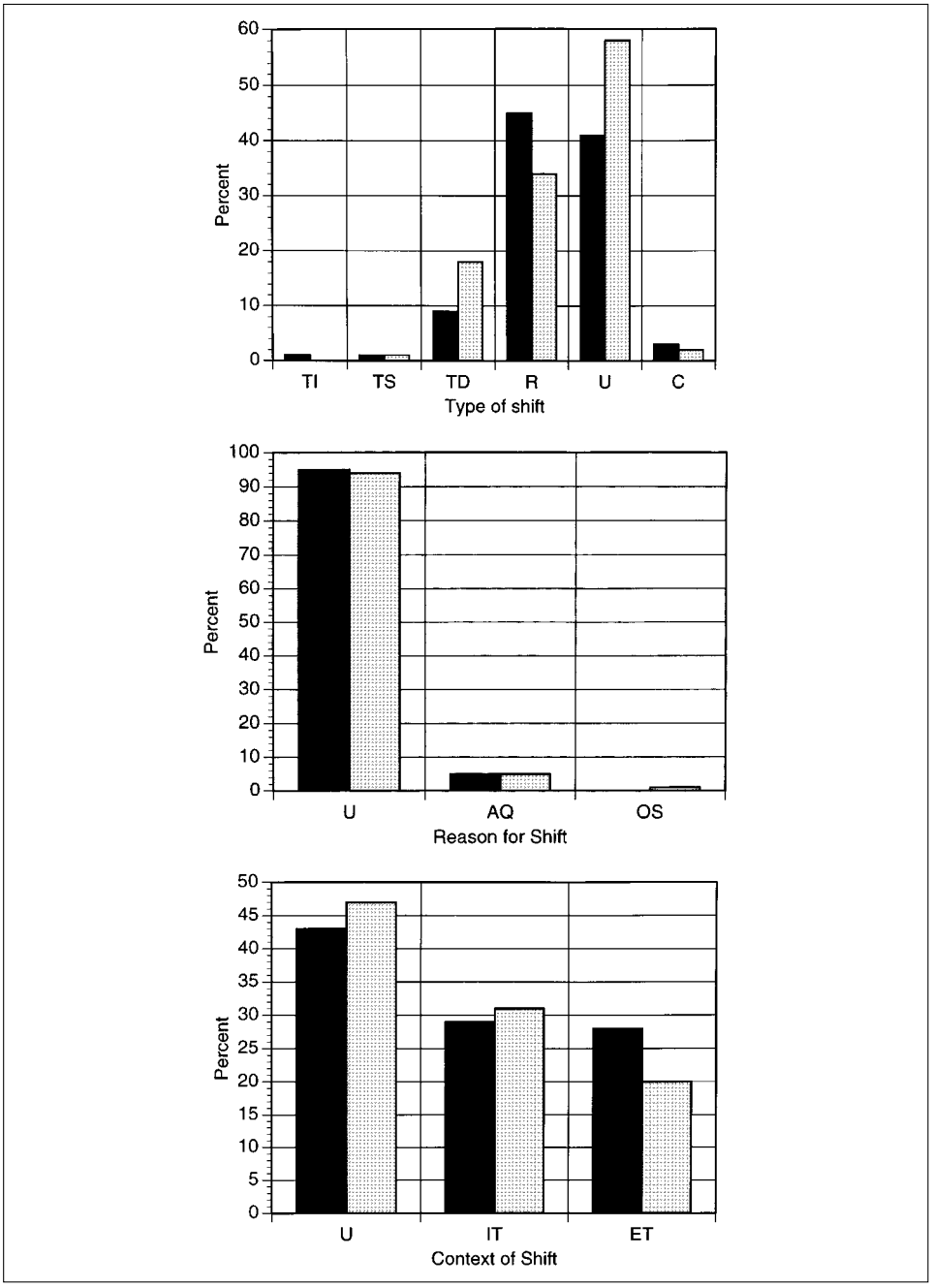

Fig. 3 – *Distribution of topic shift characteristics. Black columns represent the first interview, and gray columns represent the second. a) Type of shift. TI - topic initiation, TS - topic shading, TD - topic drift, R - renewal of topic, U - unexpected, C - conflation. See Table II for operational definitions. b) Reasons for shift. U - unknown, AQ - answering previous question, OS - reacting to an outside stimulus. See Table II for operational definitions. c) Context of shift. U - unknown, IT - topic previously initiated by the patient, ET - topic previously initiated by examiner. See Table II for operational definitions.*

DISCUSSION

LB presented with an acute amnesia and a confusional state. Her initial lethargy and dysarthric speech resolved within a few days. However, her dense amnesia and confusional state persisted for at least a year after her stroke. She had minimal insight into her deficits. Six months after her stroke when asked if she had any problems with her thinking, she laughed and replied that she was "a bit addled", but she had been that way all her life. Her MRI scans showed bilateral medial thalamic lesions in the vascular territories of the paramedian thalamic artery which often bifurcates off a single pedicle from the posterior cerebral artery (Castaigne et al., 1981; Von Cramon et al., 1985). LB's lesions involved the mammillothalamic and presumably the amygdalofugal tracts, the intralaminar nuclei, and also parts of the dorsomedial nuclei and ventral lateral nuclei bilaterally. The ventral anterior nuclei may have also been partially involved. LB's initial rCBF brain SPECT scan showed thalamic hyperperfusion compatible with luxury perfusion following the acute injury (Lassen, 1994). Her subsequent SPECT scan showed thalamic and basal ganglia hypeperfusion. The basal ganglia hypoperfusion makes sense with her intralaminar nuclei lesion, since intralaminar nuclei have major striatal efferent connections (Groenewegen and Berendse, 1994; Jones, 1985). LB also had general diminished cerebral blood flow and further reductions in the right frontal and left temporal and parietal lobes.

Patients with bilateral paramedian median thalamic lesions, like LB, typically have amnesia, decreased levels of arousal and often vertical eye movement abnormalities (Bogousslavsky et al., 1988b; Guberman and Stuss, 1983; Tatemichi, Steinke, Duncan et al., 1992). Their amnesia is thought to be a 'disconnection' amnesia resulting from deficits in accessing memories that are stored in a widely distributed fashion (Warrington and Weiskrantz, 1982). The notion of a 'disconnection' amnesia is further supported by neuro-anatomic observations. These patients invariably have involvement of the mammillothalamic tract and the ventral amygdalofugal pathways (Crosson, 1992; Gentilini, De Renzi and Crisi, 1987; Graff-Radford et al., 1990; Von Cramon et al., 1985). Disruption of these white matter tracts presumably disconnects the frontal lobes from the ventral-posterior cortical regions. The resulting amnesia is then caused primarily by a search and retrieval deficit. LB's discourse suggested a retrieval problem (discussed below) in keeping with the 'disconnection' hypothesis of diencephalic amnesia. Thus, LB's amnesia and lesion location are characteristic of the syndrome of diencephalic amnesia after paramedian thalamic infarcts.

To describe LB as amnestic, while accurate, does not adequately convey the striking phenomenology of her condition. Her confabulations and often bizarre responses, mixed with occasional appropriate responses were the dominant clinical feature. She frequently perseverated, or had intrusions in her speech. Her output when produced without the imposition of an external structure was disconnected and had unpredictable shifts to and from fragments of different themes.

LB performed quite normally on most lexical and morpho-syntactic parameters of spontaneous speech, despite having a severely disordered speech output. The proportion of closed class words, noun/pronoun ratio, presence of

obligatory determiners, noun/verb ratio and frequency of verb inflections in her speech was normal. She also produced close to normal proportions of words in sentences and well formed sentences. Her disordered output could not be accounted for by disruptions of local feature of language production and was distinct from aphasic disturbances sometimes observed in patients with left tuberothalamic infarcts (Bogousslavsky et al., 1986b). She also seemed to be able to access overlearned scripts such as reciting the days of the week, the alphabet or short rhyming jingles. However, her disorder was most evident in unstructured discourse.

We think LB's deficit is better characterized as a dynamic disorder of thought rather than a language disorder per se. Shanon (1989) argued that thought sequences may be expressed verbally in ways that offer insight into the workings of the mind. Other investigators speculate that thalamo-cortical circuit dysfunction may underlie thought disorders (Crosson and Hughes, 1987; Steriade, Jones and Llinas, 1990). We are not claiming that LB's thought disorder is isomorphic with schizophrenic symptomatology. Rather we wish to underscore its ideational nature, in which the coherence of underlying streams of thought is disrupted. Luria (1977) similarly drew attention to an unstable and fluctuating disorder of speech associated with thalamic lesions, which he felt was not aphasic, but rather "a disturbance of a deeper nature".

Confabulations in patients with paramedian thalamic infarcts (Bogousslavsky, Ferrazzini, Regli et al., 1988a) have been described as resembling 'psychotic speech' (Meissner, Sapir, Kokmen et al., 1987). See Appendix 5 for an example of LB's speech that seemed 'psychotic'. During conversation, LB shifted thematic topics frequently. The timing of these shifts was unpredictable and the shifts were usually untethered contextually. At times she would maintain thematic coherence across several sentences; at other times thematic shifts were evident within utterances. Elements of more than one theme were sometimes fused in single utterances. Her inability to maintain or shift thematic topics in a contextually appropriate manner seems to be a deficit in the regulation of James 'transitive' aspects of consciousness. It is reminiscent of Lhermitte's observation that these patients have trouble with the ordering of ideas, and passages from one story seemed to slide into others in their narratives (Lhermitte, 1984). The themes into which LB unexpectedly shifted were often intrusions from previous topics. These intrusions were evenly divided into topics that she had introduced and those introduced by the examiner. Traces of these themes from the conversation had been encoded and stored sufficiently to resurface (albeit unpredictably) as thematic intrusions. Yet, at the end of both conversations LB was unable to volitionally recall any topic discussed. The fact that new information was encoded sufficiently to later intrude upon her discourse but could not be retrieved volitionally is compatible with a 'disconnection' amnestic state.

LB's inability to maintain and shift themes appropriately, contrasts with patient PS, reported by Hodges and McCarthy (1993), who also had bilateral paramedian thalamic infarcts. PS had significant deficits in learning new material and had an extensive retrograde autobiographical amnesia. His dominant clinical feature was the persistent claim that he was home from the navy on leave from active service during World War II. His memory for early parts of his life was

fragmentary and could not be integrated into a coherent life story. His retrograde amnesia was interpreted as a deficit of a 'thematic retrieval framework'. Hodges and McCarthy (1993) suggest that episodic memory is retrieved in hierarchically structured 'organization packets'. At the highest level, thematic frameworks are retrieved which provide pointers that guide the selection of lower level detailed information. The authors suggest that PS was pathologically stuck in the 'navy' theme that became a default frame for the reconstruction of his past and incorporation of new events from his present.

LB's deficit complements PS's thematic retrieval deficit. Both LB and PS were unable to retrieve contextually appropriate themes. PS is tied to a specific thematic framework in an obligatory manner. By contrast, LB is unable to remain tethered to a particular framework for any substantial duration. Her deficit results in unpredictable and rapid shifts in the thematic frameworks guiding her responses. Many of LB's 'confabulatory' responses might not have been confabulatory in the specific sense of fictional re-creations (Berlyn, 1972). Fantastic confabulations, or fictional re-creations, are associated with frontal dysfunction (Kapur and Coughlan, 1980; Stuss, Alexander, Lieberman et al., 1978). However, recent reports suggesting that the neural basis for these kinds of confabulation involves orbito-frontal and medial frontal rather than dorsolateral prefrontal dysfunction (Benson, Djenderedjian, Miller et al., 1996) support our notion that LB's 'confabulations' may have been of a different nature. LB's peculiar comments may have represented fragmented themes surfacing unpredictably. From the vantage point of an external observer, without knowledge of the specific thematic framework guiding her verbal output, she is confused, confabulatory and at times frankly bizarre. A striking example of this phenomenon was her drilling her adult daughter on math problems, something she had regularly done, but over thirty years before her hospital admission. In keeping with our observations, Shnider and coworkers have recently reported data to suggest that spontaneous confabulations are associated with an inability to recognize the temporal order of stored information and reflect a confusion of memory traces from diverse events rather than a loss of the trace themselves (Shnider, Daniken and Gutbrod, 1996).

Could LB's behavior be ascribed to a working memory or executive attention deficit? She does not have a phonological rehearsal deficit as shown by her ability to repeat digits and phrases. Her deficit could be due to a specific form of an executive attentional disturbance along the lines suggested by Norman and Shallice (1986). According to their model, a large number of thoughts compete for cognitive resources at any given instance. A 'supervisory attentional system' modulates which thoughts will be activated and which inhibited. Dysregulation of this system could presumably result in the kind of speech observed in LB. It remains unclear whether such an attentional disturbance could be modality specific. Luria thought his similar case with a 'quasi-aphasic' disorder had a material specific disorder (Luria, 1977). He suggested that a partial disturbance of vigilance focused specifically on speech processes was responsible for his patient's deficits.

The neural mechanisms underlying LB's deficit in maintaining and appropriately shifting themes during spontaneous discourse are not clear. Her

amnesia probably resulted from the disruption of the mammillothalamic tracts (Graff-Radford et al., l990; Von Cramon et al., 1985). However, confusion is not a necessary concomitant of diencephalic amnesia. LB's confusional state probably resulted from additional damage to the dorsomedial and intralaminar nuclei. The dorsomedial nuclei have reciprocal connections with the dorsolateral prefrontal cortices (Jones, 1985). The intralaminar nuclei have widespread, topographically organized, cortical reciprocal connections and major striatal connections (Groenewegen and Berendse, 1994; Jones, 1985). Damage restricted to the intralaminar nuclei unilaterally may produce clinically significant distractibility without amnesia (Mennemeier, Fennell, Valenstein et al., 1992). LB's dramatic presentation may have been the consequence of a dense amnesia combined with the widespread effects of intralaminar and midline thalamic damage.

We do not know if LB's thalamic lesions produced non-specific cortical abnormalities or if they contribute to her deficits more directly. Thalamic lesions may produce cortical diaschisis (Baron, Levasseur, Mazoyer et al., 1992; Levasseur, Baron, Sette et al., 1992; Pepin and Aurey-Pepin, 1993). Midline and intralaminar thalamic nuclei are thought to be critical for cortical recruitment responses (Jones, 1985), perhaps through the regulation of thalamocortical and striatocortical circuits (Groenewegen and Berendse, 1994). LB's rCBF brain SPECT scans showed areas of abnormal cortical perfusion. As such, her deficits may have been a consequence of abnormally functioning deafferented frontal and left temporal-parietal cortices. The frontal lobes are conceptualized as critical for the maintenance of 'script like' events, that is, themes encompassing some temporal duration or regulating the temporal organization of behavior (Fuster, 1989; Grafman, 1994; Shallice and Burgess, 1991). Left temporal-posterior parietal regions encompass neural networks thought to be critical for the access to and retrieval of conceptual knowledge (Damasio, Grabowski, Tranel et al., 1996). It is possible that LB's dramatically abnormal discourse results from the abnormality of frontal regulation and maintenance of scripts combined with problems of access to her conceptual knowledge. As such, her deficits caused by cortical deafferentation may be more severe and dramatic than deficits associated with stable lesions restricted to the frontal or left temporal lobes.

An alternate hypothesis posits a more direct thalamic neural mechanism for LB's deficit. The very notion of a thematic framework guiding memory searches and discourse implies highly orchestrated neural activity. The search and selection of detailed factual information bound to local elements of language, such as phonological and lexical selection and syntactic structuring, coordinated with pragmatic aspects of communication must somehow cohere on-line in discourse. Facial expressions, gestures, body posture somehow yoked to these themes manifest a coordinated state of consciousness. The problem of orchestrating neural activity over widely distributed brain regions coordinated in real time has been termed the binding or the global mapping problem (Llinas and Pare, 1991). Oscillatory neuronal activity of about 40 Hz are thought to be a candidate mechanism binding neural activity widely distributed in space within temporal epochs (Crick and Koch, 1990; Steriade, McCormick and Sejnowski, 1993b). Magnetoencephalographic recordings in awake humans reveal the

presence of continuous 40 Hz oscillations over the entire cortical mantle (Llinas and Ribary, 1993). Thalamo-cortical circuits may regulate such oscillations over distributed cortical regions via diffuse cortical projections with reentrant back projections (Steriade et al., 1990). Intralaminar nuclei neurons generate 40 Hz oscillations (Steriade, Dossi and Contreras, 1993a). These specific oscillations are postulated to play a critical role in the binding necessary for the unity of cognitive experience (Crick and Koch, 1990; Llinas and Ribary, 1993; Newman, 1995). Perhaps damage to the intralaminar nuclei impairs the binding of neural activity over widely disparate neural structure. In its mild form, this impairment might result in increased distractibility (Mennemeier et al., 1992). In its severe form, perhaps combined with amnesia, this impairment might result in an obligatory entrapment within a specific thematic framework (Hodges and McCarthy, 1993), or in the untethering of thematic frames from volitional control or contextual constraints.

In conclusion, we think that LB's confusion is not explained by disruptions of local features of language. We echo Luria's suggestion (from 20 years ago) that this dramatically disordered speech associated with thalamic lesions is the surface manifestation of a deeper dynamic disorder. This disorder is characterized by thematic instability and fragmentation. Damage to midline thalamic nuclei may have widespread cortical effects resulting in the inability to maintain and appropriately shift themes within discourse. The instability of thematic frameworks results in frequent and unpredictable thematic shifts as evidenced by abnormalities of the "rhythm of language".

Acknowledgements. We thank Lisa Santer for a critical reading of the manuscript and Angela Armstrong for discourse transcriptions.

References

BARON, J., LEVASSEUR, M., MAZOYER, B., LEGAULT-DEMARE, F., MAUGUIERE, F., PAPPATA, S., JEDYNAK, P., DEROME, P., CAMBIER, J., TRAN-DINH, S., and CAMBON, H. Thalamocortical diascisis: positron emission tomography in humans. *Journal of Neurology, Neurosurgery and Psychiatry, 55:* 935-942, 1992.

BENSON, D.F., DJENDEREDJIAN, A., MILLER, B.L., PACHANA, N.A., CHANG, L., ITTI, L., ENG, G.E., and MENA, I. Neural basis of confabulation. *Neurology, 46:* 1239-1243, 1996.

BERLYN, N. Confabulation. *British Journal of Psychiatry, 120:* 31-39, 1972.

BERNDT, R.S. Sentence processing in aphasia. In M. Sarno (Eds.), *Acquired Aphasia*. New York: Academic Press, 1991, pp. 223-269.

BLOOM, R.L. Hemispheric responsibility and discourse production: contrasting patients with unilateral left and right brain damage. In R.L. Bloom, L.K. Obler, S.D. Santi and J.S. Ehrlich (Eds.), *Discourse Analysis and Applications*. Hillsdale: Lawrence Erlbaum Associates, 1994, pp. 81-94.

BOGOUSSLAVSKY, J., FERRAZZINI, M., REGLI, F., ASSAL, G., TANABE, H., and DELALOYE-BISCHOF, A. Manic delirium and frontal-like syndrome with paramedian infarction of the right thalamus. *Journal of Neurology, Neurosurgery, and Psychiatry, 51:* 116-119, 1988a.

BOGOUSSLAVSKY, J., REGLI, F., and USKE, A. Thalamic infarcts: Clinical syndromes, etiology, and prognosis. *Neurology, 38:* 837-848, 1988b.

CASTAIGNE, P., LHERMITTE, F., BUGE, A., ESCOUROLLE, R., HAUW, J.J., and LYON-CAEN, O. Paramedian thalamic and midbrain infarcts: Clinical and neuropathological study. *Annals of Neurology, 10:* 127-148, 1981.

CHANG, L.T. A method for attenuation correction in radionuclide computed tomography. *IEEE Transactions of Nuclear Sciences, 26:* 638-643, 1978.

CHATTERJEE, A., and MENNEMEIER, M. Anosognosia for hemiplegia: patient retrospections. *Cognitive Neuropsychiatry, 1:* 221-237, 1996.

CRICK, F., and KOCH, C. Towards a neurobiological theory of consciousness. *Seminars Neuroscience, 2:* 263-275, 1990.
CROSSON, B. *Subcortical Functions in Language and Memory*. New York: The Guilford Press, 1992.
CROSSON, B., and HUGHES, C. Role of the thalamus in language: is it related to schizophrenic thought disorder? *Schizophrenia Bulletin, 13:* 605-621, 1987.
CROW, B. Topic shifts in couples' conversation. In R. Craig and K. Tracy (Eds.), *Conversational Coherence: Form, Structure and Strategy*. Beverly Hills, CA: Sage, 1983.
DAMASIO, H., GRABOWSKI, T., TRANEL, D., HICHWA, R.D., and DAMASIO, A.R. A neural basis for lexical retrieval. *Nature, 380:* 499-505, 1996.
DELIS, D., KRAMER, J., KAPLAN, E., and OBER, B. *The California Verbal Learning Test Adult Version: Manual*. New York: Psychological Corporation, 1987.
FUSTER, J. *The Prefrontal Cortex* (2nd ed.). New York: Raven Press, 1989.
GARCIA, L.J., and JOANETTE, Y. Conversational topic-shifting analysis in dementia. In R.L. Bloom, L.K. Obler, S. DeSanti and J.S. Ehrlich (Eds.), *Discourse Analysis and Applications*. Hillsdale, New Jersey: Lawrence Erlbaum Associates, 1994.
GENTILINI, M., DE RENZI, E., and CRISI, G. Bilateral paramedian thalamic artery infarcts: report of eight cases. *Journal of Neurology, Neurosurgery, and Psychiatry, 50:* 900-909, 1987.
GRAFF-RADFORD, N.R., TRANEL, D., VAN HOESEN, G.W., and BRANDT, J.P. Diencephalic amnesia. *Brain, 113:* 1-25, 1990.
GRAFMAN, J. Alternative frameworks for the conceptualization or prefrontal lobe functions. In F. Boller and J. Grafman (Eds.), *Handbook of Neuropsychology, Vol. 9*. Amsterdam: Elsevier Science Publishers, 1994, pp. 187-202.
GROENEWEGEN, H.J., and BERENDSE, H.W. The specificity of the 'nonspecific' midline and intralaminar thalamic nuclei. *Trends Neuroscience, 17:* 52-58, 1994.
GUBERMAN, A., and STUSS, D. The syndrome of bilateral paramedian thalamic infarction. *Neurology, 33:* 540-546, 1983.
HAINES, D. *Neuroanatomy. An Atlas of Structure, Section, and Systems* (4rth ed.). Baltimore: Williams and Wilkins, 1995.
HODGES, J.R., and MCCARTHY, R.A. Autobiographical amnesia resulting from bilateral paramedian thalamic infarction: A case study in cognitive neurobiology. *Brain, 116:* 921-940, 1993.
JAMES, W. *The Principles of Psychology*. New York: Dover Publications, Inc., 1890.
JONES, E. *The Thalamus*. New York: Plenum Press, 1985.
KAPUR, N., and COUGHLAN, A.K. Confabulation and frontal lobe dysfunction. *Journal of Neurology, Neurosurgery, and Psychiatry, 43:* 461-463, 1980.
LASSEN, N.A. Imaging brain infarcts by single-photon emission tomography with new tracers. *European Journal of Nuclear Medicine, 21:* 189-190, 1994.
LEVASSEUR, M., BARON, J.C., SETTE, G., LEGAULT-DEMARE, F., PAPPATA, S., MAUGUIERE, F., BENOIT, N., TRAN-DINH, S., DEGOS, J., LAPLANE, D., and MAZOYER, B. Brain energy metabolism in bilateral paramedian thalamic infarcts. *Brain, 115:* 795-807, 1992.
LHERMITTE, F. Language disorders and their relationship to thalamic lesions. *Advances in Neurology, 42:* 99-113, 1984.
LIU, H.G., HARRIS, J.M., INAMPUDI, C.S., and MOUNTZ, J.M. Optimal reconstruction filter parameters for multi-head brain SPECT: dependence on count activity. *Journal of Nuclear Medicine Technology, 23:* 251-257, 1995.
LLINAS, R., and PARE, D. Of dreaming and wakefulness. *Neuroscience, 44:* 521-535, 1991.
LLINAS, R., and RIBARY, U. Coherent 40-Hz oscillation characterized dream states in humans. *Proceedings of National Academy of Sciences, 90:* 2078-2081, 1993.
LURIA, A.R. On quasi aphasic speech disturbances in lesions of deep structures of the brain. *Brain and Language, 4:* 432-459, 1977.
MATTIS, S. Mental status examination for organic mental syndrome in the elderly patient. In L. Bellak and T.B. Karasu (Eds.), *Geriatric Psychiatry*: New York: Grune and Stratton, 1976.
MEISSNER, I., SAPIR, S., KOKMEN, E., and STEIN, S.D. The paramedian diencephalic syndrome: A dynamic phenomenon. *Stroke, 18:* 380-385, 1987.
MENNEMEIER, M., FENNELL, E., VALENSTEIN, E., and HEILMAN, K. Contributions of the left intralaminar and medial thalamic nuclei to memory. *Archives of Neurology, 49:* 1050-1058, 1992.
MILNER, A.D., and RUGG, M.D. *The Neuropsychology of Consciousness*. New York: Academic Press, 1992.
MOUNTZ, J.M. Quantification of the SPECT brain scan. In L.M. Freedman (Eds.), *Nuclear Medicine Annual*. New York: Raven Press, 1991, pp. 67-98.
MOUNTZ, J.M. Brain SPECT: 1994 Update. In L.M. Freeman (Ed.), *Nuclear Medicine Annual*. New York: Raven Press, 1994, pp. 1-54.
MOUNTZ, J.M., WILSON, M.W., WOLFF, C.G., DEUTSCH, G., and HARRIS, J.M. Validation of a reference method for correlation of anatomic and functional brain images. *Computerized Medical Imaging Graphics, 18:* 163-174, 1994a.

MOUNTZ, J.M., ZHANG, B., LIU, H.-G., and INAMPUDI, C. A reference method for correlation of anatomic and functional brain images: Validation and clinical application. *Seminars in Nuclear Medicine, 24:* 256-271, 1994b.

NEWMAN, J. Thalamic contributions to attention and consciousness. *Consciousness and Cognition, 4:* 172-193, 1995.

NORMAN, D., and SHALLICE, T. Attention to action: willed and automatic control of behavior. In R.J. Davidson, G.E. Schwartz and D. Shapiro (Eds.), *Consciousness and Self-Regulation*. New York: Plenum Press, 1986.

PEPIN, E., and AUREY-PEPIN, L. Selective dorsolateral frontal lobe dysfunction associated with diencephalic amnesia. *Neurology, 43:* 733-741, 1993.

RUSSELL, E.W. A multiple scoring method for the assessment of complex memory functions. *Journal of Consulting Clinical Psychology, 43:* 800-809, 1975.

SAFFRAN, E.M., BERNDT, R.S., and SCHWARTZ, M.F. The quantitative analysis of agrammatic production: procedure and data. *Brain and Language, 37:* 440-479, 1989.

SHALLICE, T., and BURGESS, P. Higher-order cognitive impairments and frontal lobe lesions. In H. Levin, H. Eisenberg and A. Benton (Eds.), *Frontal Lobe Function and Dysfunction*. New York: Oxford University Press, 1991, pp. 125-138.

SHANON, B. Thought sequences. *European Journal of Cognitive Psychology, 1:* 129-159, 1989.

SHNIDER, A., DANIKEN, C.V., and GUTBROD, J. The mechanisms of spontaneous and provoked confabulations. *Brain, 119:* 1365-1375, 1996.

STERIADE, M., DOSSE, R.C., and CONTRERAS, D. Electrophysiologic properties of intralaminar thalamocortical cells discharging rhythmic (40Hz) spike-bursts at 1000 Hz during waking and rapid eye movement sleep. *Neuroscience, 56:* 1-9, 1993a.

STERIADE, M., JONES, E., and LLINAS, R. *Thalamic Oscillations and Signalling*. New York: John Wiley and Sons, 1990.

STERIADE, M., MCCORMICK, D., and SEJNOWSKI, T.J. Thalamocortical oscillations in the sleeping and aroused brain. *Science, 262:* 679-685, 1993b.

STUSS, D.T., ALEXANDER, M.P., LIEBERMAN, A., and LEVINE, H. An extraordinary form of confabulation. *Neurology, 28:* 1166-1172, 1978.

TATEMICHI, T.K., STEINKE, W., DUNCAN, C., BELLO, J.A., ODEL, J.G., BEHRENS, M.M., HILAL, S.K., and MOHR, J.P. Paramedian thalamopeduncular infarction: Clinical syndromes and magnetic resonance imaging. *Annals of Neurology, 32:* 162-171, 1992.

VON CRAMON, D.Y., HEBEL, N., and SCHURI, U. A contribution to the anatomical basis of thalamic amnesia. *Brain, 108:* 993-1008, 1985.

WARRINGTON, E.K., and WEISKRANTZ, L. Amnesia: A disconnection syndrome. *Neuropsychologia, 20:* 233-249, 1982.

Anjan Chatterjee, UAB Neurology, 1720 7th Ave South, SC454, Birmingham, AL 35294-0017, USA. Tel.: (205) 934-3847; Fax: (205) 975-7365; email: a_chatterjee@email.neuro.uab.edu.

(Received 18 September 1996; accepted 5 March 1997)

APPENDIX

LB's most striking clinical feature was her apparent confusion and bizarre responses. To convey the quality of her phenomenology we have included examples of extended excerpts. Examples of coding are included at the beginning of the shift, following the abbreviations listed in Table II.

(1) LB has been told that she was being videotaped for research purposes. We think that this information served as the seed for 'research' appearing in her subsequent responses. We do not know the context for the girl scouts' theme. However, towards the end of this excerpt both research and girls scouts were merged into a single utterance.

E: *Do you know how long you have been here?*
LB: *I've been here about two days,* /1.R, 2.U, 3.ET/ *doing a research project for another group of girls.*
E: *What kind of research project are you doing?*
LB: *This girls scout project, with... I was a background project.*
E: *Do you think this a hospital?*
LB: *No. No, this is...* /1.U, 2.U, 3.U/ *You don't do any more ground work* /1.U, 2.U, 3.U/ *I suppose we do in learning about people.*
E: *Have you been ill at all?*
LB: *No. The only illness I have had was diagnosed by your company as a result of* /1.U, 2.U, 3.U/ *setting on duty for around here, for an over extended length of time, when I was examined.* /1.U, 2.U, 3.U/ *I think yesterday you had... as a result of...*
E: *How's your thinking?*
LB: *It's been a lot better.* /1.U, 2.U, 3.U/ *I influenced from... and* /1.R, 2.U, 3.ET/ *our research has been outdoor work of bees and butter... interior.*
E: *Where are you from?*
LB: *I am from Texas.*
E: *Did you grow up there?*
LB: *Yes.*
E: *How did you come to Alabama from Texas?*
LB: *As a research scientist.*
E: *What were you doing research in?*
LB: /1.C, 2.U, 3.ET/ *My sister was in research with the girl scouts and I just kind of followed in her tracks.*

(2) Sometimes LB simply confabulated responses. The source of these confabulations could not be determined from the context of the conversation or any information available about LB.

E: *Do you know where you are? Where is this place?*
LB: *South America.*
E: *South America?*
LB: /1.TD, 2.U, 3.ET/ *S-O-U-T-H, That's the way to spell South America.*
E: *Why are you in South America?*
LB: *My husband. He's in South America. He's supposed to be there tonight.*

(3) At times, LB became 'stuck in set' and would thematically perseverate as the following excerpt demonstrates. We elected to count such perseverations as topic shifts since LB shifts to her stuck-in-set topic rather than the new topic introduced be the examiner.

E: *Can you name for me as many of the states of the US that you can think of?*
LB: *Alabama, Mississippi, Georgia, Kentucky...*
E: *Keep going.*
LB: *Kentucky, Wyoming. (prolonged pause, stays quiet).*
E: *Ma'am?*
LB: *Yes?*
E: *What are you thinking of?*

LB: Alabama, Mississippi, Georgia, Chicago. Chicago is... Mississippi and Georgia are states. Georgia is. Mississippi, Alabama, they're towns (another long pause).
E: What is the matter?
LB: /1.R, 2.U, 3.ET/ *Alabama, Georgia. (long pause).*
E: Ma'am, can you describe your house for me?
LB: Which house?
E: The house you live in.
LB: /1.R, 2.U, 3.ET/ *Alabama, Georgia, Maine, Florida. (long pause).*
E: I'd like you to try something else. Can you name as many animals as you can think of?
LB: /1.R, 2.U, 3.ET/ *Alabama, Georgia, Mississippi.*
E: Ma'am, can you name as many animals as you can think of?
LB: Fox, rabbit.

(4) Attempts to get LB to narrate fairy tails were unsuccessful. She says enough (Cinderella-attic, sisters; Red Riding Hood-woods) to show that she made the correct association. However, a 'home' theme that was present at different times in her discourse intrudes. The peculiar numbering from 11 to 13 associated with Christmas persists across attempts at both fairy tales.

E: Do you remember the Cinderella story?
LB: Home up in the attic.
E: That's right. She had a home up in the attic.
LB: She didn't get along with her sisters. /1.R, 2.US 3.ET/ *Be home before Christmas. 11 things like Christmas. Find 11 things like Christmas.*
E: *What about 11 things like Christmas?*
LB: /1.R, 2.U, 3.ET/ *She was at home.* /1.C, 2.U, 3.ET/ *Her home was 12 things for Christmas.*
E: What else happened in the Cinderella story? (non answer)
Did she go to a dance?
LB: She went to... /1.U, 2.U, 3.U/ *store.*
E: Did she go to a dance? A ball?
LB: A ball is a big dance. /1.R, 2.U 3.IT/ *Home.*
E: Do you remember Red Riding Hood?
LB: She was out in the woods, but, she... /1.R, 2.U, 3.IT/ *I don't know anything about her home, her parents. Oh, yes.* /1.R, 2.U, 3.IT/ *13 things. There are 13 people, 13 things to remember. 13 things at home. There were 13 people who lived where she lived.*
E: Where who lived?
LB: /1.C, 2.U, 3.ET/ *13 different Christmases.*

(5) LB's discourse, at time, was sufficiently bizarre as to fit previous descriptions of similar patients having 'psychotic speech'.

E: Can you tell me the Cinderella story?
LB: Not tonight.
E: How about another fairy tale? Can you tell me Sleeping Beauty?
LB: Don't they all have the same story? Are they... /1.R, 2.U, 3.ET/ *fairy godmother who would...*
E: Who would do what?
LB: Exploit the boy with tales or stories that he would not believe were they told in any other context. /1.U, 2.U, 3.U/ *I can't see him.* /1.U, 2.U, 3.U/ *He would have to experience... There aren't any more mosquitoes that serve in the civilian guard. Serve.* /1.R, 2.U, 3.ET/ *For him to believe it, and so with all stories.*

(6) Sometimes a question or word seemed to trigger a train of thought in LB, only loosely related to the initial suggestion. In the following excerpt, LB remains within the general bounds of her theme for over three minutes, the longest period during the time she was

recorded. She was a deeply religious woman. In addition to the poetry of her output, what is not easily conveyed in the following excerpt is the cadence, prosody and modulation of volume reminiscent of a minister giving a sermon.

E: *Do you know what month this is ma'am?*
LB: *We're in February.*
E: *Actually it is December. Can you think of what holiday happens in December?*
LB: *Oh! December, you know.* /1.TD, 2.U, 3.ET/ *December is our greatest month, is our greatest gift. Our greatest prophet is supposed to have come in December and given us our greatest gift, a child who was like nothing else in this world.*
E: *How do we celebrate that day?*
LB: *We call it Christmas and we give gifts. We know what is in people's heart is true or is false. Just December means everything is perfect. December means everybody is good. December means I love you and you love me. December means all is good, all is perfect, all is God. December means what that sound means, all is well, all will move in the right direction, all is wise, all is wisdom, all is light, all is truth, all is goodness. December means there is no separation one from another. December means you come through me and learn all you need to learn and I go through you and learn all I need to know. December means God is open for all his people to use. God is there for all his people to use. God is one for all his people to use. God is perfect. He's whatever number we need. God is perfect. He's perfect. He's whatever number we need. God is perfect.*