Transcortical sensory aphasia: revisited and revised

Dana Boatman,1,2,5 Barry Gordon,1,5 John Hart,1,5 Ola Selnes,1 Diana Miglioretti3 and Frederick Lenz4

Departments of 1Neurology, 2Otolaryngology, 3Biostatistics and 4Neurosurgery and 5The Krieger Mind/Brain Institute, Johns Hopkins Medical Institutions, Baltimore, Maryland, USA

Summary
Transcortical sensory aphasia (TSA) is characterized by impaired auditory comprehension with intact repetition and fluent speech. We induced TSA transiently by electrical interference during routine cortical function mapping in six adult seizure patients. For each patient, TSA was associated with multiple posterior cortical sites, including the posterior superior and middle temporal gyri, in classical Wernicke’s area. A number of TSA sites were immediately adjacent to sites where Wernicke’s aphasia was elicited in the same patients. Phonological decoding of speech sounds was assessed by auditory syllable discrimination and found to be intact at all sites where TSA was induced. At a subset of electrode sites where the pattern of language deficits otherwise resembled TSA, naming and word reading remained intact. Language lateralization testing by intracarotid amobarbital injection showed no evidence of independent right hemisphere language. These results suggest that TSA may result from a one-way disruption between left hemisphere phonology and lexical–semantic processing.

Keywords: transcortical sensory aphasia; Wernicke’s aphasia; cortical auditory disorders

Abbreviations: CV = consonant–vowel; CVC = consonant–vowel–consonant; IAI = intracarotid sodium amobarbital injection; TSA = transcortical sensory aphasia

Introduction
Transcortical sensory aphasia (TSA) is characterized by impaired auditory comprehension, with intact repetition and fluent speech (Lichtheim, 1885; Goldstein, 1948). The sparing of repetition distinguishes TSA from other receptive aphasias and agnosias, including Wernicke’s aphasia and pure word deafness. Both repetition and auditory comprehension depend on intact phonological processing for early decoding of the speech signal (Liberman et al., 1967; Luria, 1976; McCarthy and Warrington, 1984). Because repetition is spared in TSA, phonological processing is assumed to be, at least partially, intact. Phonological processing is associated with Wernicke’s area, defined traditionally as the posterior two-thirds of the left superior and middle temporal gyri (Wernicke, 1874; Luria, 1976; Damasio, 1981). Lesions involving this area are often associated with impaired repetition and auditory comprehension, as seen in Wernicke’s aphasia and pure word deafness (Wernicke, 1874; Luria, 1976; Selnes et al., 1985).

In addition to phonological processing, comprehension of spoken speech requires access to lexical–semantic information, including word meanings (Luria, 1976; Pisoni and Luce, 1987). (The combined reference to lexical and semantic processing was chosen by convention and because our study did not examine directly their potential dissociability.) The ability of normal listeners to repeat non-words suggests that lexical–semantic mediation is not required for repetition (McCarthy and Warrington, 1984; Coslett et al., 1987). Impaired lexical–semantic processing has been associated with widely disparate cortical regions, including the temporal lobe, the temporo-occipital junction, the parietal lobe, the fusiform gyrus and the frontal lobe (Damasio, 1981; Heilman et al., 1976, 1981; Kertesz et al., 1982). In addition to phonological processing, comprehension of spoken speech requires access to lexical–semantic information, including word meanings (Luria, 1976; Pisoni and Luce, 1987). (The combined reference to lexical and semantic processing was chosen by convention and because our study did not examine directly their potential dissociability.) The ability of normal listeners to repeat non-words suggests that lexical–semantic mediation is not required for repetition (McCarthy and Warrington, 1984; Coslett et al., 1987). Impaired lexical–semantic processing has been associated with widely disparate cortical regions, including the temporal lobe, the temporo-occipital junction, the parietal lobe, the fusiform gyrus and the frontal lobe (Damasio, 1981; Heilman et al., 1981; Kertesz et al., 1982; Alexander et al., 1989; Hart and Gordon, 1990; Hart et al., 1998; Otsuki et al., 1998).

A number of models of TSA have been proposed. The most widely accepted is the disconnection model, which stipulates a bidirectional disconnection between phonology and lexical–semantic processing (Lichtheim, 1885; Goldstein, 1948; Geschwind, 1965). Sparing of repetition, with impaired comprehension, is attributed to the isolation of Wernicke’s area from lexical–semantics, presumed to be located more posteriorly (Geschwind et al., 1968; Heilman et al., 1981; Kertesz et al., 1982).

The exceptions to the disconnection model, including patients whose lesions extend into Wernicke’s area or who have spared naming abilities, have engendered alternative accounts (Heilman et al., 1976, 1981; Kertesz et al., 1982; Berthier et al., 1991). One explanation is that the lexical–semantic system itself is impaired (Luria, 1976; Alexander...
Transcortical sensory aphasia

et al., 1989). Others have argued that TSA reflects the contribution of right hemisphere language capabilities (Heilman et al., 1981; Berthier et al., 1991). Alternatively it has been suggested that TSA reflects partial damage to Wernicke’s area, leaving sufficient phonological capabilities for repetition, but not comprehension (Goldstein, 1948).

Yet another explanation is that impaired auditory–verbal memory is responsible for the pattern of language deficits associated with TSA, in that repetition appears to be intact because it is typically tested only with single words, not phrases (Shallice and Warrington, 1977; Friedrich et al., 1984). Finally, the dissociability of input and output connections between other language functions raises the possibility that TSA may result from a one-way disruption between phonology and lexical–semantic processing (Heilman et al., 1976).

TSA has been studied primarily in stroke patients, although it has been difficult to conduct systematic studies. One reason is that TSA occurs relatively infrequently in this population. Moreover, lesions associated with aphasia tend to be relatively large, potentially involving multiple functionally distinct areas. Furthermore, lesion localization studies of patients with TSA have typically relied on group comparisons, which are subject to individual differences. Finally, stroke patients are usually studied during recovery, when cortical reorganization and atypical patterns of language processing may be present.

We avoided a number of these potential limitations by studying a series of adult seizure patients in whom TSA was induced transiently by electrical interference during routine cortical function mapping. Direct cortical electrical interference, also known as cortical stimulation, is a well-established technique for mapping language and motor functions in patients who are surgical candidates for treatment of intractable seizures (Penfield and Roberts, 1959; Ojemann, 1983; Lesser et al., 1987). The effects of electrical interference are temporary (~5 s), discretely localized (~1 cm²) and highly reproducible (Lesser et al., 1987; Gordon et al., 1990, 1997; Nathan et al., 1993; Boatman et al., 1995). We were able, therefore, to study patients acutely and by within-subject comparisons. Moreover, all of our patients underwent language lateralization testing by intracarotid sodium amobarbital injection (IAI), also known as the Wada test (Wada and Rasmussen, 1960).

Methods

Subjects

Six right-handed adult seizure patients, three men and three women (mean age 34 years), were identified from a larger population of 20 consecutive patients undergoing cortical function mapping, by the following criteria: (i) seizure foci outside of traditional perisylvian language areas, as determined by direct recording of EEG activity from the subdural electrode arrays (Crone et al., 1998); (ii) no evidence of cortical abnormalities on MRI; (iii) full-scale IQ scores $\geq 80$ on the Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981); (iv) normal pure tone hearing thresholds (e.g. $\leq 25$ dB) at 250–8000 Hz, with no history of hearing, speech or oral-motor deficits; and (v) electrode coverage of posterior perisylvian cortex. Additional patient characteristics are summarized in Table 1. All six patients had complex partial seizures; patients 3 and 6 had secondary generalized seizures. All patients provided fully informed consent in compliance with our institution’s clinical research protocols.

Surgical procedures

At surgery, a $6 \times 8$ electrode array and one or more $2 \times 8$ electrode strips were implanted over the left lateral cortical surface. Electrode coverage included the superior, middle and inferior temporal gyri and, in some patients, the pre- and post-central gyri, the frontal lobe, the inferior parietal lobe and anterior portions of the occipital lobe. Differences in extent of electrode coverage were determined by each patient’s clinical circumstances. Electrodes were composed of platinum iridium discs, 2.3 mm in exposed diameter, spaced 1 cm apart (centre-to-centre) in medical-grade Silastic.

Stimuli and tasks

Seven language tasks were administered during electrical interference testing to assess repetition, auditory comprehension, naming, word and paragraph reading, spontaneous speech, and syllable discrimination. Four of the tasks (auditory comprehension, naming, word reading, syllable discrimination) were also administered during language lateralization testing by IAI. To further assess lateralization of semantic functions, patients were asked to make object categorization judgements (object/non-object, semantic relatedness) by picture pointing. A pool of 80 stimulus words was developed for testing repetition, picture naming, word reading and syllable discrimination. Words were chosen based on comparable frequency of occurrence (Francis and Kucera, 1982) and were familiar to patients, as confirmed by baseline testing.

Repetition

During electrical interference testing, 80 words were presented live-voice, one per trial, for patients to repeat. At sites where TSA was induced, phrasal repetition was tested informally by asking patients to repeat three to four phrases of five to eight words each, that included one of the single word stimuli (e.g. ‘the boy took his bat to the game’). Time permitting, repetition was also tested with 15 non-words (e.g. ket). Repetition was not evaluated during IAI testing.

Picture naming

Fifty black-and-white figure drawings corresponding to concrete nouns from the original stimulus list were presented...
Table 1 Patient demographics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Gender</th>
<th>Age (years)</th>
<th>Primary aetiology</th>
<th>Seizure onset age (years)</th>
<th>Seizure type</th>
<th>Medications</th>
<th>FSIQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Female</td>
<td>28</td>
<td>Epilepsy</td>
<td>18</td>
<td>CPS</td>
<td>Dilantin</td>
<td>107</td>
</tr>
<tr>
<td>2</td>
<td>Female</td>
<td>18</td>
<td>Epilepsy</td>
<td>5</td>
<td>CPS</td>
<td>Tegretol</td>
<td>81</td>
</tr>
<tr>
<td>3</td>
<td>Female</td>
<td>38</td>
<td>Epilepsy</td>
<td>14</td>
<td>CPS</td>
<td>Depakote</td>
<td>110</td>
</tr>
<tr>
<td>4</td>
<td>Male</td>
<td>25</td>
<td>Epilepsy</td>
<td>1.5</td>
<td>CPS</td>
<td>Tegretol</td>
<td>85</td>
</tr>
<tr>
<td>5</td>
<td>Male</td>
<td>45</td>
<td>Epilepsy</td>
<td>5</td>
<td>CPS</td>
<td>Gabapentin</td>
<td>81</td>
</tr>
<tr>
<td>6</td>
<td>Male</td>
<td>47</td>
<td>Epilepsy</td>
<td>39</td>
<td>CPS</td>
<td>Tegretol</td>
<td>115</td>
</tr>
</tbody>
</table>

FSIQ = full scale IQ score; CHI = closed head injury; CPS = complex partial seizures; SGS = secondary generalized seizures.

for patients to name. A subset of 20 of these items was taken from the Boston Naming Test (Kaplan et al., 1983). During IAI testing, naming was assessed with four of the picture naming stimuli. Patients were also asked to name four everyday objects, two of which matched the picture naming stimuli.

Word reading
Eighty orthographic representations (38 font size) of the same stimuli used for the picture naming and repetition were presented at a comfortable reading distance. The format for stimulus presentation was identical to that of the naming task. Four of these stimuli were administered during IAI testing.

Paragraph reading
During electrical interference testing, patients were given one of three paragraphs, including the Grandfather Passage (Darley et al., 1969). Paragraph reading was not assessed during IAI testing.

Spontaneous speech
A speech sample was elicited during electrical interference testing by asking patients to discuss predetermined topics of interest (e.g. their families, hobbies). Spontaneous speech was not assessed during IAI testing.

Auditory comprehension
During electrical interference testing, auditory comprehension was assessed by the Token Test (De Renzi and Vignolo, 1962). Between 10 and 15 single-step commands were administered at each site (e.g. ‘move the green square’). We used only single-step commands to ensure sufficient response time during the period of current activation. Because patients were supine for IAI testing, manipulation of tokens was not possible. Instead patients were given one-step commands involving pointing or touching (e.g. ‘point to your nose’). To assess comprehension further, patients performed semantic picture-word matching and category judgements (Hart et al., 1993).

Syllable discrimination
To assess patients’ phonological capabilities, 25 consonant–vowel–consonant (CVC) word pairs were generated, contrasted by the initial consonant (e.g. cat–bat). At sites where TSA was induced with electrical interference, we also administered 70 consonant–vowel (CV) stimulus pairs. Fifty of the CV pairs were contrasted either by consonant (e.g. /pa–ba/) or by vowel (e.g. /ba–bi/). Both consonant ($n = 30$) and vowel ($n = 20$) contrasts were included to ensure that multiple aspects of phonological processing were tested. The remaining 20 pairs were identical. Patients were asked to circle ‘same’ or ‘different’ on a response form. A subset of four CVC pairs was administered during IAI testing. In anticipation of possible speech-motor deficits after IAI, patients were trained to give verbal as well as gestural responses.

Language testing
Baseline testing
Individual baseline performance levels were obtained on all tasks before IAI or electrical interference testing for comparison with each patient’s performance during IAI and electrical interference testing. Baseline testing also confirmed patients’ familiarity with the language stimuli. Before initiating electrical interference testing, patients were re-tested after electrode implantation to ensure comparable baseline performance levels.

IAI testing
All patients underwent IAI testing before electrode implantation. For each patient the left carotid artery was injected with 125 mg of amobarbital through a femoral catheter (Hart et al., 1993). Speech and language functions were tested immediately after contralateral hemiparesis and
ipsilateral EEG slowing. Because of the short duration of amobarbital activation (~5 min), the number of stimuli administered was limited to four per task (Boatman et al., 1998).

**Electrical interference testing**

Patients were tested individually 2–3 days after surgery while they were awake, fully responsive and performing at their pre-surgery baseline levels. During testing, an electrical current was generated between adjacent electrode pairs by producing 300 μs square-pulse waves of alternating polarity, at a rate of 50 pulses/s, for 5-s intervals (Lesser et al., 1987; Gordon et al., 1997). Current thresholds were established at each pair by increasing current intensity (0.5–1.0 mA steps) until the maximal level of 15 mA was obtained. If afterdischarges occurred, testing was repeated at a later time. Single trials with afterdischarges were excluded from analysis. Language was not tested at sites where sensory or motor effects were induced.

The language battery was administered at all electrode sites covering the lateral left posterior cortex. A subset of 10–15 stimuli was randomly selected and administered for each task at each electrode pair to avoid fatiguing or over-familiarizing patients. Stimuli were presented immediately after the onset of the electrical current to ensure the full effects, with one stimulus presented in each trial. Auditory stimuli were presented live-voice by a trained female clinician who was a native speaker of American English. Task order was randomized across electrode pairs.

**Data analysis**

Deviations from patients’ baseline responses during IAI or electrical interference testing were scored as incorrect. For all tests, individual trials were worth 1 point. During electrical interference testing, responses that occurred after the 5-s period of current activation were also scored as incorrect. Trials with afterdischarges (<1% of total trials) were excluded from analysis. Deficits were identified clinically as more than one deviation from individual patient’s baseline responses.

The larger number of syllable discrimination stimuli administered permitted statistical analyses. Significant within-patient deviations from baseline were determined by using McNemar’s matched pairs test (Armitage and Berry, 1994). To compare each patient’s CV versus CVC syllable discrimination performances and their consonant versus vowel discrimination performances, we used the Fisher’s exact test of no association in a 2 × 2 table (Armitage and Berry, 1994). Unlike the standard $\chi^2$ goodness-of-fit test, Fisher’s exact test of no association between type of task and performance is appropriate for small expected cell frequencies, as is the case here where patients made few vowel discrimination errors. To examine the overall relationship between repetition and phonological processing, the Cochran Mantel–Haenszel $\chi^2$ test was calculated, stratifying by patient (Mantel and Haenszel, 1959).

Electrode locations were determined from intraoperative photographs and CT scans. To compare electrode locations across patients, electrode sites were linearly normalized within a standard brain atlas (Talairach and Tournoux, 1988), referencing the sylvian fissure and the anterior pole of the temporal lobe.

**Results**

**Baseline results**

Patients were at least 96% accurate on all language tasks before IAI or electrical interference testing.

**IAI results**

Language was suppressed entirely in all six patients after left IAI. Patients showed no evidence of independent right hemisphere syllable discrimination, auditory comprehension, naming or word reading capabilities. Moreover, they were unable to make semantic categorization judgements using verbal or non-verbal responses. These results provide no evidence of independent right hemisphere phonological or lexical–semantic capabilities in the six patients tested.

**Electrical interference results**

A total of 81 electrode pairs were tested (mean = 14, range = 6–18 pairs per patient) across all six patients (Fig. 1). At 29 of the 81 pairs tested (Fig. 2), a pattern of deficits consistent with TSA was elicited (mean = 5, range = 3–8 pairs per patient). These 29 sites were located primarily in the posterior superior and middle temporal lobe (Table 2). At all 29 sites, auditory comprehension, as measured by the Token Test, was severely impaired. Patients were unable to perform correctly any of the 10–15 single-step verbal commands administered during electrical interference. All reported hearing but not understanding the examiner. Accuracy of word and non-word repetition was ≥93%. Although spontaneous speech remained fluent, all six patients made numerous semantic (e.g. pencil > stick) and phonological (e.g. nearly > /orli/) paraphasias during electrical interference testing. At all TSA sites, patients performed flawlessly on syllable discrimination (CV, CVC). At 19 of the 29 TSA sites, across all six patients, naming was impaired. In almost all cases, patients gave no response during the 5-s period of current activation, frequently reporting that they were unable to ‘think of the word.’

For five of six patients, naming and word reading remained intact at 10 electrode pairs, where the pattern of language deficits was otherwise identical to that elicited at other TSA sites (Fig. 3).

Wernicke’s aphasia was induced at a single electrode pair in the posterior superior or middle temporal gyri of each patient (Fig. 4). At this site, auditory comprehension,
Fig. 1 Location of electrodes in posterior left cortex where language was tested across all six patients. Electrode locations have been normalized within a standard brain atlas.

Fig. 2 Location of electrode sites where TSA was induced by electrical interference. Auditory comprehension was impaired, repetition was spared and speech remained fluent.

repetition and naming were impaired, while speech remained fluent. Repetition errors, for words and non-words, consisted primarily of no responses. This was the only site where syllable (CVC and CV) discrimination was impaired. Patients discriminated vowels better than consonants. This difference was highly significant for five of the six patients tested (\( P < 0.001 \), Fisher’s exact test) and borderline significant for the sixth patient (\( P = 0.080 \), Fisher’s exact test). No differences were observed for CVC versus CV discrimination (\( P > 0.147 \), Fisher’s exact test). A significant relationship
was also observed between word repetition and syllable (CVC, CV) discrimination \((P = 0.001, \text{ Cochran Mantel–Haenszel } \chi^2 \text{ test})\) across sites. Because of the small sample size, however, the validity of this association must be viewed cautiously.

**Discussion**

TSA was induced transiently in all six of our patients by electrical interference through electrodes implanted on lateral left posterior cortex. To our knowledge, this is the first time TSA has been investigated with this technique. For each patient we identified multiple TSA sites, located in the temporal lobe, the temporo-occipital region and the parietal lobe. Although these areas have been identified separately in previous stroke studies, electrical interference mapping identified multiple sites in the same patients. At a subset of sites where repetition remained intact but comprehension was impaired, naming and word reading also remained intact. Results of language lateralization testing by IAI showed no evidence of independent right hemisphere phonological or lexical–semantic capabilities. These findings cannot be explained entirely within current models of TSA and provide the framework for a revised model that incorporates a one-way disruption of access from phonology to otherwise intact left hemisphere lexical–semantic processing.

**Table 2 Number of TSA sites induced during electrical interference testing**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Electrode sites</th>
<th>STG</th>
<th>MTG</th>
<th>ITG</th>
<th>TOJ</th>
<th>PL</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>2</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>12</td>
<td>18</td>
<td>7</td>
<td>4</td>
<td>2</td>
<td>43 sites (29 pairs)</td>
</tr>
</tbody>
</table>

STG = superior temporal gyrus; MTG = middle temporal gyrus; ITG = inferior temporal gyrus; TOJ = temporo-occipital junction; PL = parietal lobe.

**Implications for previous models**

A premise of the disconnection model is that Wernicke’s area is spared and isolated in TSA (Lichtheim, 1885; Goldstein, 1948; Geschwind, 1965). In our patients, TSA was elicited at multiple sites along the posterior superior and middle temporal gyri, in classical Wernicke’s area (Wernicke, 1874; Luria, 1976; Damasio, 1981; but see also Bogen and Bogen, 1976). Although Wernicke’s area has been implicated in previous studies of stroke patients with TSA, it has been difficult to determine the precise role of this area because of concurrent damage to other cortical regions (Goldstein, 1948; Kertesz et al., 1979; Berthier et al., 1991). Results of our electrical interference mapping studies indicate that sparing of Wernicke’s area is not a necessary condition for TSA.

The sparing of naming and word reading, despite impaired auditory comprehension, suggests that access to phonology was intact from other left hemisphere language systems,
including lexical–semantics. Whereas phonology may be accessed directly in word reading (Coltheart, 1985), it is mediated by lexical–semantic information in naming (Gordon, 1997). The sparing of naming suggests that both the lexical–semantic system and its connections to phonology remained intact (see also Gordon, 1982). A previous report of TSA with intact naming invoked right hemisphere lexical–semantic capabilities, making this finding more compatible with the disconnection model (Heilman et al., 1981). This cannot explain our patients’ intact naming, however, because IAI testing showed no evidence of independent right hemisphere lexical–semantic or phonological capabilities.

It may be argued that we induced pure word deafness and not TSA at those sites where only auditory comprehension was impaired. The concurrent sparing of repetition is not consistent, however, with this hypothesis. Furthermore, pure word deafness is usually associated with bilateral or deep unilateral left hemisphere lesions. In our study, electrodes were implanted only over the left cortex. Also, effects of electrical interference are restricted to cortical grey matter (Nathan et al., 1993).

Phonological processing, as measured by syllable (CV, CVC) discrimination, was strongly correlated with patients’ repetition performance. When phonological processing was impaired in Wernicke’s area, repetition of words and non-words was also impaired, as was naming. This pattern has been reported previously in stroke patients, as well as patients undergoing electrical interference language mapping (Wernicke, 1874; Luria, 1976; Selnes et al., 1985; Ojemann, 1991; Boatman et al., 1995). Their poor consonant discrimination is consistent with the view that consonant perception is especially dependent on phonological processing (Liberman et al., 1967; Blumstein, 1990). Conversely, when phonological processing was intact, repetition of words and non-words was also intact. It has been argued that TSA reflects either a partial phonological impairment or mediation by right hemisphere phonological processing (Goldstein, 1948; Berthier et al., 1991). Our results fail to support either hypothesis. With TSA, patients’ syllable discrimination performance did not deviate from baseline. Moreover, IAI testing showed no evidence of independent right hemisphere phonological capabilities. This is not to say that the right hemisphere is incapable of phonological processing. Other studies, including one of our own, have documented right hemisphere phonological capabilities in some individuals (Berthier et al., 1991; Boatman et al., 1998). This does not appear to be the case, however, for the six patients studied here.

The sparing of phrasal repetition with TSA suggests that patients’ auditory–verbal memory capabilities were most likely sufficient for phrasal comprehension, thereby addressing the alternative possibility of an underlying auditory–verbal memory deficit (Shallice and Warrington, 1977; Friedrich et al., 1984). It may be argued that impaired comprehension in TSA reflects an underlying deficit in syntactic processing. Although we did not test this possibility directly, it seems unlikely because syntactic deficits are usually associated with frontal lesions (Ritter von Stockert, 1972; Grodzinsky, 1984), whereas our testing was restricted to posterior cortical areas.

Although TSA occurs relatively infrequently in stroke patients, it was induced at multiple sites in all six of our
patients. We also found fewer strictly posterior cortical sites associated with TSA than reported previously in the stroke literature. These discrepancies may reflect methodological differences. The proximity of TSA to sites associated with phonological processing increases the likelihood that structural lesions would induce Wernicke’s aphasia, compared with the more circumscribed electrical interference effects. The smaller number of posterior TSA sites identified in this study could also reflect limited posterior electrode coverage. Future studies with more extensive posterior coverage may help resolve this discrepancy. Another potential issue is that transient TSA induced by electrical interference may differ, in as yet unknown ways, from TSA in stroke patients. Finally, although our patients were screened to exclude those with functional or structural abnormalities, and results of cortical function mapping showed no evidence of atypical language organization, the long-term effects of their seizures may differentiate them from other populations.

**Revised model of TSA**

The sparing of Wernicke’s area is a basic premise of the disconnection model of TSA. Our results indicate, however, that sparing of Wernicke’s area is not a necessary condition for TSA. Wernicke’s area has also been implicated in several previous studies of stroke patients with TSA (Kertesz et al., 1982; Berthier et al., 1991). However, it has been difficult to confirm this association because stroke lesions typically involve multiple areas. Because effects of electrical interference are relatively circumscribed, it was possible to induce TSA within Wernicke’s area without involvement of other cortical areas.

At a subset of sites where the pattern of language deficits otherwise resembled TSA, naming was spared. This suggests that TSA does not reflect an impairment of the lexical–semantic system itself, but rather impaired access to lexical–semantic information. The sparing of naming with intact repetition and impaired comprehension cannot be explained within traditional models of TSA. It has been suggested that the sparing of naming reflects mediation by right hemisphere lexical–semantic functions (Heilman et al., 1981). However, IAI testing in our patients showed no evidence of independent right hemisphere lexical–semantic capabilities. A more likely explanation is that there is a one-way disruption between otherwise intact left hemisphere phonology and lexical–semantic processing. One implication is that either the current definition of TSA be revised to include the possibility of intact naming, or that a separate TSA subtype be defined, as others have suggested (Heilman et al., 1976). The rarity of TSA cases in which naming is spared argues for the latter. However, because a naming impairment is often included as one of the clinical criteria for identifying TSA, the incidence of such cases may have been underestimated. Regardless of which way this issue resolves, our results confirm that a one-way disruption can occur between otherwise intact left hemisphere phonology and lexical–semantic processing in patients who have impaired auditory comprehension, with spared repetition and fluent speech.

**Acknowledgements**

We wish to thank Barbara Czyzck for assistance with patient testing, Moona Alishost for technical assistance, and Dr Stephen Reich and Dr Pamela Talalay for insightful comments on earlier drafts. We also wish to thank especially Dr Brenda Rapp, Dr Michael McCloskey and Dr Marie Josephtanturier for thoughtful and timely discussions. This research was supported by NIH grant DC03081 to D.B.

**References**


