

SHORT REPORT

# Conduction aphasia elicited by stimulation of the left posterior superior temporal gyrus

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**Abstract**

**Objective**—Disruption of fascicular tracts that connect Wernicke’s to Broca’s areas is the classic mechanism of conduction aphasia. Later work has emphasised cortical mechanisms.

**Methods**—To determine the distribution of language on dominant cortex, electrical cortical stimulation was performed using implanted subdural electrodes during brain mapping before epilepsy surgery.

**Results**—A transient, isolated deficit in repetition was elicited with stimulation of the posterior portion of the dominant superior temporal gyrus.

**Conclusion**—This finding suggests that cortical dysfunction, not just white matter disruption, can induce conduction aphasia.

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Conduction aphasia is thought to result from lesions of the arcuate fasciculus that disconnect receptive from expressive language regions. It consists of impaired repetition with preserved comprehension, naming, and reading. Literal paraphasias are frequent, and ideomotor apraxias can also be present.

The lesions that most often produce conduction aphasia involve the white matter underlying the dominant supramarginal gyrus.<sup>1-3</sup> The disconnection between expressive and receptive cortical regions was hypothesised by Wernicke and led to the first description of conduction aphasia. However, it has been reported in lesions that spare white matter, and is unexpectedly absent in some cases involving the classic lesion.<sup>4-7</sup> Some of these difficulties in localisation have arisen because naturally occurring lesions may indiscriminately involve both white and grey matter. These inconsistencies, in part, support an alternative hypothesis that conduction aphasia may be mediated by a specific region of cortex.<sup>8</sup>

We report a case of transient conduction aphasia elicited by electrical cortical stimulation of the posterior superior temporal gyrus. The selective and reversible impairment of a specific region of cortex shows that conduction aphasia may be induced by means other than disconnection.

**Case report**

A 35 year old right handed woman had intractable complex partial seizures consisting of parosmias, tachycardia, and confusion of a year’s duration. Biopsy of the left temporal pole at another institution had disclosed a left frontotemporal oligodendroglioma.

The patient presented to the comprehensive epilepsy program 4 months later with continued seizures. Neurological examination was normal with findings of intact comprehension, repetition, and naming. Brain MRI disclosed a large, non-enhancing, low intensity lesion in the left frontotemporal region abutting the insula and extending to the basal ganglia (figure (A-C)). The arcuate fasciculus appeared compromised anteriorly. Continuous EEG with video monitoring showed seizure onset in the left temporal region.

Speech was fluent on the preoperative neuropsychological battery. On the Reitan-Indiana aphasia screening test the patient transposed syllables when repeating the word “episcopal” forming “ecispopal.”

For seizure localisation and cortical mapping, intracranial monitoring with subdural grids and occipitally inserted hippocampal depth electrodes was performed before left temporal lobectomy and partial subfrontal tumour resection. Subsequent histopathology confirmed the presence of oligodendroglioma.

Four months postoperatively the patient was tested with the Boston aphasia severity rating scale. Impaired repetition with mixed paraphasic errors became evident only when tested with complex, low probability vocabulary.

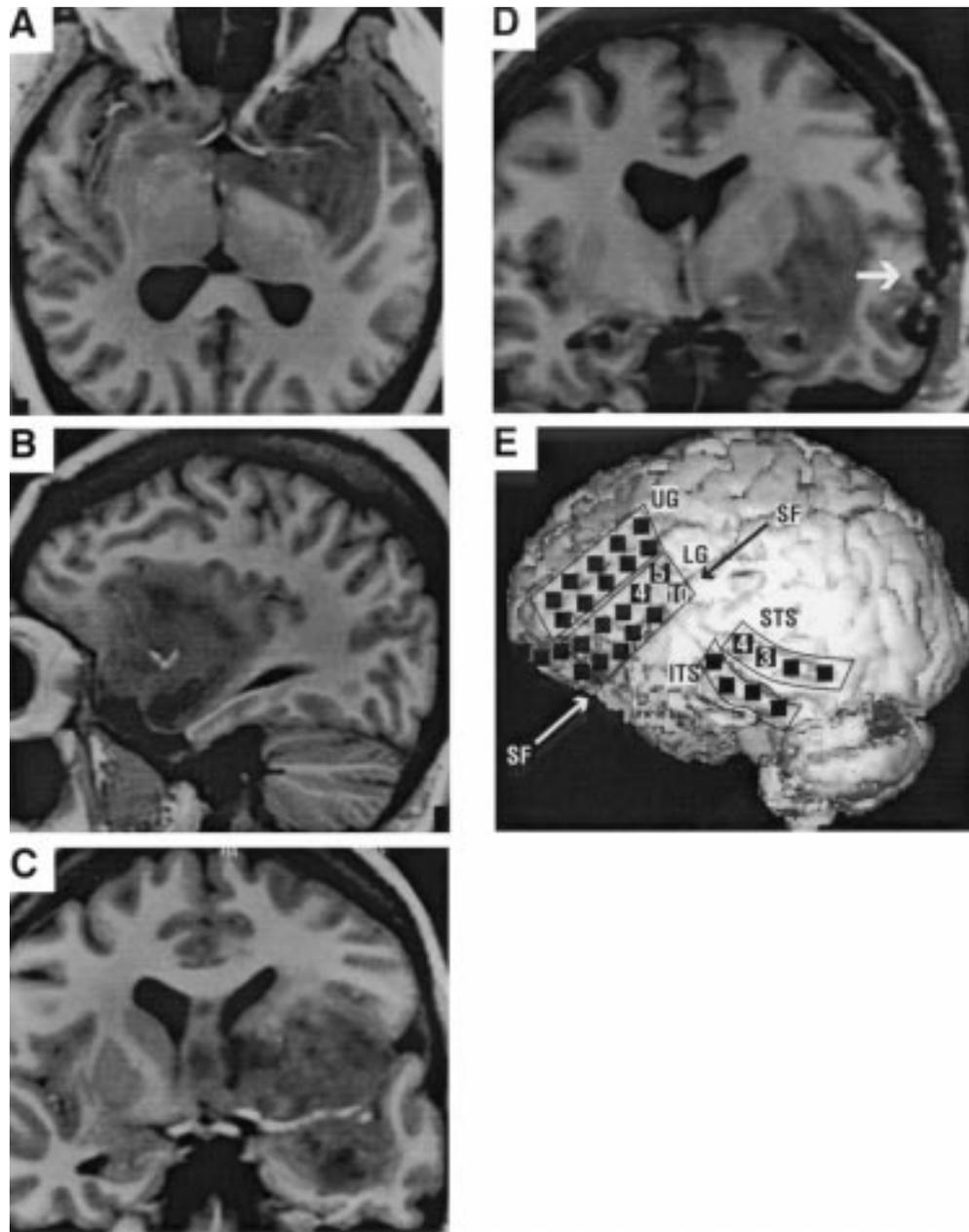
**Methods**

Informed consent was obtained before procedures were carried out. Subdural grids and strips (intercontact distance=1 cm) were placed on the left frontal lobe and temporal lobe as shown on the figure (D and E). We stimulated adjacent pairs of contacts using a 50 Hz square wave signal starting at a duration of 2 seconds and an intensity of 4.5 mA. We increased duration to 5 seconds and increased current by 1 mA increments until (1) a response occurred, (2) an afterdischarge or clinical seizure occurred, or (3) no response was achieved at a stimulus of 12.5 mA/5 seconds.

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(A) Axial, (B) sagittal, and (C) coronal views of a T1 weighted, uncontrasted MRI taken before placement of subdural electrodes demonstrates the large, frontotemporal cystic mass of the dominant hemisphere. The arcuate fasciculus is intact posteriorly but is difficult to identify anteriorly near Broca's region. (D) Coronal view after electrode placement confirming location of electrode STS 4 upon the superior temporal gyrus (white arrow). (E) Three dimensional MRI reconstruction showing the array of subdural electrodes. Stimulation at pairs LG4, 5, and 10 caused either global or non-fluent aphasia, and stimulation at STS3,4 caused conduction aphasia. STS and ITS=superior and inferior temporal strip; UG and LG=upper and lower grid; SF=Sylvian fissure.

### Results

Stimulation in adjacent contact pairs (lower grid electrodes 4, 5, and 10 in the figure (E)) induced anomia and speech arrest in Broca's region. Stimulations of 5 seconds duration and 6 mA intensity at electrodes 3 and 4 of the superior temporal strip (figure (E)) induced a language disorder similar to conduction aphasia: (1) recitation was normal; (2) naming "thumb," "index finger," and "tie" was normal; (3) command following—"show me your thumb"—was normal; and (4) repetition—"Bill Clinton", "Commonwealth of Virginia"—was reproducibly impaired. No paraphrastic errors were found. No afterdis-

charges were elicited. Stimulations at other sites had no clinical manifestations.

### Discussion

Our finding of an isolated deficit of repetition elicited by electrical cortical stimulation is relevant because it runs counter to the prevailing view that disconnection of a white matter tract is necessary for conduction aphasia. The precise localisation offered by cortical stimulation suggests that a focal region of the posterior superior temporal gyrus of the language dominant hemisphere can mediate language dysfunction most consistent with conduction aphasia. Because practical considerations limit

complex testing during intracranial electrical stimulation, we were only able to test the basic abilities of naming, recitation, repetition, and command following. We were not able to test other findings that define the full syndrome of conduction aphasia.<sup>2</sup>

The arcuate fasciculus is a white matter tract that runs from Wernicke's area in the posterior superior temporal gyrus, arches around the sylvian fissure, and runs anteriorly from the inferior parietal lobe to the inferior frontal lobe of Broca's region.<sup>3</sup> Wernicke postulated that a lesion of the arcuate fasciculus that disconnected receptive from expressive centres would produce a deficit in repetition, or "conduction aphasia". Others have proposed that a single cortical centre was responsible for integration of receptive and expressive regions yet was independent of them.<sup>8</sup>

Evidence from subjects with conduction aphasia usually supports the concept of disconnection. In these studies (usually patients with strokes), disruption of the arcuate fasciculus is obligate with variable involvement of adjacent regions of suprasylvian or subsylvian cortex.<sup>1-3</sup> Circumscribed lesions of the arcuate fasciculus that spared overlying cortex have been offered as evidence of disconnection,<sup>9-11</sup> but white matter lesions are not able to differentiate between the relative importance of disruption of the arcuate fasciculus versus disconnections of overlying neurons along its course.

Physiological findings have also supported disconnection as the mechanism of conduction aphasia. Regional blood flow determined by xenon CT was absent in Broca's region in stroke patients with conduction aphasia, suggesting functional disconnection.<sup>12</sup> However, studies of cortical strokes, in determinations of cortical versus subcortical mechanisms, can be misleading because regions of destruction involve both cortex and the arcuate fasciculus.

Finally, electrical stimulation of eloquent cortex produced both Broca's and Wernicke's aphasias but not conduction aphasia,<sup>13</sup> suggesting that conduction aphasia is not cortically mediated. Notably, in this series of patients with implanted subdural electrodes, the testing paradigm involved mainly reading aloud, and repetition may not have been adequately tested.<sup>13</sup> Thus this finding may have been missed in other patients and may not be unique to our patient.

Other studies suggest that disconnection may not be the only mechanism of conduction aphasia. Some cases of conduction aphasia were caused by lesions that clearly spared the arcuate fasciculus.<sup>4-5</sup> Similarly, lesions of the arcuate fasciculus have not always resulted in conduction aphasia.<sup>6</sup> Furthermore, physiological data provided by PET imaging does not clearly support the disconnection theory. In one study of stroke and conduction aphasia, cerebral metabolic patterns had no clear correlation to clinical findings,<sup>7</sup> suggesting that functional disconnection is not necessary to produce conduction aphasia.

Our findings support the view that conduction aphasia is a cortically based phenomenon. At the least, this case shows that cortex in

addition to its connecting white matter tracts is necessary to mediate repetition. Two findings support this conclusion. Firstly, the large frontotemporal tumour compromised the arcuate fasciculus but spared the overlying cortex (figure). Despite the white matter lesion, the patient had no preoperative evidence of a significant conduction aphasia (although subtle findings were disclosed after partial resection). Secondly, electrical stimulation of the superior temporal gyrus elicited transient conduction aphasia, specifically demonstrating that cortical function is necessary for normal repetition.

The large frontotemporal tumour could confound our interpretation. It is theoretically possible that eloquent cortex was abnormally represented as a consequence of a chronic mass lesion. By this "remapping" process, stimulation of a makeshift language area may have unexpectedly resulted in conduction aphasia, even though this region might not normally mediate this function in other subjects. This process seems unlikely as the tumour displaced no other language functions, such as expressive language in Broca's region.

Another consideration is the possibility that electrical stimulation was not confined to the cortex, and non-specific electrical disruption of underlying arcuate fasciculus may account for our findings. Basic electrical properties and previous studies suggest that neuronal activation caused by electrical stimulation remains highly confined to the immediate vicinity of the stimulating electrodes, especially at the low current required.<sup>14-15</sup> Furthermore, there was no measurable afterdischarge that could spread to surrounding regions and confound results. Electrical stimulation, unlike clinicopathological correlations in stroke, more selectively separates cortical from white matter dysfunction.

In our patient, the posterior superior temporal gyrus was spared during partial resection of the tumour, in part because of our mapping of conduction aphasia, but mainly because the tumour involved more anterior portions of the temporal lobe. Our findings suggest that stimulation paradigms should test for conduction aphasia if the area of potential resection involves the posterior, perisylvian cortex. Risk and benefits of a resection amidst eloquent cortex must be calculated on an individual basis.

In summary, these findings show that dysfunction of the posterior superior temporal cortex can induce a syndrome similar to conduction aphasia. This study suggests that conduction aphasia is not simply a disconnection syndrome but can result from stimulation of the posterior perisylvian cortex.

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