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A multimodal mapping study of conduction aphasia with impaired repetition and spared reading aloud

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ABSTRACT

The present study explores the functional neuroanatomy of the phonological production system in an Italian aphasic patient (SP) who developed conduction aphasia of the reproduction type following brain surgery. SP presented with two peculiar features: (1) his lesion was localized in the superior temporal gyrus, just posterior to the primary auditory cortex and anterior/inferior to and neighboring the Sylvian parietal temporal (Spt) area, and (2) he presented with severely impaired repetition and spelling from dictation of words and pseudowords but spared reading-aloud of words and pseudowords.

Structural, functional, fiber tracking and intraoperative findings were combined to analyze SP's pattern of performance within a widely used sensorimotor control scheme of speech production.

We found a dissociation between an interrupted sector of the arcuate fasciculus terminating in STG, known to be involved in phonological processing, and a part of the arcuate fasciculus terminating in MTG, which is held to be involved in lexical-semantic processing. We argue that this phonological deficit should be interpreted as a disorder of the feedback system, in particular of the *auditory* and *somatosensory target maps*, which are assumed to be located along the Spt area. In patient SP, the spared part of the left arcuate fasciculus originating in MTG may support an unimpaired reading performance, while the damaged part of the left arcuate fasciculus originating in STG may be responsible for his impaired repetition and spelling from dictation.

1. Introduction

Conduction aphasia (CA) is a language deficit characterized by disordered repetition and good comprehension (e.g., Benson and Ardila, 1996). According to Wernicke's (1874) associationist model of language processing, CA is caused by a disconnection between the area processing recognition of sound images of words (the posterior superior part of the temporal lobe, also known as Wernicke's area) and the area where the motor images of words are represented (the posterior part of the left inferior frontal gyrus, also known as Broca's area), owing to a lesion of the left arcuate fasciculus (Wernicke, 1874; see De Bleser et al., 1993 for a review). The deficit is due to an impaired encoding of phonological word forms and their mapping to the associated sequential articulatory gestures. CA is characterized by phonemic paraphasias, phonemic neologisms, continuous repairs, and conduite d'approche behavior (repetitive self-correction attempts) (Anderson et al.,. 1999; Buchsbaum et al., 2011; Fridriksson et al., 2010; Goodglass, 1992; Hickok, 2000; Hillis, 2007).

Besides the classical Wernicke's description of CA, a further condition of disproportionate repetition deficit has been reported by Shallice and Warrington (1977) who distinguished between *Reproduction* and *Repetition* subtypes. In the repetition subtype of CA (see also Caplan et al., 1986), patients do not present with phonemic paraphasias, phonemic neologisms, continuous repairs, and conduites d'approche but with a selective damage of phonological short-term memory.

The functional damage causing reproduction AC, rather than as a mere disconnection between the phonological input and output lexicons, is usually localized at the level of the phonological output buffer (Baldo et al., 2008; Caramazza et al., 1986; Romani, 1992;

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Shallice et al., 2000) or at the level of the interface between the buffer and the phonological output lexicon. Activation in areas involving the left frontal inferior gyrus and including parts of the middle frontal and inferior precentral gyri (Chein and Fiez, 2001) might correspond to functional processing in the phonological output buffer (Jacquemot and Scott, 2013). In particular, Baddley (2003) localized the phonological output buffer in Broca's area/the premotor cortex (for a review see Jacquemot and Scott, 2013). However, this localization is not consistent with a frequently reported lesion in CA patients, namely damage to the left temporal/inferior parietal area, although a lesion of the posterior temporal/parietal area might impact on Broca's area/the premotor cortex since the arcuate fasciculus connects these areas. Furthermore, a functional damage to the phonological output buffer hypothesized by the cognitive information processing models (e.g. Patterson, 1996) implies a disproportionate phonological deficit not only in repetition but also during reading-aloud of words and pseudowords (a prediction that is actually implicit also for CA following Wernicke–Lichtheim's account, since reading-aloud, too, would require a functional association pathway running in the external capsule, such as the arcuate fasciculus).

According to one of the recent dual-stream models of speech processing (Hickok and Poeppel 2007), CA is a deficit of sensory-motor integration for speech. In other words, a disproportionate phonological deficit in repetition can be explained as a damage to a system serving as interface between the auditory target and the corresponding motor speech output (Hickok, 2000; Hickok et al., 2000, 2003; Hickok and Poeppel, 2004, 2007). Thus, the deficits in repetition and speech production characterizing CA of the reproduction subtype reflect an impairment in the capacity of *auditory* representations of speech to constrain and guide the corresponding articulatory representations (Hickok, 2000; Hickok and Poeppel, 2004; Wise et al., 2001). Hickok and Poeppel argue that inputs to the auditory-phonological network define the auditory targets of speech acts. The predicted auditory consequences of a motor speech unit can be checked against the auditory target. If the anticipated speech sounds match the auditory targets, such representation remains active and a normal articulation process follows; if they mismatch, a correction signal is generated to activate the correct motor unit. A lesion to the Sylvian parietal-temporal (Spt) area would disrupt the ability to generate forward predictions in the auditory cortex and thus the ability to perform internal feedback monitoring.

In the present study, we analyzed the phonological processing of SP, a conduction aphasic patient suffering a profound repetition deficit, fluent but paraphasic speech output, and preserved auditory comprehension. SP presented with two peculiar features: (i) impaired word and pseudoword repetition and spelling from dictation but spared word and pseudoword reading abilities, and (ii) his lesion was localized in the superior temporal gyrus, just posterior to the primary auditory cortex and anterior/inferior to the Spt area. If SP's deficits were of the "repetition subtype" (Shallice and Warrington, 1977), his pattern of performance (repetition deficit, spelling deficit, and spared reading aloud) could have simply been explained as due to a phonological short-term memory deficit. However, this view fails to account for SP's phonemic paraphasias, phonemic neologisms, continuous repairs and conduite d'approche behavior.

In addition, the dissociation between spared reading and impaired repetition and writing to dictation is unusual (Ardila, 2010; Benson et al., 1973) showing that phonemic errors in reading-aloud (Goodglass, 1992) as well as writing (from mild spelling difficulties to profound agraphia) (Bernal and Ardila, 2009) parallel the phonological production errors in CA (see also (Caramazza et al., 1981) for a similar observation). In fact, very few studies on CA investigated written expression in detail (Balasubramanian, 2005). None of the literature cases in which the patients' reading and spelling performance was reported showed a pattern of damage with impaired repetition and spelling from dictation but no reading-aloud deficit, as found for patient SP (see Fridriksson et al., 2010 for a review).

In the present multi-modal mapping study we investigated the functional neuroanatomy of SP's performance. We combined structural and functional brain imaging techniques, intraoperative stimulation mapping as well as neuropsychological testing to better understand the neurological and functional substrates involved in CA with impaired phonological output in repetition, but spared phonological output in reading-aloud. The dissociation shown by SP cannot be explained by a standard information processing model of word and pseudoword processing (e.g. Patterson, 1986), because models postulate that the phonological deficit characterizing reproduction AC does necessarily involve both repetition and reading-aloud performance. As already reported, the same prediction is implicitly suggested by Wernicke and Lichtheim's account of CA, since reading-aloud requires the contribution of the arcuate fasciculus via the external capsule. We focused on the examination of the dorsal stream and, in particular, made reference to an anatomical dissociation reported by Glasser and Rillings (2008) between the part of the superior longitudinal fasciculus (SLF) terminating in the middle temporal gyrus, (MTG, which is the anatomical counterpart of the dorsal stream), and the part terminating in the superior temporal gyrus (STG) (see Catani et al., 2005 for a different reconstruction account of the SLF).

2. Materials and methods

2.1. Participants

SP is a 42-year-old, right-handed (Oldfield, 1971), monolingual native speaker of Italian. He has an educational level of 13 years and works as a fireman. He was admitted to the Udine General Hospital one week before the study start for a seizure with loss of consciousness. A neurological examination did not reveal any focal deficits and any overt signs of language impairment. The patient had no family history of developmental language problems or learning disabilities nor had suffered neurological problems in the past.

Conventional T2-weighted MR imaging revealed a low-grade glioma (approximately 6.4 cc in volume). According to the Anatomy toolbox (Eickhoff et al., 2005), the region of interest (ROI) drawn on the patient's lesion and normalized to the MNI template was localized in the left superior and middle parts of the temporal lobe and lays posterior to the primary auditory cortex (as evidenced in Fig. 1A and C).

The patient's fMRI and DTI maps were compared with those obtained from a group of 18 monolingual Italian native speakers acting as controls (12F, 6M; mean age 47.7 ± 7.6, age range 35–61; handedness: mean laterality index 94.0 ± 9.8, range 66.7–100; education: mean 13 years ± 0).

All participants had normal or corrected-to-normal vision and reported no history of neurological illness (except for the patient), psychiatric disease, or drug abuse. The participants' informed consent to participate in the study was obtained in line with the Declaration of Helsinki. The study was approved by the local Ethics Committee.

The glioma was removed surgically about 20 days after the clinical diagnosis. Fig. 1B and D portrays the removal of the tumor from the left hemisphere.