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Research report

Fundamental deficits of auditory perception in Wernicke's aphasia

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ABSTRACT

Objective: This work investigates the nature of the comprehension impairment in Wernicke's aphasia (WA), by examining the relationship between deficits in auditory processing of fundamental, non-verbal acoustic stimuli and auditory comprehension. WA, a condition resulting in severely disrupted auditory comprehension, primarily occurs following a cerebrovascular accident (CVA) to the left temporo-parietal cortex. Whilst damage to posterior superior temporal areas is associated with auditory *linguistic* comprehension impairments, functional-imaging indicates that these areas may not be specific to speech processing but part of a network for generic auditory analysis.

Methods: We examined analysis of basic acoustic stimuli in WA participants ($n = 10$) using auditory stimuli reflective of theories of cortical auditory processing and of speech cues. Auditory spectral, temporal and spectro-temporal analysis was assessed using pure-tone frequency discrimination, frequency modulation (FM) detection and the detection of dynamic modulation (DM) in "moving ripple" stimuli. All tasks used criterion-free, adaptive measures of threshold to ensure reliable results at the individual level.

Results: Participants with WA showed normal frequency discrimination but significant impairments in FM and DM detection, relative to age- and hearing-matched controls at the group level ($n = 10$). At the individual level, there was considerable variation in performance, and thresholds for both FM and DM detection correlated significantly with auditory comprehension abilities in the WA participants.

Conclusion: These results demonstrate the co-occurrence of a deficit in fundamental auditory processing of temporal and spectro-temporal non-verbal stimuli in WA, which may have a causal contribution to the auditory language comprehension impairment. Results are discussed in the context of traditional neuropsychology and current models of cortical auditory processing.

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1. Introduction

Wernicke's aphasia (WA) is an acquired language impairment characterised by severely impaired single-word comprehension and repetition with fluent but disordered speech. WA most commonly results from a cerebrovascular accident (CVA) to the left posterior temporo-parietal cortex, affecting areas involved in semantic, phonological and auditory processing. The close proximity of these posterior temporal lobe language-related cognitive systems often results in lesions to this region impacting multiple systems. As a result, WA is a behaviourally and cognitively heterogeneous disorder. The presence of semantic and phonological impairments have been documented in WA (Baker et al., 1981; Blumstein et al., 1977; De Renzi et al., 1972; Ogar et al., 2011; Robson et al., 2012b). In contrast, while auditory processing deficits have been suggested in WA (Auerbach et al., 1982; Kirshner et al., 1981; Polster and Rose, 1998), they have been little explored experimentally. This study investigated fundamental, non-linguistic auditory processing in WA in relation to current models of cortical auditory processing and explored the relationship with auditory language comprehension.

Whilst WA has been shown to be globally cognitively heterogeneous, the classical view suggested an instability in phonological word-form representations as the cause of the comprehension deficit (Luria, 1976; Luria and Hutton, 1977). This view is consistent with recent behavioural findings that suggest a phonological impairment is a critical cognitive component of WA (Robson et al., 2012b) and the classical lesion distribution in WA, which overlaps with phonological-processing regions in the left superior temporal lobe (Price et al., 2005). The current study investigated participants who could be described as having *classical* WA, i.e., individuals with core phonological deficits. Two pieces of evidence, however, support previous proposals that phonological deficits in WA may be associated with a more fundamental impairment in auditory analysis. Firstly, behavioural work has shown that individuals with WA can have deficits in discriminating phonemes with very different acoustic structures, for instance in distinguishing a /b/from an /f/ (Robson et al., 2012a). If only damage to more abstract phonological representations had occurred, one might expect that early auditory processing would still allow detection of a difference between the stimuli based on their considerably different acoustic structure. Secondly, lesions in WA frequently include primary and non-primary auditory regions in the left-hemisphere (Bogen and Bogen, 1976; Ogar et al., 2011). These regions respond to both generic and speech-related acoustic stimuli: noise (Binder et al., 2000), pure tones (Binder et al., 2000; Hall et al., 2000), modulated tones (Binder et al., 2000; Hall et al., 2000), frequency sweeps (Husain et al., 2004), harmonic sounds (Menon et al., 2002), and phonological stimuli (e.g., Benson et al., 2001; Binder et al., 2000; Price, 2010; Scott et al., 2006). These neural activation patterns imply that speech and non-speech perception systems may be subserved by the same, or highly overlapping, cortical network.

Neural activations in response to verbal and non-verbal sounds (in contrast to rest) are strongly bilateral. However, contrasts between different types of acoustic stimuli reveal differential response patterns, reflecting a hierarchical and in part lateralized organisation within the network. The auditory cortices display a functional architecture similar to the homologous organisation intensively studied in the macaque brain (Chevillet et al., 2011; Petkov et al., 2006). Primary auditory regions appear maximally responsive to auditory stimuli with the most simple acoustic structure; with the surrounding secondary and tertiary association auditory cortices responding preferentially to increasingly complex auditory stimuli (Rauschecker and Tian, 2004; Tian and Rauschecker, 2004). In addition, functional asymmetries between the left and right-hemisphere have been proposed, whereby the left and right auditory cortices display differential sensitivity to acoustic properties. Specifically, the right-hemisphere has been suggested to process spectral information (how energy is distributed across the frequency spectrum) (Schonwiesner et al., 2005a,b; Zatorre and Belin, 2001) and of changes in the spectrum over longer time windows of several hundreds of milliseconds (Boemio et al., 2005), while the left-hemisphere has been proposed to preferentially respond to rapid changes over shorter time windows of less than 50 msec (Boemio et al., 2005).

The lesion distribution in WA corresponds to left-hemisphere regions implicated in both speech and non-speech auditory analysis, but leaves the possibility that intact right-hemisphere auditory structures could be able to support non-verbal auditory analysis. However, current neuropsychological evidence indicates that unilateral brain lesions can cause fundamental auditory processing impairments, which are, for the most part, consistent with the neuroimaging literature and theories of auditory network organisation. Non-verbal auditory processing deficits have been identified in a range of unilateral lesions with mixed aetiology including CVA (Biedermann et al., 2008; Bungert-Kahl et al., 2004; Divenyi and Robinson, 1989; Fink et al., 2006; Robin et al., 1990), temporal or frontal lobectomy following epilepsy (Samson and Zatorre, 1988; Samson et al., 2002) and tumour (von Steinbuchel et al., 1999). Such studies have demonstrated dissociations between groups of individuals with right and left-hemisphere pathology. Individuals with right-hemisphere lesions showed more frequency and more severe impairments for spectrally based judgement tasks (such as frequency discrimination, pitch matching and timbre analysis), whilst individuals with left-hemisphere lesions show more frequent and severe impairments in temporal judgement tasks (including gap detection, pattern judgements and click fusion) (Divenyi and Robinson, 1989; Robin et al., 1990; Samson and Zatorre, 1988; Samson et al., 2002). In particular, auditory temporal processing impairments have been associated with (pure) word deafness; a condition resulting in an isolated speech perception deficit with intact speech production, typically resulting from bilateral lesions (Albert and Bear, 1974; Auerbach et al., 1982; Griffiths et al., 2010; Otsuki et al., 1998; Pinard et al., 2002; Wang et al., 2000), and in approximately 30% from unilateral lesions to the left superior temporal lobe (Poeppel, 2001). Such individuals show impairments in discriminating verbal and non-verbal stimuli containing rapid temporal modulations (Albert and Bear, 1974; Slevc et al., 2011; Stefanatos et al., 2005). This impairment is thought to disproportionally affect speech