



The cortical signature of impaired gesturing: Findings from schizophrenia



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ABSTRACT

Schizophrenia is characterized by deficits in gesturing that is important for nonverbal communication. Research in healthy participants and brain-damaged patients revealed a left-lateralized fronto-parieto-temporal network underlying gesture performance. First evidence from structural imaging studies in schizophrenia corroborates these results. However, as of yet, it is unclear if cortical thickness abnormalities contribute to impairments in gesture performance. We hypothesized that patients with deficits in gesture production show cortical thinning in 12 regions of interest (ROIs) of a gesture network relevant for gesture performance and recognition. Forty patients with schizophrenia and 41 healthy controls performed hand and finger gestures as either imitation or pantomime. Group differences in cortical thickness between patients with deficits, patients without deficits, and controls were explored using a multivariate analysis of covariance. In addition, the relationship between gesture recognition and cortical thickness was investigated. Patients with deficits in gesture production had reduced cortical thickness in eight ROIs, including the pars opercularis of the inferior frontal gyrus, the superior and inferior parietal lobes, and the superior and middle temporal gyri. Gesture recognition correlated with cortical thickness in fewer, but mainly the same, ROIs within the patient sample. In conclusion, our results show that impaired gesture production and recognition in schizophrenia is associated with cortical thinning in distinct areas of the gesture network.

1. Introduction

Gestures are an important and integral part of communication (Goldin-Meadow and Alibali, 2013; Hostetter, 2011). They are not only important for language production and comprehension in verbal communication, but also play a crucial role in nonverbal communication, as they may transmit information on their own (Goldin-Meadow and Alibali, 2013).

Research on gestures may either focus on recognition and interpretation of gestures, or on gesture production (Walther and Mittal, 2016). The production of gestures can be investigated in two domains: imitation (following demonstration) and pantomime (after verbal instruction). The semantic categories of gestures can be meaningless,

communicative (intransitive), or object-related (transitive) (Vanbellingen et al., 2010).

Gestures are compound actions that involve the coordinated interplay of several brain regions. The neural correlates of gesture processing have been extensively studied in fMRI experiments in healthy participants (Andric and Small, 2012; Yang et al., 2015). Planning, pantomime of tool use, and communicative gestures activate a left-hemispheric fronto-parieto-temporal network (Bohlhalter et al., 2009; Hermsdorfer et al., 2007; Johnson-Frey et al., 2005; Kroliczak and Frey, 2009; Niessen et al., 2014). Here, we refer to it as “gesture network”, that includes superior and inferior frontal as well as parietal areas and superior and middle temporal cortices (see Fig. 1) and also contributes to gesture recognition and interpretation. However, various gesture

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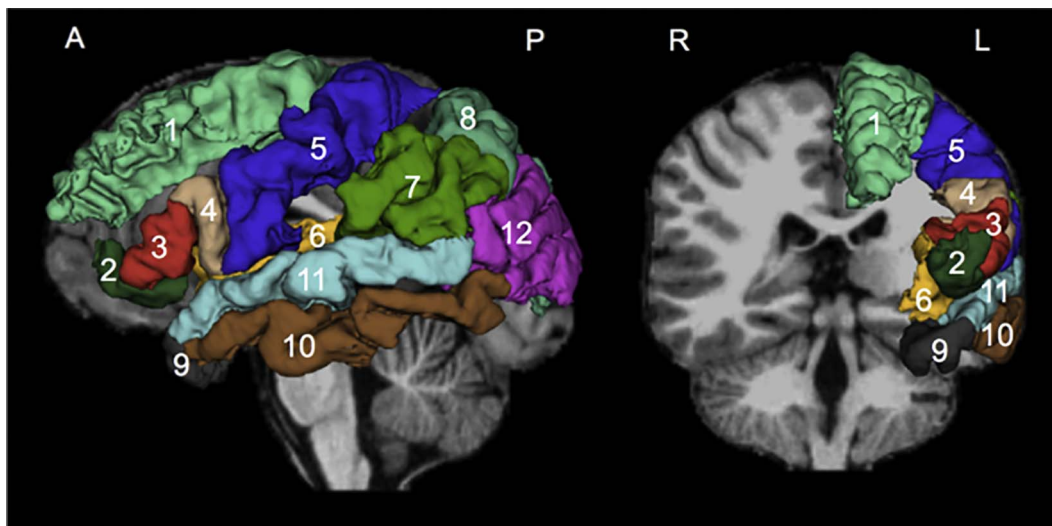


Fig. 1. Cortical regions of interest. The model is shown on a sagittal and coronal view and was created from a randomly selected case using the model maker module of Slicer 4.5. It is superimposed on the individual T1-weighted image. The regions of interest are based on FreeSurfer colors: superior frontal gyrus: 1, pars orbitalis of IFG: 2, pars triangularis of IFG: 3, pars opercularis of IFG: 4, precentral gyrus: 5, insula: 6, supramarginal gyrus: 7, superior parietal lobe: 8, temporal pole: 9, middle temporal gyrus: 10, superior temporal gyrus: 11, inferior parietal lobe: 12.

types seem to depend on different regions within this network. For example, pantomime of tool use activates some additional regions compared to imitation of meaningless gestures, such as the triangular part of the inferior frontal gyrus (IFG), the middle temporal gyrus (MTG), the supramarginal gyrus (SMG), and the intraparietal sulcus (Vry et al., 2015).

Studies investigating gesture recognition and comprehension have focused on varying contextual information, demonstrating distinct involvement of brain areas within the gesture network. For example, a meta-analysis suggests three different networks: a perceptual-motor network (including the premotor cortex as well as parietal and temporal regions), a semantic network (consisting of temporal and frontal regions) and a social emotive network (comprising of the IFG, the insula, and the putamen) (Yang et al., 2015).

Taken together, fMRI studies in healthy participants reveal associations to the left-lateralized gesture network for gesture production (planning or execution), recognition and comprehension.

Gesture is severely impaired in patients with apraxia following left hemispheric brain damage. Lesion mapping studies in apraxia have investigated pantomime of tool use and imitation of meaningless and meaningful gestures. Pantomime of tool use has been associated with the IFG, the occipito-temporal cortex, the parietal cortex, premotor and (pre-) central regions, and the insula (Goldenberg et al., 2007; Hoeren et al., 2014; Manuel et al., 2013; Weiss et al., 2016). Imitation of meaningful gestures can be attributed to the left IFG, the middle frontal gyrus, the premotor cortex, the SMG, and pre-central regions, whereas imitation of meaningless gestures relies predominantly on areas of the parietal lobe, for example on the angular gyrus, postcentral areas, and only small parts of the IFG (Goldenberg, 2009; Hoeren et al., 2014; Mengotti et al., 2013; Weiss et al., 2016).

To summarize, as in healthy participants, all apraxic deficits in brain-damaged patients can be attributed to damage in the left-lateralized gesture network (Buxbaum et al., 2014; Weiss et al., 2016), and different gesture types show associations with distinct regions within this network.

Patients with schizophrenia show both fewer and disturbed non-verbal behaviors (Lavelle et al., 2013; Lavelle et al., 2014). They have specific impairments in gesture recognition and production (imitation and pantomime) (Matthews et al., 2013; Park et al., 2008; Walther et al., 2015; Walther et al., 2013a; White et al., 2016). A high correlation between both tasks argues further for a generalized gesture

deficit (Walther et al., 2015). Gesture impairments are present at disease onset (Stegmayer et al., 2016b), are related to reduced social competence (Park et al., 2008), and gesture production and recognition predict functional outcome and negative symptoms after 6 months (Walther et al., 2016).

Studies of neural correlates of gesture production and recognition in schizophrenia have been sparse, but corroborate the results found in studies in healthy or brain-damaged participants. Patients with schizophrenia show aberrant brain function during gesture recognition, particularly with functional dysconnectivity between the superior temporal sulcus (STS) and the IFG when processing metaphoric gestures (Straube et al., 2014). Likewise, an fMRI study of gesture production found impaired gesture planning and execution to be linked to reduced activation in the dorsolateral prefrontal cortex (DLPFC) and increased activation of the inferior parietal lobe (IPL) (Stegmayer et al., 2017). Furthermore, patients with schizophrenia with gesture deficits had reduced grey matter volume in the left IFG, the right insula, the temporal pole (TP), and the anterior cingulate cortex, compared to healthy controls (Stegmayer et al., 2016a).

Taken together, the evidence suggests that structural and functional alterations in the gesture network contribute to the pathophysiology of impaired gesture performance in schizophrenia. So far, structural brain imaging analyses were limited to grey matter volume. However, schizophrenia is also associated with reduced cortical thickness (Goldman et al., 2009). Despite the fact that grey matter volume is a composite of cortical thickness and surface area, volume is more closely linked to surface area than to thickness (Winkler et al., 2010). Measurements of cortical thickness and surface area, in turn, are genetically and phenotypically independent (Panizzon et al., 2009; Winkler et al., 2010) and each shows a different pattern of development during adolescence, especially in regions important for social cognition (Vijayakumar et al., 2016). Furthermore, surface area seems to be more influenced by genetic factors than thickness, as most volumetric differences between first-degree relatives of schizophrenia and healthy controls are related to surface area rather than thickness (Goghari et al., 2007).

Even though cortical thinning in schizophrenia may be influenced by genetic effects, it is more associated to disease factors, as thinning occurs around the time of illness onset and is absent before onset (Sprooten et al., 2013). In addition, cortical thinning might not be progressive over the course of the illness, again arguing that pathological changes occur in a limited vulnerable phase around illness onset

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