Neuropsychologia 62 (2014) 202–208

Contents lists available at ScienceDirect

Neuropsychologia

journal homepage: www.elsevier.com/locate/neuropsychologia

The role of the superior temporal lobe in auditory false perceptions: A transcranial direct current stimulation study

Peter Moseley*, Charles Fernyhough, Amanda Ellison

Psychology Department, Durham University, South Road, Durham DH1 3LE, UK

ARTICLE INFO

Article history:
Received 27 May 2014
Received in revised form
4 July 2014
Accepted 28 July 2014
Available online 10 August 2014

Keywords:
Auditory verbal hallucinations
Signal detection
Superior temporal gyrus
tDCS

ABSTRACT

Neuroimaging has shown that a network of cortical areas, which includes the superior temporal gyrus, is active during auditory verbal hallucinations (AVHs). In the present study, healthy, non-hallucinating participants ($N=30$) completed an auditory signal detection task, in which participants were required to detect a voice in short bursts of white noise, with the variable of interest being the rate of false auditory verbal perceptions. This paradigm was coupled with transcranial direct current stimulation, a non-invasive brain stimulation technique, to test the involvement of the left posterior superior temporal gyrus in the creation of auditory false perceptions. The results showed that increasing the levels of excitability in this region led to a higher rate of 'false alarm' responses than when levels of excitability were decreased, with false alarm responses under a sham stimulation condition lying at a mid-point between anodal and cathodal stimulation conditions. There were also corresponding changes in signal detection parameters. These results are discussed in terms of prominent cognitive neuroscientific theories of AVHs, and potential future directions for research are outlined.

1. Introduction

Auditory verbal hallucinations (AVHs) are the experience of hearing a voice in the absence of any speaker. Although experienced by between 60% and 80% of people with a diagnosis of schizophrenia (Sartorius et al., 1986), the experience is also reported by approximately 1.5–3% of the general population (Beavan, Read & Cartwright, 2011; Tien, 1991). Neuroimaging findings relating to AVHs have been variable, but tend to show that AVHs coincide with activation in areas of the temporal lobe such as the superior temporal gyrus (STG), and frontal lobe areas such as the inferior frontal gyrus (IFG) and anterior cingulate cortex (ACC) (Allen et al., 2012).

The STG encompasses primary auditory cortex (PAC), as well as secondary auditory cortices such as Wernicke's area/the temporoparietal junction (TPJ), and the planum temporale (PT). Due to its importance in auditory processing, the role of the STG in AVHs (and associated cognitive mechanisms), particularly in the left hemisphere, has been extensively studied. For example, repeated measurements have shown tonic hyperactivity in left STG in patients with a diagnosis of schizophrenia who experience AVHs (Homan et al., 2013). Meta-analytic findings show that, in people who experience AVHs, PAC shows reduced activation to external auditory stimuli, but increased activation to internally generated information such as AVHs (Kompus, Westerhausen & Hugdahl, 2011). In addition, patients with a diagnosis of schizophrenia show reduced attenuation in auditory cortex when using inner speech (Simons et al., 2010), and reduced attenuation in somatosensory cortex when experiencing tactile stimulation (Shergill et al., 2014). These findings may reflect failures of internal forward models to successfully attenuate activity in response to self-produced actions (Ford & Mathalon, 2005), and/or biased attentional processes (Kompus et al., 2011). Finally, using offline repetitive transcranial magnetic stimulation (rTMS) or transcranial direct current stimulation (tDCS) to decrease activity in Wernicke's area (left posterior STG) as a treatment protocol has been shown to reduce the frequency of AVHs (Brunelin et al., 2012; Hoffman et al., 2013; Slotema, Blom, van Lutterveld, Hoek & Sommer, 2014), possibly due to effects on activity in other auditory cortical areas in the left STG (Kindler et al., 2013).

The above evidence suggests that the left pSTG plays a crucial role in the generation and/or experience of AVHs. This is in concordance with neuroimaging evidence suggesting that, among other areas, the superior temporal gyrus is active in the neurotypical brain during verbal self-monitoring (Allen et al., 2007; McGuire et al., 1995), and when a voice is falsely detected in white noise (Barkus, Stirling, Hopkins, McKie & Lewis, 2007), an error that people who experience AVHs make more often

* Corresponding author. Tel.: +44 191 334 3251.
E-mail address: peter.moseley@dur.ac.uk (P. Moseley).

http://dx.doi.org/10.1016/j.neuropsychologia.2014.07.032
0028-3932/

(Brookwell, Bentall & Varese, 2013). Nevertheless, the majority of available evidence regarding the role of the STG comes from fMRI and, due to the inherently correlational nature of neuroimaging, it is hard to draw conclusions about the causality of the role of this brain area in AVHs.

Whilst attempts to treat AVHs using neurostimulation of STG or TPJ are suggestive of the critical importance of these regions, and of surrounding auditory cortical areas (Kindler et al., 2013; Moseley, Fernyhough, & Ellison, 2013), it remains to be determined how neural activations relate to underlying cognitive mechanisms. For example, if the STG is causally involved in the genesis of AVHs, it should be possible to both increase and decrease AVH frequency by modulating the level of activity accordingly. Whilst this is clearly not possible in a clinical sample due to ethical issues, one previous approach has been to use a signal detection task, in which healthy participants are asked to listen to bursts of white noise, and respond whether they believe a voice is present (Bentall & Slade, 1985). This approach enables an analysis of 'correct' perceptions, as well as 'false' perceptions (or 'false alarm' responses).

Previous research suggests that individuals with a diagnosis of schizophrenia who hallucinate, and non-clinical participants who report more frequent hallucinatory experiences, are more likely to falsely perceive a voice in the noise (Barkus et al., 2011; Brookwell et al., 2013; Varese, Barkus & Bentall, 2011). These studies employ signal detection analysis, and suggest that this finding is due to a difference in response bias (i.e., how willing participants are to accept that an ambiguous stimuli is present) between hallucinators and non-hallucinators, rather than a change in sensitivity to the task (the ability to distinguish between signal and noise). This is important, as it implies that individuals who experience AVHs do not have a 'deficit' on the task, but instead simply exhibit a different style of responding. However, in a study by Vercammen, de Haan and Aleman (2008) using a similar paradigm, participants who experienced AVHs showed both a lower response bias *and* lowered sensitivity to the task, suggesting that the group differences may be more complex than a response bias. Of equal importance, false perceptions on this task are associated with high levels of activation in, among other areas, the STG (Barkus et al., 2007), even compared to correct perceptions of a voice in the noise. This suggests that high levels of activity in the STG might be associated with false alarm responses in this task, perhaps reflecting a tendency to misattribute internal, self-generated processes to an external source, as in AVHs.

Nevertheless, as discussed, evidence that activity in the STG is the *cause* of false alarm responses in a signal detection task is lacking. To address this, we utilised a form of non-invasive brain stimulation, transcranial direct current stimulation (tDCS), to modulate excitability in the left posterior STG (pSTG) of non-clinical, non-hallucinating participants. tDCS involves running a weak electrical current between two electrodes in contact with the participant's scalp, depolarising (anodal) or hyperpolarising (cathodal) membrane potentials of underlying neurons, resulting in a decrease in potential activity under the cathode and an increase in potential activity under the anode (Nitsche & Paulus, 2000). Furthermore, once stimulation has stopped, a reduction in GABA concentration under the anodal electrode and glutamate concentration under the cathodal electrode can be observed (Stagg & Nitsche, 2011), as well as short-lasting behavioural effects (Hummel & Cohen, 2006).

There are two main advantages of using non-clinical samples to study hallucination-like experiences:1) results are not confounded by anti-psychotic medication or additional symptoms of psychosis; 2) it would not be ethical to attempt to increase cortical excitability in a population which may already experience potentially pathological over-activity in superior temporal regions. Our objective was to test whether modulating excitability in left pSTG would lead to a change in the number of false perceptions that participants would make on an auditory signal detection task. Specifically, given findings that levels of activity in this region are related to both AVHs and false perceptions on auditory signal detection, we hypothesised that increasing the excitability of the posterior STG using anodal stimulation would lead to an increase in false alarms, whereas decreasing excitability using cathodal stimulation would lead to a decrease in the number of false alarms.

2. Materials and methods

2.1. Participants

The sample consisted of 30 right-handed participants (7 males, 23 females), aged 18–26 (M=20.6, SD=2.67). Participants were considered ineligible to take part if they reported any hearing problems, or any history of neurological or psychiatric disorder. All gave written informed consent in accordance with the Declaration of Helsinki, and ethical approval was provided by Durham University Ethics Committee. Participants were paid £15 for participation, and were naive to the aim of the study, simply being told that the study was investigating 'auditory perception'.

2.2. Signal detection task

The stimuli used in the signal detection task were similar to those used by Barkus et al. (2007, 2011), in which participants were asked to detect a voice stimulus embedded in white noise. The voice stimuli were identical to those used by Barkus et al.; a neutral, androgynous voice reading text from an instruction manual, which was segmented into 1-s clips. To set the volume levels in the task, we ran a small pilot study (N=8, none of whom took part in the main study), in which participants listened to a continuous burst of white noise, within which the voice clips were played, at a gradually ascending volume level. Participants were simply asked to respond with a button press when they heard a voice, and each pilot participant's threshold was defined as the point at which they heard three consecutive voices. For the main task, we then set the volume levels at the point at which 100%, 75%, 50% and 25% of participants in the pilot study consistently detected the voices (henceforth referred to as volume levels 4, 3, 2 and 1, respectively).

The stimuli for the main task consisted of 144 5-s bursts of white noise. During 80 bursts, a voice was present for the middle 1 s ('voice-present' trials). In the voice-present trials, voices were played at one of the four volume levels, which were kept constant across all participants (a requirement of the analysis, based on signal detection theory). The remaining 64 'voice-absent' trials consisted of the white noise, with no embedded voice. Each burst was followed by 3 s of silence, in which the participant was instructed to respond with a button press whether they believed a voice was present in the noise (yes/no). The stimuli were pseudo-randomly ordered, so that none of the five possible trial types (voice-absent, plus four voice-present volume levels) was presented more than three times in a row. Participants were not informed how often a voice was likely to be present, but were told that voices may be present at a variety of volumes. The task was separated into two blocks, each lasting 576 s, with a 5 min break between the blocks.

2.3. Transcranial direct current stimulation

Participants received 15 min of tDCS, using a Magstim Eldith DC stimulator. A 1.5 mA current was delivered to the first 14 participants, but for the final 16 participants this was decreased to 1 mA, after two participants experienced a mild headache following stimulation. The current was delivered through rubber electrodes placed in saline-soaked sponges, held in place by two rubber straps. One electrode (5×5 cm=25 cm^2) was positioned over the left posterior superior temporal gyrus (pSTG), over electrode site CP5 according to the EEG 10–20 system. This system ensures that the electrode montage is adjusted for differing head sizes between participants, and has been used previously to target the superior temporal gyrus, and more specifically, Wernicke's area (You, Kim, Chun, Jung & Park, 2011). The second electrode (5×7 cm=35 cm^2) was positioned above the right eye, as in other tDCS studies (Ball, Lane, Smith & Ellison, 2013; Ellison et al., 2014). A contralateral location was chosen as this is the most commonly used in the tDCS literature (Nitsche et al., 2008). The difference in electrode size ensured that the stimulation under the superior temporal electrode reached a higher current density than under the larger electrode. There were three stimulation conditions over the pSTG: anodal, cathodal and sham stimulation. Each participant received each type of stimulation in separate sessions, with each session separated, where possible, by 7 days (mean no. days between Sessions 1–2=7.47, SD=1.55, range=6–14; mean no. days between Sessions 2–3=7.80, SD=2.51, range=3–14). The order in which