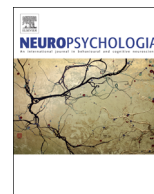




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Auditory lateralisation deficits in neglect patients

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ABSTRACT

Although visual deficits due to unilateral spatial neglect (USN) have been frequently described in the literature, fewer studies have been interested in directional hearing impairment in USN. The aim of this study was to explore sound lateralisation deficits in USN. Using a paradigm inspired by Tanaka et al. (1999), interaural time differences (ITD) were presented over headphones to give the illusion of a leftward or a rightward movement of sound. Participants were asked to respond "right" and "left" as soon as possible to indicate whether they heard the sound moving to the right or to the left side of the auditory space. We additionally adopted a single-case method to analyse the performance of 15 patients with right-hemisphere (RH) stroke and added two additional measures to underline sound lateralisation on the left side and on the right side. We included 15 patients with RH stroke (5 with a severe USN, 5 with a mild USN and 5 without USN) and 11 healthy age-matched participants. We expected to replicate findings of abnormal sound lateralisation in USN. However, although a sound lateralisation deficit was observed in USN, two different deficit profiles were identified. Namely, patients with a severe USN seemed to have left sound lateralisation impairment whereas patients with a mild USN seemed to be more influenced by a systematic bias in auditory representation with respect to body meridian axis (egocentric deviation). This latter profile was unexpected as sounds were manipulated with ITD and, thus, would not be perceived as coming from an external source of the head. Future studies should use this paradigm in order to better understand these two distinct profiles.

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1. Introduction

Unilateral spatial neglect (USN) is a common deficit following a right cerebral lesion. It corresponds to a failure to orient towards, respond to or report the detection of stimuli located in the contralesional side of space (Heilman and Valenstein, 1979). According to theoretical models, USN has been related to impairments of spatial representation (Bisiach and Luzzatti, 1978; Rizzolatti and Berti, 1993) and/or spatial attention (Heilman and Watson, 1977; Kinsbourne, 1987; Mesulam, 1998; Posner and Cohen, 1984;). Although USN-related spatial deficits have been frequently studied in the visual domain, fewer studies have investigated associated auditory deficits.

Three different auditory impairments have been described in

USN: auditory neglect (De Renzi et al., 1989), auditory extinction (De Renzi et al., 1984) and directional hearing deficits (Bisiach et al., 1984; Kerkhoff et al., 1999, 2012, 2013; Pavani et al., 2004, 2001; Tanaka et al., 1999; Vallar et al., 1995; Zimmer et al., 2003). In this study, we focused on directional hearing abilities, namely the ability to localise sound direction. Directional hearing is usually assessed by two kinds of tasks: a sound localisation task (in free or virtual fields) or a sound lateralisation task. Studies on sound localisation have revealed USN-related sound mislocalisations, predominantly on the contralesional side (Pavani et al., 2004, 2001).

Bisiach et al. (1984) were the first to introduce a new auditory task testing sound lateralisation, which can be assimilated to an auditory midline task. In this task, patients were asked to rotate a knob until the sound (received through headphones) was perceived to originate from a central position. The lateral position of sounds was generated using an Interaural Intensity Difference (IID) cue. The authors found shifts in sound lateralisation towards the ipsilesional side in 2 patients with USN. Both patients needed greater intensity to the left ear to perceive the sound as originating

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from a central position, which indicated that a sound had to be physically lateralised on the left side to be perceived in the middle by these patients. Tanaka et al. (1999) replicated this finding with sound lateralisation that was based on interaural time differences (ITDs). Forty-four patients with left or right-hemisphere lesions and 22 healthy participants were assessed. Participants were asked to move a lever to the right or to the left according to where they perceived the sound to originate from. This paradigm began with a “right trial”: the sound moved progressively to the right until the participant moved the lever to the right. The ITD was recorded each time there was pressure on the lever. Then, the sound moved from this right position to the left until the lever was pressed to the left. This corresponds to a “left trial”. This was repeated to obtain 10 measures of ITD for 10 consecutive stable trials: 5 right trials and 5 left trials. Two measures were taken into account: the amplitude of the ITDs and the midpoint. The amplitude was defined as the average amplitude between the left and right trials and corresponded to the ability to detect a sound shift from the centre. The midpoint of ITDs was defined as the average midpoint between the left and right trials. This study showed disturbances of sound lateralisation for right brain-damaged patients: these patients had an auditory midpoint deviated to the left side whereas no deviation was found for left brain-damaged patients and healthy controls. These disturbances correlated with the presence of a USN (10 USN patients were included in this study). According to the authors, this result was compatible with a rightward shift of perceived sound position. Zimmer et al. (2003) reassessed the ability of the auditory lateralisation with ITDs in patients with USN. Unexpectedly, they found half of their patients unable to perform the task. However, in this study, patients were tested in the acute phase (during the 2 first weeks after the stroke onset) and, thus, the USNs were very severe, which could explain their results. The other half of the patients showed, as in the paper of Tanaka et al. (1999), a leftward deviation of the auditory midpoint.

Other studies (Kerckhoff et al., 1999, 2012, 2013; Vallar et al., 1995) also used an “auditory midpoint” task. Contrary to the two previous studies based on ITDs and IIDs in which sounds were perceived as coming from the inside of the head, sounds were perceived as coming from an external source. This was achieved by presentation of external sounds in the free-field (Vallar et al., 1995) or within virtual auditory space using head-related transfer functions (HRTFs) over headphones (Kerckhoff et al., 1999, 2012, 2013). The results of these studies contrast with those of the two previous ones deficits (Bisiach et al., 1984; Tanaka et al., 1999) with a rightward deviation in the midpoint of ITD. In other words, patients with USN reported that sounds were aligned with their subjective midline when they were actually located on the right side of space. According to Pavani et al. (2004), these contradictory results can be explained by the perceived origin of the sounds. When participants were asked to compare an external sound to their head/body line, this task did not only rely on sound lateralisation processing but also on how the subject perceived their head/body midline to be oriented with the external environment. This means that in these tasks, there was an influence of an egocentric reference deviation. This was not the case in studies with ITD and IID, as sounds were perceived as coming from a source located inside the head. Therefore, these tasks appear to be better adapted to assess sound lateralisation deficits in USN.

However, the explanation for the deviation of the auditory midpoint in the study of Tanaka et al. (1999) was quite succinct. The authors linked the deviation of the auditory midpoint to the deviation that could be underlined in visual tasks, which could be influenced by an egocentric deviation. This meant that the authors did not directly link their results to sound lateralisation impairment, contrary to Pavani et al. (2004). Moreover, only two

measures were analysed: midpoint and amplitude. These two chosen measures could not fully capture the complexity of the observed deficits in patients with USN, and could not answer to a potential influence of egocentric deviation. As an example, a global leftward shift in auditory midpoint could be a result of either a global leftward shift in the lateralisation of sounds in both the left and right hemifields, or a leftward shift in the lateralisation of sounds in only one of the hemifields. These two explanations should be questioned in the specific case of USN. Moreover, no individual information was provided for the patients and the severity of their USN was not taken into account nor documented.

In our study, the aim was to replicate the results found by Tanaka et al. (1999) with 15 right-lesion patients (10 with USN and 5 without) with a paradigm inspired by their own. Taking this one step further, we also compared each patient to a control group of 11 healthy participants for the amplitude and the midpoint but also for two additional measures: left trials mean and right trials mean (single-cases analyses).

We hypothesised that, according to Tanaka et al. (1999), patients with USN would have a leftward deviation of the auditory midpoint. According to Pavani et al. (2004), this abnormal midpoint would only be due to abnormal left trials, underlying difficulties to lateralise sounds only for the left side of space. Deviations in both left and right trials could rather underline a shift of the subjective head/body midline, which would normally not be found here since the sounds were perceived as inside the head (Pavani et al., 2004). That is to say we hypothesised that, contrary to Tanaka et al. (1999), the midpoint measure did not correspond to the subjective middle of auditory space and did not make sense contrary to the analyses of left trials and right trials means. Additionally, we hypothesised that the severity of visual USN would impact the degree of difficulties in lateralising left-side sounds. Finally, we expected abnormal amplitudes for patients with USN but also for some right brain-damaged patients without USN, as in Tanaka et al. (1999).

2. Material and methods

2.1. Participants

Fifteen patients were recruited from Reeducation and Rehabilitation centres in Northern France. Each patient had a right cerebral lesion confirmed by an MRI and was able to understand and perform the task. Their audiogram can be considered as normal: each patient was able to detect sounds between 125 Hz and 6000 Hz at 50 dB presented either to the right or the left ear. Five patients had a USN that can be qualified as severe, 5 had a mild USN syndrome and the last 5 had no USN (see Table 1). In order to evaluate the severity of USN on the evaluation day, a line bisection task and a Bell test (extracted from the “Batterie d’Evaluation de la Négligence spatiale” (BEN), Azouvi et al., 2006) were performed by all participants. Patients who massively failed both tasks were considered as having a severe USN. If only one test was mildly failed, patients were considered as having a mild USN. The 5 patients without USN had normal scores on these two tasks and had never presented signs of USN but had a right cerebral lesion. Patients were tested during the first 6 months after the cerebral lesion onset, except patient 1 (one and a half years after the stroke) and patient 14 (one year after two cerebral embolisations).

The control subjects included 11 healthy volunteers (8 women and 3 men) with a mean age of 53.2 (SD: ± 4.4). They had no neurological or auditory deficits.

The four groups did not differ in terms of age ($p=0.298$, Kruskal-Wallis test).

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