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Visual change detection recruits auditory cortices in early deafness

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ABSTRACT

Although cross-modal recruitment of early sensory areas in deafness and blindness is well established, the constraints and limits of these plastic changes remain to be understood. In the case of human deafness, for instance, it is known that visual, tactile or visuo-tactile stimuli can elicit a response within the auditory cortices. Nonetheless, both the timing of these evoked responses and the functional contribution of cross-modally recruited areas remain to be ascertained. In the present study, we examined to what extent auditory cortices of deaf humans participate in high-order visual processes, such as visual change detection. By measuring visual ERPs, in particular the visual MisMatch Negativity (vMMN), and performing source localization, we show that individuals with early deafness (N = 12) recruit the auditory cortices when a change in motion direction during shape deformation occurs in a continuous visual motion stream. Remarkably this "auditory" response for visual events emerged with the same timing as the visual MMN in hearing controls (N = 12), between 150 and 300 ms after the visual change. Furthermore, the recruitment of auditory cortices for visual change detection in early deaf was paired with a reduction of response within the visual system, indicating a shift from visual to auditory cortices of part of the computational process. The present study suggests that the deafened auditory cortices participate at extracting and storing the visual information and at comparing on-line the upcoming visual events, thus indicating that cross-modally recruited auditory cortices can reach this level of computation.

Introduction

The textbook notion that responses in modality-specific cortices are strictly defined by their peripheral afferents has undergone a substantial revision over the last decades. Studies in animals (e.g. Lakatos et al., 2007; Wallace and Stein, 2000) and humans (e.g. Giard and Peronnet, 1999; Molhom et al., 2002) indicate that the first steps of multisensory integration take place at very early levels of processing, already within primary and secondary sensory cortices (for a review see Ghazanfar and Schroeder, 2006). Remarkable evidence of sensory cortices interdependence is their potential to redefine their responses in the context of deafness or blindness (Dormal and Collignon, 2011; Merabet and Pascual-Leone, 2009; Voss and Zatorre, 2012). In early and congenitally deaf humans, auditory cortices can respond to visual (Fine et al., 2005; Finney et al., 2001, 2003; Karns et al., 2012) or tactile stimuli (Auer et al., 2007; Karns et al., 2012; Levanen et al., 1998), as well as bimodal combinations of visual and somatosensory inputs (Karns et al., 2012). Similarly, in early and congenitally blind individuals the deprived visual cortices proved responsive in a variety of auditory (Bedny et al., 2010; Collignon et al., 2009, 2011; Poirier et al., 2006; Wolbers et al., 2011) and tactile tasks (Cohen et al., 1997; Sadato et al., 1996, 2002; Stilla et al., 2008); (but see also Sathian and Stilla, 2010 for negative findings).

While cross-modal recruitment of early sensory areas in deafness and blindness is now well established, the precise constraints and limits of these plastic changes remain to be ascertained. One emerging view is that recruitment of brain circuits to serve a different sensory modality (e.g., vision instead of audition in the brain of deaf people) occurs within the wired computational functions specific to that brain circuit. As originally proposed by Pascual-Leone and Hamilton (2001), these brain circuits are "metamodal brain centers that perform particular computational operations without specific reference to type of sensory input […] It is these processes, and hence the cortical modules performing them, that are subsequently exploited and shaped by the demands of the sensory modalities that require them." (Pascual-Leone and Hamilton, 2001, p.17). While this view has now received consistent support in blindness (Collignon et al., 2011; Reich et al., 2011; but see Bedny et al., 2011) in deafness only studies on deaf animals converge toward

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a similar conclusion (e.g. Lomber et al., 2010; Meredith et al., 2011; Wong et al., 2013). In deaf humans, no study to date has shown the existence of metamodal brain centers, which could retain the specified function for an intact modality (except for studies on language (Emmorey et al., 2003, 2007; Mayberry et al., 2011) (see MacSweeney et al., 2008 for a review)). Parallel to the evaluation of the functional specialization (instead of modality-specialization) of early sensory areas, a relevant topic of investigation concerns which level of processing cross-modally recruited areas can achieve. For instance, while in blind adults visual cortices proved responsive to higher-order levels of information processing already at the level of V1 (e.g. short term memory, Bedny et al., 2012; Likova, 2012), in deaf adults, the activation of auditory cortices has been reported only for tactile stimulation and for visual moving stimuli on which very easy tasks, if any, were required (e.g. Fine et al., 2005; Karns et al., 2012; Levanen et al., 1998).

In the present study we examined to what extent auditory cortices of deaf humans participate in processing higher-order features of visual information, namely, the detection of changes in a visual object. To this aim we exploited a well-known electrophysiological marker of sensory expectancies, i.e., the MisMatch Negativity response (MMN Näätänen et al., 1978; Tiitinen et al., 1994). First discovered in the auditory modality, this marker reveals an automatic process of change-detection, in which the auditory system uses the preceding stimuli and their regularity as reference to detect any change in the auditory scene, even when the auditory stream is unattended (for reviews see Näätänen et al., 2001, 2010). In terms of the biological relevance, the MMN is believed to initiate an involuntary capture of attention toward an abrupt event that differs in some aspects from the previous regular stream. Its relevance is so high that it appears at early stages of ontogenesis as in newborns (Carral et al., 2005). In humans and other species, the MMN is mainly generated in auditory cortices (for a review see Alho, 1995); note that some studies showed also the participation of a frontal component (Giard et al., 1990); (reviewed in Deouell, 2007). Studies on auditory MMN set the basis for the notion of a primitive intelligence in the auditory system, dedicated to alerting and orienting in the environment (but see also May and Tiitinen, 2010 for an alternative account to explain MMN).

Interestingly, and most importantly for the present work, a visual counterpart of the auditory MMN, i.e., a visual MMN (from now on termed vMMN), sharing many of the properties characterizing the auditory MMN, has also been extensively documented in the last decade (for a review see Czigler, 2013; Kimura et al., 2011; Pazo-Alvarez et al., 2003). Similarly to the auditory MMN, to be able to investigate the properties of the vMMN, it is important that both the regularity and its violation remain outside the focus of attention (i.e., participants are instructed to focus their visual attention on a different task). As for the classic MMN, vMMN has been documented for violations of visual regularities in several elementary visual features, including motion direction (Kremláček et al., 2006; Pazo-Alvarez et al., 2004), forms in motion (Besle et al., 2005), stimulus orientation (Astikainen et al., 2008), spatial frequency (Maekawa et al., 2005) and color (Czigler et al., 2004). In addition, vMMN has been revealed in response to higher order visual violations, such as the conjunction of visual features like color and moving direction (Winkler et al., 2005), the violation of temporal regularities (Czigler et al., 2006b), the omission of visual stimuli within an otherwise regular stream (Czigler et al., 2006a), the appearance of an irregular change in the visual stream instead of a regular one (Czigler et al., 2006a), changes in facial expressions (Astikainen and Hietanen, 2009; Chang et al., 2010; Gayle et al., 2012; Stefanics et al., 2012; Susac et al., 2010; Zhao and Li, 2006), changes in symmetry (Kecskés-Kovács et al., 2013b), presentation of left vs. right hand stimuli (Stefanics and Czigler, 2012) or changes in the gender of a face (Kecskés-Kovács et al., 2013a). Differently from the auditory MMN, the vMMN is usually recorded over occipital electrodes or parieto-occipital electrodes (Maekawa et al., 2005) in the 200–400 ms latency range.

Most interestingly for the present study, the interaction between visual and auditory MMNs has also been documented. Using MEG, Besle et al. (2007) found that stimuli deviating in auditory and visual features from an audio-visual regularity elicited a significant audio-visual MMN (avMMN), i.e. an avMMN triggered only by the violation of the conjunction of auditory and visual features. The presence of this avMMN (Besle et al., 2007) demonstrates that in hearing individuals the visual and the auditory change-detection systems interact.

In the present study, we compared vMMN in a group of adults with bilateral early deafness and a group of age-matched hearing controls, using a visual oddball paradigm with the stimuli originally developed by Giard and Peronnet (1999). The rationale for adopting these stimuli was twofold. First, they consist in a central visual stimulus (a circle) that constantly moves changing its shape (transient deformation of the circle into a horizontal or a vertical ellipse). Visual motion processing has been repeatedly shown to recruit the auditory areas of early deaf individuals compared to hearing controls (Fine et al., 2005; Finney et al., 2001, 2003), and behavioral studies have documented enhanced discrimination abilities in deaf compared to hearing individuals specifically when using visual moving stimuli (e.g. Hauthal et al., 2013a; Neville and Lawson, 1987). Second, Besle et al. (2005, 2007, 2013) in their work on audio-visual MMN used precisely the stimuli developed by Giard and Peronnet (1999). Thus, using these stimuli should provide a useful comparison for any response coming from the temporal lobe in deaf individuals in our visual-only task.

Predictions were as follows. As auditory cortices cross-modally reorganize to respond to visual input in deaf people, auditory areas could participate in vMMN generation. This would imply that auditory cortices of deaf people not only contribute to the processing of visual motion as previously suggested by fMRI and MEG research (Fine et al., 2005; Finney et al., 2001, 2003), but they could also contribute to higher level motion processing, namely the detection of visual changes (in motion direction or shape) in the environment. This result would suggest that, following auditory deprivation, the deafferented auditory cortices are still capable of processing change-detection information, albeit conveyed through vision instead of audition. Moreover, given the high temporal resolution of EEG, a result in this direction could provide important information on when in time the visual and the auditory contributions to the processing of change-detection take place in the deaf population. This result could therefore better define the role of the recruitment of auditory cortices in this process. Alternatively, if visual change detection in deaf people remains a visually specific function, vMMN generators should be comparable between deaf and hearing participants.

Finally, as a secondary aim, we took advantage of the large number of repeated standard stimuli to examine to what extent visual motion (or the change in form resulting from motion) recruits auditory cortices in deaf people. A role of auditory cortices in the processing of moving stimuli in deaf adults has been previously documented using fMRI, but the temporal dynamics of this recruitment could not be fully ascertained (Fine et al., 2005; Finney et al., 2001). The only available study using MEG (Finney et al., 2003) did not explore the temporal dynamic of the visual evoked response in auditory cortices. Interestingly, one recent EEG study that compared cochlear implant recipients to hearing controls (Sandmann et al., 2012), suggested a recruitment of auditory cortices in response to visual moving stimuli already at the latency of the visual P1 component (~110 ms) (see also Doucet et al., 2006). Our secondary aim was thus to characterize the neural sources and temporal dynamics of the responses to the standard stimuli in early deaf adults compared with hearing controls. Innovatively in respect to previous studies, our moving stimuli were continuously displayed on the screen and thus motion onset was dissociated from stimulus onset.

Methods

Participants

Twelve profoundly deaf individuals (mean age = 35.9 years, SE = 3, range 20–51 years old; mean years of education = 19, SE = 0.86; 7

female; see Table 1) were recruited at the National Association for Deaf (Ente Nazionale per la protezione e assistenza dei Sordi, Trento, Italy). All deaf participants had bilateral profound hearing loss (>90 dB in the better ear), and reported to have acquired deafness within the first year of life (8 had congenital deafness). None became deaf due to systemic causes that could also affect vision, and none received a cochlear implant. All deaf individuals were proficient sign-language users by self-report: 5 learned Italian Sign Language (LIS) as first language, the other seven learned Italian as first language and later learned LIS as a second language at various ages (mean 13.5 years old). Twelve hearing controls (mean age = 31.6 years, SE = 2.2, range 23–45 years old; mean years of education = 21.9, SE = 1.3; 6 females) were also recruited to take part in the study. No hearing participant was familiar with sign language. All participants had normal or corrected-to-normal vision and were right-handed by self-report.

The study was approved by the ethical committee at the University of Trento (Italy), and written informed consent was obtained from all participants prior to testing. Participants received a payoff of 18 euros for participating in the study.

Stimuli

Visual stimuli consisted in the transient deformation of a circle into an ellipse, either in the horizontal or in the vertical direction (see Giard and Peronnet, 1999 for a detailed description). The circle had a diameter of 4.55 cm and was displayed on a video screen (75 Hz refresh rate) placed 130 cm in front of the subjects' eyes, subtending a visual angle of 2°. The amount of deformation in either direction relative to the diameter of the circle was 33% and lasted 107 ms (same amount of time to recover the original shape). Between each deformation the circle remained present on the screen. The SOA was 587 ms. A cross (subtending 0.2° of visual angle) was presented at the center of the circle and served as a fixation point as well as a target (see Fig. 1). The cross disappeared briefly (133 ms), at unpredictable moments during each block. Participants were instructed to fixate the central cross and press a button as fast as possible every time it disappeared (in 17% of trials). They were instead instructed to ignore the circle and its deformations. These visual stimuli matched exactly those adopted in several previous studies (Fort et al., 2002a,b, 2005; Giard and Peronnet, 1999) and particularly in the studies by Besle and colleagues which examined audio-visual regularities and mismatches (Besle et al., 2005, 2007, 2013).

The experiment comprised 8 experimental blocks. In each block, the circle deformed in one direction (either horizontal or vertical) in 76% of the trials (standard) and it deformed in the opposite direction in the remaining 14% of trials (deviant). Visual MMN was estimated in the difference between ERPs to the deviants and ERPs to the standards. Importantly, to ensure that the obtained vMMN could not be attributed to physical differences between the standard and deviant stimuli, the visual features of the standard and the deviant were swapped for half of the experimental blocks. Thus, the vMMN represents uniquely the neural response to the visual change (that can be perceived as a change in visual motion direction or in the shape of the stimulus). The appearance of the deviant stimulus was pseudo-randomized: deviants were always preceded by at least three standard stimuli, and they could never follow a trial including the target nor appear in conjunction of it. As a consequence, the appearance of a deviant was completely separated from the task relevant stimuli (as in Besle et al., 2005, 2007, 2013).

Event-related potentials

The EEG was recorded (analog bandwidth: 0.1–200 Hz, sampling rate: 1 kHz) from 34 electrodes using the International 10–20 System extended montage (documentation in http://www.easycap.de). Standard 10–20 sites were Fp1, Fp2, F7, F3, Fz, F4, F8, T7, C3, Cz, C4, T8, T5 (P7), P3, Pz, P4, T6 (P8), O1 and O2. Additional intermediate sites were FC5, FC1, FC2, FC6, TP9 (M1), CP5, CP1, CP2, CP6, TP10 (M2), PO3, PO4, PO9, Iz and PO10. All scalp channels were referenced to the nose. Horizontal eye-movements were monitored with a bipolar montage from electrodes at left and right outer canthi. First, the EEG signals were digitally filtered (low-pass filter with a 40-Hz cut-off). The blinks and eye movement related artifacts were eliminated using an Independent Component Analysis (ICA (Comon, 1994; Mennes et al., 2010); runica version, implemented on EEGLab (Delorme and Makeig, 2004); running in MATLAB, http://www.mathworks.com/). The ICA was conducted for each participant on the whole data recording decomposing it into 16 components. To guide the selection of the components to remove we visually inspected the dynamic of each component, its distribution on the scalp, its distribution across trials and its power spectrum. Only components clearly showing blinks, eye related activity and heart beat (when detectable) were removed. Subsequently, trials with signals exceeding ± 80 μV at any electrode were excluded from averaging. In addition, we excluded from the analysis the responses to all standards following a deviant, or following the fixation cross disappearance, and all trials including the participant's response.

ERPs to standard and deviant signals were averaged over a period of 800 ms including 200 ms pre-stimulus. EEG epochs were corrected relative to a [−100, 0 ms] pre-stimulus baseline with respect to both standard and deviant signal onsets. Scalp potential maps and scalp current densities were generated using spherical spline interpolation (Perrin et al., 1987, 1989). All EEG data were analyzed with the ELAN-pack software developed at the Brain Dynamics and Cognition team in Lyon (Aguera et al., 2011), together with EEGLab (Delorme and Makeig, 2004) and sLORETA (Pascual-Marqui, 2002).

Statistical approach

To minimize the influence of individual differences in topographies as well as statistical multiple comparisons the ERPs to standard stimuli

Table 1
Deaf individuals' characteristics.

Participant	Age (years)	Age at deafness onset (months)	Hereditary	Etiology	Age sign language acquisition (years)	Education (years)
1	20	8/9	Yes	Genetic	Birth	22
2	51	Congenital	Yes	Genetic	Birth	14
3	22	Congenital	Yes	Genetic	2	21
4	45	Congenital	No	Genetic	18	18
5	42	Congenital	Unknown	Unknown	Unknown	18
6	36	Congenital	No	Unknown	12	20
7	43	3	Unknown	Ototoxic drugs	6	25
8	44	Congenital	No	Unknown	10	18
9	40	Congenital	Yes	Genetic	Birth	17
10	32	3/4	Yes	Unknown	Unknown	18
11	28	Congenital	Yes	Genetic	Birth	15
12	20	12	No	Unknown	14	18

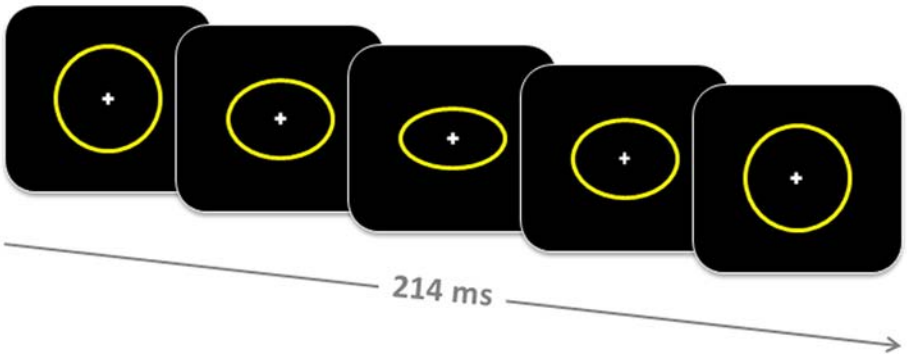

Fig. 1. Example of the deformation of the circle into an ellipse in the horizontal direction.

and the vMMN (measured in the difference between the ERPs to deviant stimuli and ERPs to standards) were grouped into 3 clusters for each hemisphere (L and R for left and right) that comprised three electrodes each (see Fig. 2a) covering the frontal, central and posterior areas of the scalp. The clusters were defined as follows: LF (F3, FC1, FC5) and RF (F4, FC2, FC6) frontal clusters, LC (C3, CP5, CP1) and RC (C4, CP6, CP2) central clusters, LP (PO3, P7, O1) and RP (PO4, P8, O2) posterior clusters.

To analyze the responses to standard stimuli, we calculated the mean amplitude of each ERP cluster within a time window of ± 20 ms around the P1 and N1 peak latencies measured from the grand-averages across the two groups of subjects. When appropriate, the time courses of a component dynamic were compared between groups. To this aim the signal was averaged within running 10/20 ms time windows that covered a specific ERP deflection. Running 10-ms windows were used to evaluate the P1 wave (lasting approximately 60 ms to include both group waves) while 20-ms windows were used for the MMN (lasting approximately 140 ms to include both groups waves). The main analyses on standard ERPs were conducted on the mean amplitudes of the early P1 and N1 components, using mixed ANOVAs with clusters of electrodes (F, L, P) and hemisphere (left, right) as within-participants factors and group (deaf individuals, hearing controls) as between-participants factor. When appropriate the Fisher test was used for post-hoc comparisons.

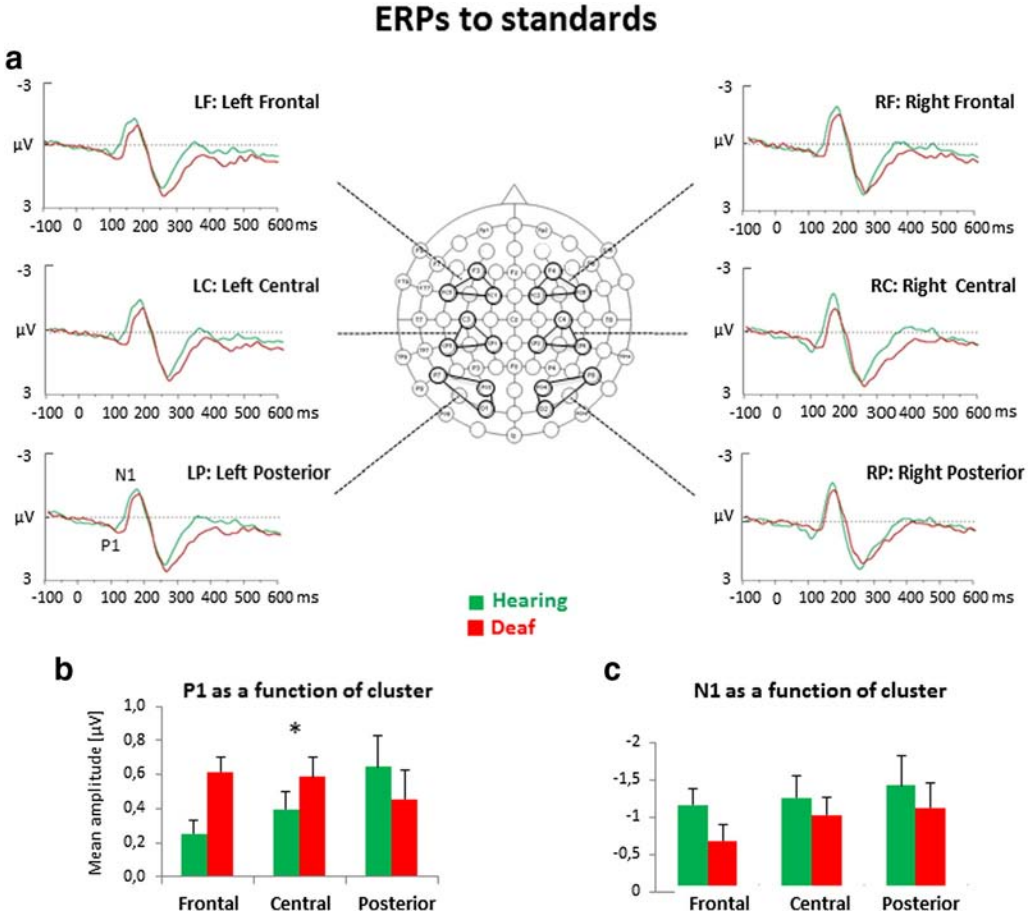

Fig. 2. (a) ERPs to standard stimuli (onset of circle deformation, averaged for vertical and horizontal motions) at each cluster and hemisphere. (b) P1 mean amplitude for each group as a function of each cluster. Deaf individuals display a more frontally distributed P1. (c) N1 mean amplitude for each group as a function of each cluster. No between groups differences emerged.

Dipole modeling

To further compare the vMMNs between hearing controls and deaf participants, their intracerebral sources were modeled using a 3-concentric spherical shell model of the head and two symmetrical equivalent current dipoles (ECDs) with free orientations for representing the brain generators (Elan package). Although there are probably multiple sources active during the vMMN process, the 2-dipole model allowed us to estimate an "average" vMMN location for each group, with the assumption that any difference in vMMN activities between the two subjects' groups should result in different locations of the dipoles in the two groups. In addition the dipole modeling procedure allowed to compare its solutions with sLORETA source estimates (described below), and thus validate our findings with alternative methodologies.

Standardized low-resolution brain electromagnetic tomography

Scalp-recorded visual evoked potential (VEP) amplitudes were evaluated by sLORETA, to compute the cortical three-dimensional distribution of current density (Pascual-Marqui, 2002). sLORETA method computes the cortical distribution of neuronal electrical activity from scalp-recorded electric potentials, thus not requiring assumptions on the number of existing dipoles nor on their characteristics. This method leads to rather precise localization (Sekihara et al., 2005), although it has low spatial resolution. A recent study (Plummer et al., 2010) showed solid experimental validation for sLORETA based on intracranial recordings in epilepsy patients and showing that sLORETA provided the lowest localization error among other methods. In addition, considered the aim of the present work, a recent study by Sandmann et al. (2012) used successfully sLORETA technique to show that cochlear implanted participants displayed visual evoked responses in auditory areas (BA 41 and 42) already at the level of the visual P1 (98–108 ms) as well as decreased response over visual secondary visual cortex (BA 18) around the same latency.

Source estimates (of averaged reference data) were baseline-corrected (−100 to 0 ms), and averaged in adjacent 20-ms time windows over the time interval corresponding to the scalp-recorded visual P1 (two 20-ms windows around its peak) and over the vMMN (seven consecutive 20-ms time-windows spanning the entire 140 ms window of interest defined as starting 40 ms before the vMMN peak in hearing controls and ending 40 ms after the vMMN peak in deaf individuals, see later). These time-windows are the same as the one used in the scalp-level analysis described above. Two regions of interest (ROIs) were then defined in each hemisphere to analyze the cortical activation in response to standard events and the vMMN. A first anatomical region was defined as a combination of the visual Brodmann areas (BA) 18 and 19, a second region was the combination of the core auditory cortex (BA41) and secondary auditory cortex (BA42), in each hemisphere. The selection of these two regions of interest was based on two considerations. On the one hand, there is now experimental evidence that the auditory cortices can be activated for visual processing in deaf individuals (Fine et al., 2005; Finney et al., 2001; Karns et al., 2012). On the other hand, recent evidence confirming enhanced activation of auditory cortices during visual processing in deaf individuals (see Hauthal et al., 2013b for a trend), and in cochlear implant patients (Sandmann et al., 2012), also suggested a concurrent reduction of response within visual extrastriate regions, possibly indicating a shift of processing load from visual to auditory areas. For statistical analysis, the current density values of each region of interest (visual and auditory) were subjected to repeated-measures ANOVAs, with time window (7 time windows in case of MMNv), hemisphere (left and right) and ROI (visual and auditory) as within-subjects factors, and group (deaf individuals, hearing controls) as between-subjects factor.

The sLORETA software package was also used to perform a sub-set of the statistical analyses directly comparing source estimates at specific time windows around P1 and vMMN average peak, between groups. The methodology used is non-parametric. It is based on estimating, via randomization, the empirical probability distribution for the max-statistic (e.g., the maximum of a t statistic), under the null hypothesis. This methodology corrects for multiple testing (i.e., for the collection of tests performed for all electrodes, and for all time samples). Due to the non-parametric nature of the method, its validity does not need to rely on any assumption of Gaussianity (Nichols and Holmes, 2001). The test was performed using 5000 permutations.

Results

Standard ERPs: P1 and N1

In hearing controls and deaf participants, standard stimuli elicited expected visual ERP components over the posterior regions of the scalp. Differences between deaf and hearing participants emerged in the scalp distribution, duration, and dynamic of the early visual P1 component (see also Bottari et al., 2011).

For the P1 component analysis, we calculated for each participant the mean amplitude at each cluster (LF, LC, LP, RF, RC and RP) within a ±20-ms time window centered on the averaged P1 peak latency extracted from the grand average over the two groups (104 ms, 84–123 ms). In hearing controls, the P1 peak had a value of 0.93 μV at cluster LP (posterior left), 97 ms after the standard movement onset. In deaf individuals, the P1 peak had a value of 0.75 μV at cluster LP at 111 ms. For each participant, the mean amplitudes of each cluster in the 84–123 time window were entered in a mixed ANOVA with hemisphere (left, right) and cluster (F, C, P) as within-participants factors and group (deaf participants, hearing controls) as between-participants factor. The interaction between the cluster and group factors was significant ($F(2, 44) = 3.7$, $p = 0.03$), showing that while for hearing controls the P1 was mainly distributed over the central and posterior scalp clusters, deaf individuals displayed a more anteriorly distributed P1 component (see Figs. 2a, b; and 3a SCD maps). Post-hoc comparisons on hearing controls showed that the P1 recorded at posterior clusters did not differ from the activity at central clusters ($p = 0.1$), but significantly differed from the activity recorded at frontal clusters ($p = 0.01$). By contrast, deaf individuals showed comparable activity across all clusters ($p > 0.3$, see Fig. 2b). The analysis also revealed a significant main effect of hemisphere ($F(1, 22) = 4.1$, $p = 0.05$) showing that the P1 was more pronounced over the left compared to the right hemiscalp regardless of group (0.5 μV SE = 0.1 and 0.4 μV SE = 0.1, respectively). No other main effects or interactions were significant (all F-values < 1.6).

The visual inspection of the P1 component suggested that the deaf group displayed a prolonged P1 compared to the hearing control group. To test this difference in dynamics, we measured the mean amplitude for each participant across all clusters in 6 different time windows of 10 ms each, between 74 and 134 ms after the standard movement onset (P1 averaged peak 104 ms). We entered this measure into a mixed ANOVA with time window (6 bins of 10 ms) as within-participants factor and group as between-participants factor. The analysis revealed a main effect of time window ($F(5,110) = 2.7$, $p < 0.03$), and most importantly a significant interaction between the time window and group factors ($F(5,110) = 4.1$, $p = 0.002$, see Figs. 3 and 4). Hearing controls reached their P1 peak within the time window 3 (94–104 ms) and then the mean amplitude decreased (post-hoc analyses showed that both time windows 5 and 6 displayed smaller amplitudes compared to time window 3, while time window 6 showed smaller amplitude than all other time windows: all $p < 0.05$). Conversely, in deaf individuals, the maximal mean amplitude of P1 was reached within the 114–124 ms time window (window 5), with significantly larger amplitude than time window 2 ($p < 0.05$). These results overall suggest that the P1 lasts longer and its maximal amplitude is reached later in deaf individuals compared to hearing controls. No other main effects or interactions reached significance (all F-values < 1).

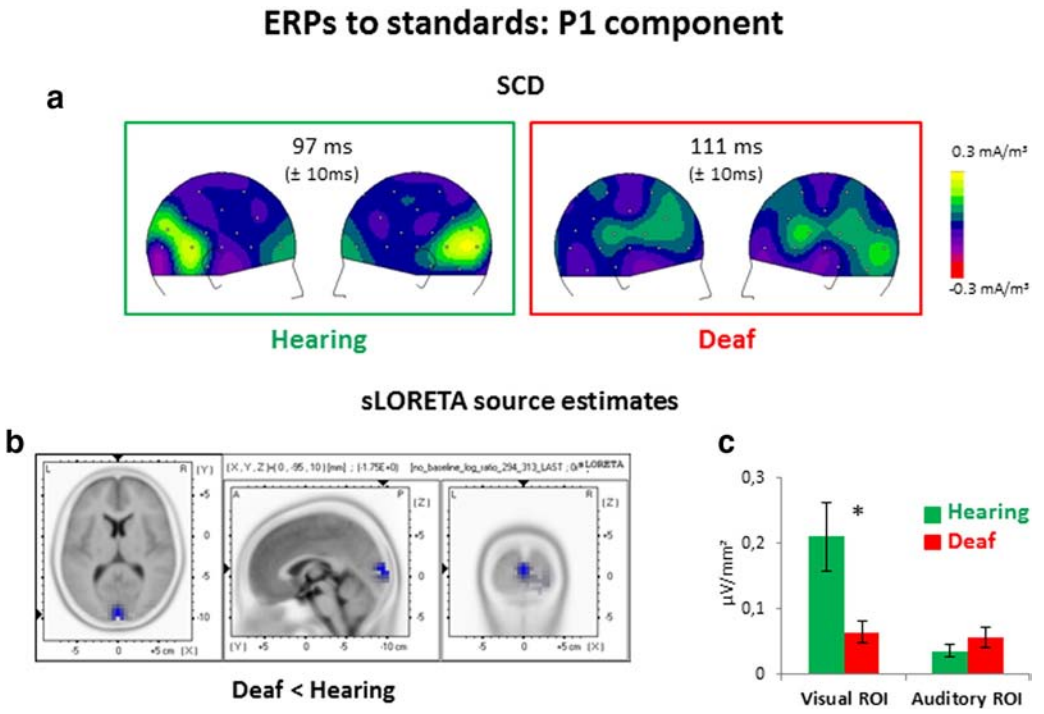

Fig. 3. (a) SCD distribution of the P1 component over a ±10 ms window around its peak latency in hearing controls and deaf participants. (b) Results of the comparison of the source estimates between the two groups on a 20-ms time window (94–114 ms) centered on the average of the two groups' P1 peak (104 ms). Deaf individuals showed reduced activity at the occipital cortex compared to hearing controls. (c) Source estimates within visual (BA 18, 19) and auditory ROIs (BA 41, 42). Hearing controls display enhanced response within the visual ROI compared to deaf individuals.

The N1 component was analyzed with the same rationale as the P1. First, we calculated for each participant the mean amplitude at each cluster (LF, LC, LP, RF, RC and RP) within a ± 20 ms window centered on the N1 peak latency averaged across all participants (166 ms, 146-186 ms). The N1 peak in hearing controls had a value of −1.8 μV at cluster RP (posterior right), 163 ms after the standard movement onset. In deaf individuals N1 peak had a value of −1.44 μV at RP at 170 ms. The mean amplitudes for each participant at each cluster in the 146–186 ms time

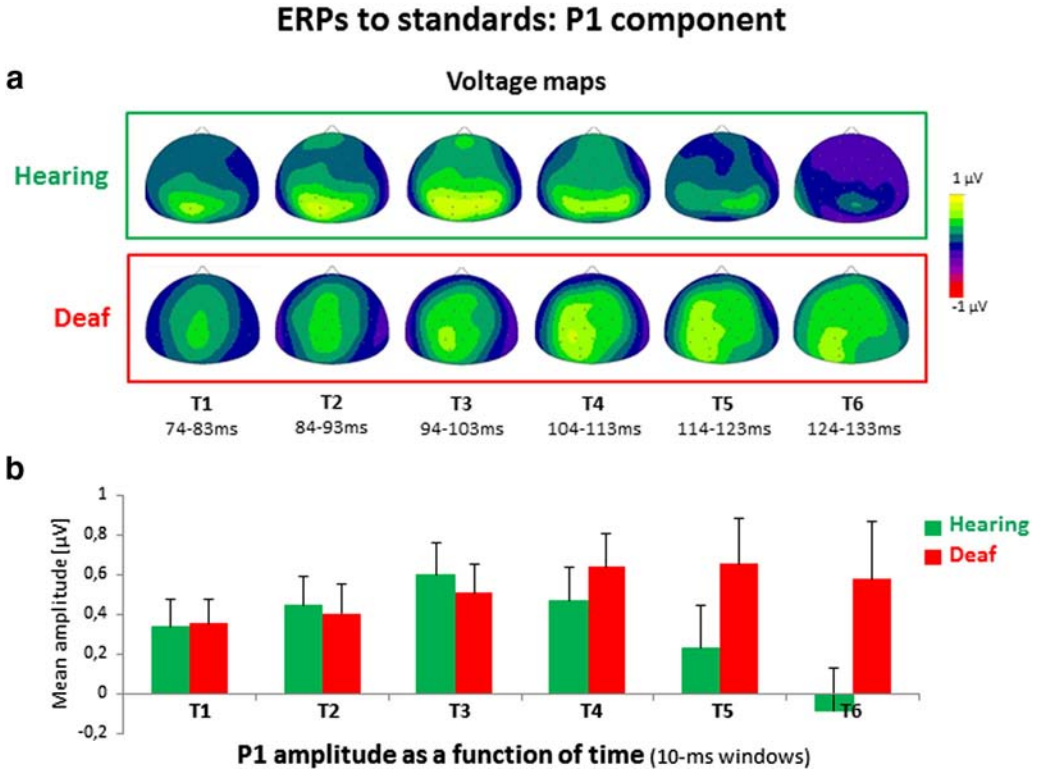

Fig. 4. (a) Potential maps illustrating the P1 dynamic over adjacent 20-ms time windows. (b) P1 mean amplitudes within each time window for each group. Deaf individuals show a prolonged P1 deflection compared to hearing controls.

window were entered in a mixed ANOVA with hemisphere (left, right) and cluster (F, C, P) as within-participants factors and group (deaf, hearing) as between-participants factor. No main effects or interactions were significant (all F-values < 3; see Fig. 2c).

P1 source analysis

Fig. 3a displays the SCD distribution of the P1 component around its peak latency in each subjects' group. These topographies suggest different P1 sources for deaf and hearing individuals. To test this hypothesis, we compared the source estimates of P1 at the latency of the peak averaged between the two groups using the sLORETA software. With respect to the first approach, we run a non-parametric test comparing the source estimates of the two groups on a 20 ms-time window [94–114 ms] centered on the average of the two groups' P1 peak (104 ms). The permutation test revealed that deaf individuals showed reduced activity at the occipital cortex (BA 18, cuneus) compared to hearing controls ($p = 0.05$, corrected for multiple comparisons; see Fig. 3b). This result is compatible with the topographic differences appearing in both ERPs and SCDs around these latencies (Fig. 3a) and supports the choice of the extrastriate visual cortices as visual ROI.

To evaluate the time course of the source estimates we run a series of ANOVAs on the selected ROIs and time windows (see Sandmann et al., 2012). Within the P1 time window (84–124 ms), the source estimates were computed for each ROI (visual and auditory) and then averaged with respect to two different time windows (84–103 and 104–113 ms) as the spatio-temporal dynamic of the P1 component was different between the two groups. These measures were entered into a mixed ANOVA with time window (84–103, 104–113 ms), hemisphere (left, right) and ROI (visual, auditory) as within-participants factors and group (deaf, hearing) as between-participants factor. The analysis revealed a main effect of time window ($F(1,22) = 7.7$, $p = 0.01$) and of ROI ($F(1,22) = 14.9$, $p < 0.001$). Most importantly the interaction between the ROI and group factors was significant ($F(1, 22) = 12.4$, $p < 0.002$ confirming a larger response in visual ROI in hearing controls compared to deaf individuals (t-test(12) $= -2.6$, $p < 0.0001$)), and also indicating a tendency for enhanced response at the auditory ROI for deaf individuals compared to hearing controls (t-test(12) $= 1.2$, $p < 0.08$, see Fig. 3c).

Deviance-specific ERPs: vMMN

Both subjects' groups showed a clear vMMN. Their period of significant amplitude was determined using a series of Wilcoxon tests comparing to zero the activity recorded at each time sample. In hearing controls, the vMMN peaked at cluster RP (posterior right) with an amplitude of 1.1 μV at 193 ms latency. In this cluster, the visual MMN was significant from 157 ms to 251 ms (94 ms duration). In deaf individuals, the vMMN peaked at cluster LC (central left) with an amplitude of 1.3 μV, at 252 ms. In this cluster the MMN was significant from 193 ms to 299 ms (106 ms duration).

To compare the vMMN between the two groups, we considered the time window starting 40 ms before the vMMN peak in hearing controls and ending 40 ms after the vMMN peak in deaf individuals (153–292 ms). The mean amplitude averaged over this 140 ms window was computed at each hemisphere and cluster and entered into a mixed ANOVA with hemisphere (left, right) and cluster (F, C, P) as within-participants factors and group as between-participants factor. The analysis revealed a main effect of cluster ($F(2, 44) = 8.03$, $p < 0.001$) and, importantly, a significant interaction between the cluster and group factors ($F(2, 44) = 10.1$, $p < 0.001$, see Figs. 5a, b). Post-hoc analyses (Fisher tests) revealed that hearing controls showed a vMMN of larger amplitude at posterior clusters than at central and frontal clusters ($p < 0.02$ and $p < 0.0001$, respectively), and also that central clusters showed a larger vMMN than frontal clusters ($p < 0.01$). Conversely deaf individuals showed a more anteriorly distributed vMMN as central clusters displayed larger amplitudes than posterior clusters ($p < 0.05$). No other main effects or interactions were significant (all Fs < 2.4).

To evaluate the time course of the two groups' vMMNs, the 140-ms time window was divided into consecutive 20-ms windows and the mean amplitude of the vMMN recorded within each of them was entered into a mixed ANOVA with time-window (153–172 ms, 173–192 ms, 193–212 ms, 213–232 ms, 233–252 ms, 253–272 ms, 273–292 ms) as within-participants factors, and group as between-participants factor. The analysis revealed a main effect of time-window ($F(6, 132) = 3.2$, $p < 0.01$) while the interaction time-window and group did not reach significance ($F(6, 132) = 1.6$, n.s.).

Dipole modeling of the vMMN

Modeling was applied on a 20-ms time window around the grand average vMMN peak of each group (see Fig. 6b for the results of the dipole modeling and SCD in the same time window). The results clearly indicate that the equivalent current dipoles were more anterior and lateralized in deaf than in hearing subjects. Hearing controls dipoles explained on average 86.22% of the variance. In deaf individuals the vMMN dipoles explained 85.3% of the variance, were sagittally oriented and laid on an area compatible with the superior temporal lobe (see Fig. 6b). To better understand the dipole fitting results we transformed the dipole coordinates into Talairach space (using Dipplot function of EEGLab and then Talairach client, tailarach.org, with a selection of ±5 mm cube around the target). In the hearing group the dipole solutions (−59, −52, −9; 59, −52, −9) were compatible with left/right posterior portions of the temporal lobe at the junction with the occipital lobe, including superior and middle temporal gyri, area 21, area 22, and area 39. Dipole coordinates in deaf participants (−51, −25, 4; 51, −25, 4) indicated a more anterior and ventral solution, compatible with the left/right superior temporal gyrus, Brodmann areas 41, and 22, and middle temporal gyrus area 21. Fig. 6b shows the vMMN dipoles for each group projected on an average MNI brain.

sLORETA source analysis of the vMMN

sLORETA procedure was used to compare the vMMN sources in two ways. First source estimates at the average (between groups) vMMN peak were compared between hearing and deaf individuals with a permutation test (as for the P1 component) and the sources estimated were extracted at each ROI (visual and auditory) again at the averaged vMMN peak. Then, to evaluate the dynamics of the vMMN sources, the time courses of the source estimates at each ROI were extracted in a longer time window, they were divided into seven bins of 20 ms each and entered as a factor in an ANOVA.

The first analysis based on the permutation test compared the source estimates of the vMMN between the two subjects' groups within a time window of 10 ms [219–229 ms] centered on the average vMMN peak (224 ms): no statistical significant difference was found between hearing and deaf participants. The lack of significant difference might be due to the highly conservative test used, combined with the small sample size (for similar results see Hauthal et al., 2013b; Sandmann et al., 2012). To deepen the comparison of the vMMN peaks (averaged within 219–229 ms latency range), the source estimates of the two groups extracted at each ROI were compared in an ANOVA with hemisphere (L, R) and ROI (visual, auditory) as within-participants factors and group (deaf, hearing) as between-participants factor. The analysis revealed a two-way interaction between the factors hemisphere and ROI ($F(1,22) = 4.5$, $p < 0.05$). Most importantly the interaction between the ROI and group factors was significant ($F(1,22) = 6.5$, $p < 0.02$, see Fig. 6c). Planned t-tests revealed that the source estimates for visual ROI showed a greater amplitude compared to the auditory ROI in hearing controls (t-test(11) $= 3.2$, $p < 0.01$), whereas deaf individuals showed comparable ROI amplitudes (t-test(11) $= -1$, n.s.).

The time courses of the source estimates were evaluated using a series of ANOVAs within specific ROIs and time windows (see Sandmann et al., 2012). In particular, we averaged the source estimates across ROIs in 7

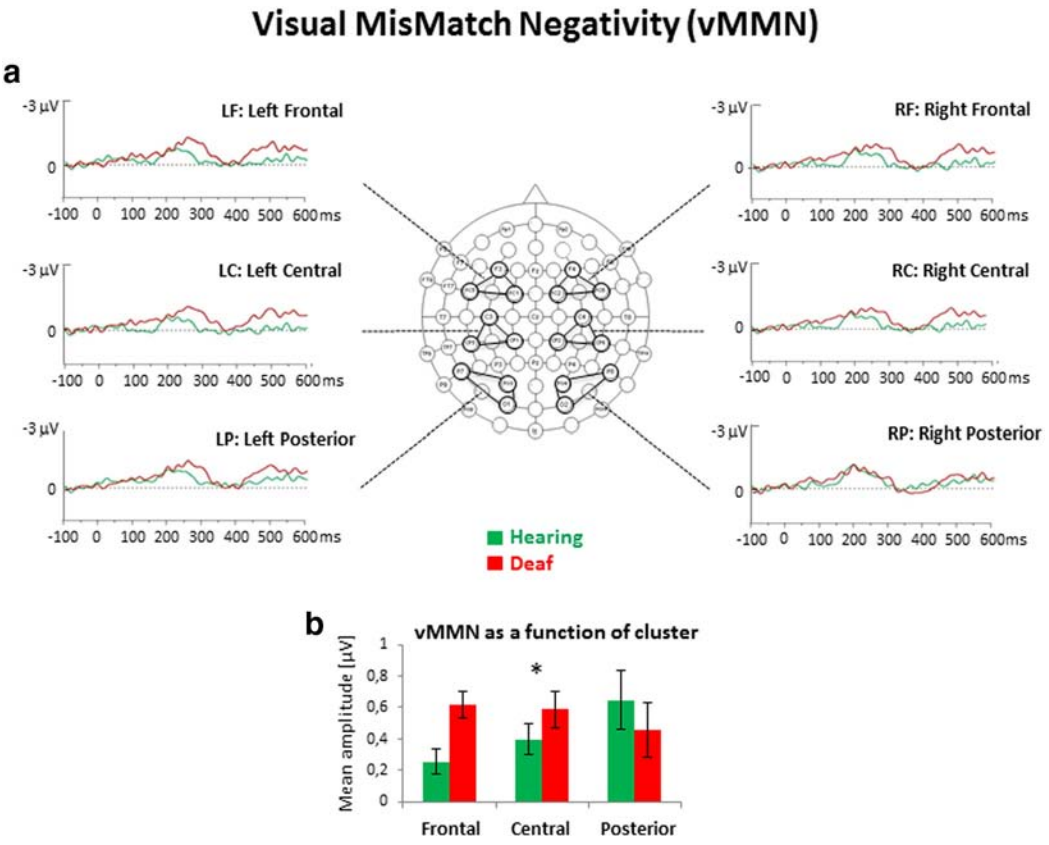

Fig. 5. (a) vMMN at each cluster of electrodes and hemisphere. (b) Mean amplitude of the vMMN at each cluster over the 153–292 ms latency range (140-ms time window, see text). While hearing controls show a posteriorly distributed vMMN, deaf individuals present anteriorly distributed vMMN topography.

time windows of 20 ms each (154–173 ms, 174–193 ms, 194–213 ms, 214–233 ms, 234–253 ms, 255–273 ms, 274–293 ms) to cover the vMMN time course (see Fig. 6c, right, to evaluate the dynamics of the source estimates at visual and auditory ROIs in the two groups). The source estimates were entered into a mixed ANOVA with time-window (7 bins) as within-participants factor, and group (deaf, hearing) as between-participants factor. The analysis revealed a main effect of time-window ($F(6, 132) = 2.9$, $p < 0.02$). The interaction between the time-window and group factors was instead not significant ($F(1,22) = 0.7$, n.s.). To compare the source estimates averaged across the whole 140-ms time window, they were entered into a mixed ANOVA with hemisphere (left, right) and ROI (visual, auditory) as within-participants factors, and group (deaf, hearing) as between-participants factor. The analysis revealed a main effect of ROI ($F(2, 22) = 9.1$, $p = 0.007$), and a significant interaction between the hemisphere and group factors ($F(1, 22) = 6.4$, $p = 0.02$). Most importantly the interaction between the ROI and group factors was also significant ($F(1, 22) = 12.1$, $p < 0.003$). Single t-tests revealed that hearing controls displayed enhanced source estimates at visual ROI ($t(22) = -2.5$, $p = 0.02$) compared to deaf individuals. Conversely deaf individuals showed enhanced source estimates at auditory ROI compared to hearing controls ($t(22) = 1.7$, $p = 0.05$, one tailed). This finding strengthens the results of dipole fitting, strongly suggesting that the visual MMN in deaf individuals is also related to the activity within the auditory cortex.

ERPs to targets

To compare between the two groups the response to a static visual event, we also analyzed the ERPs elicited by the offset of the fixation cross (target event of the visual detection task). P1 analysis was not possible as this component was virtually absent in the two groups. The N1 component was analyzed with the same rationale as for the previous analyses. First, for each participant the mean amplitude was computed at each cluster (LF, LC, LP, RF, RC and RP) within a ±20-ms time window centered on the N1 peak latency averaged across all participants (189 ms, 169–209 ms). The N1 peak in hearing controls had an amplitude of 3.55 μV at T6, 181 ms after the offset of the cross. In deaf individuals N1 peaked (3.38 μV) at T5 at 198 ms latency. For each participant the mean amplitudes at each cluster over the 169–209 ms time window were entered into a mixed ANOVA with hemisphere (left, right) and cluster (F, C, P) as within-participants factors, and group as between-participants factor. The main effect of cluster was significant ($F(1,44) = 5.4$, $p < 0.01$) as clusters C and P showed larger N1 mean amplitudes compared to cluster F ($p < 0.02$ and $p < 0.01$, respectively). In addition there was a significant interaction between the cluster and hemisphere factors ($F(2,44) = 7.2$, $p < 0.002$), confirming larger mean amplitudes for posterior clusters (RP) of the right hemisphere compared to clusters RF, LC, LF ($p < 0.03$, $p < 0.02$ and $p < 0.0001$, respectively). No main effect or interaction involving the group factor was significant (all F-values < 1; see Fig. 7).

Discussion

A core computational ability of the auditory cortices is the construction of a memory trace of the auditory scene and the automatic detection of discrepancies in the soundscape (for reviews see Näätänen et al., 2001, 2010). The principal aim of the present study was to test the hypothesis that the auditory cortex could be recruited for detecting dynamic changes in visual motion and form in people with early profound deafness. To this aim we elicited the visual MMN in deaf and hearing participants, using a visual motion stimulus consisting of a circle deforming along its vertical or horizontal axis. As a secondary aim, we also explored to what extent the repeating continuous visual motion stimuli (i.e., standards) – responsible for the formation of the memory

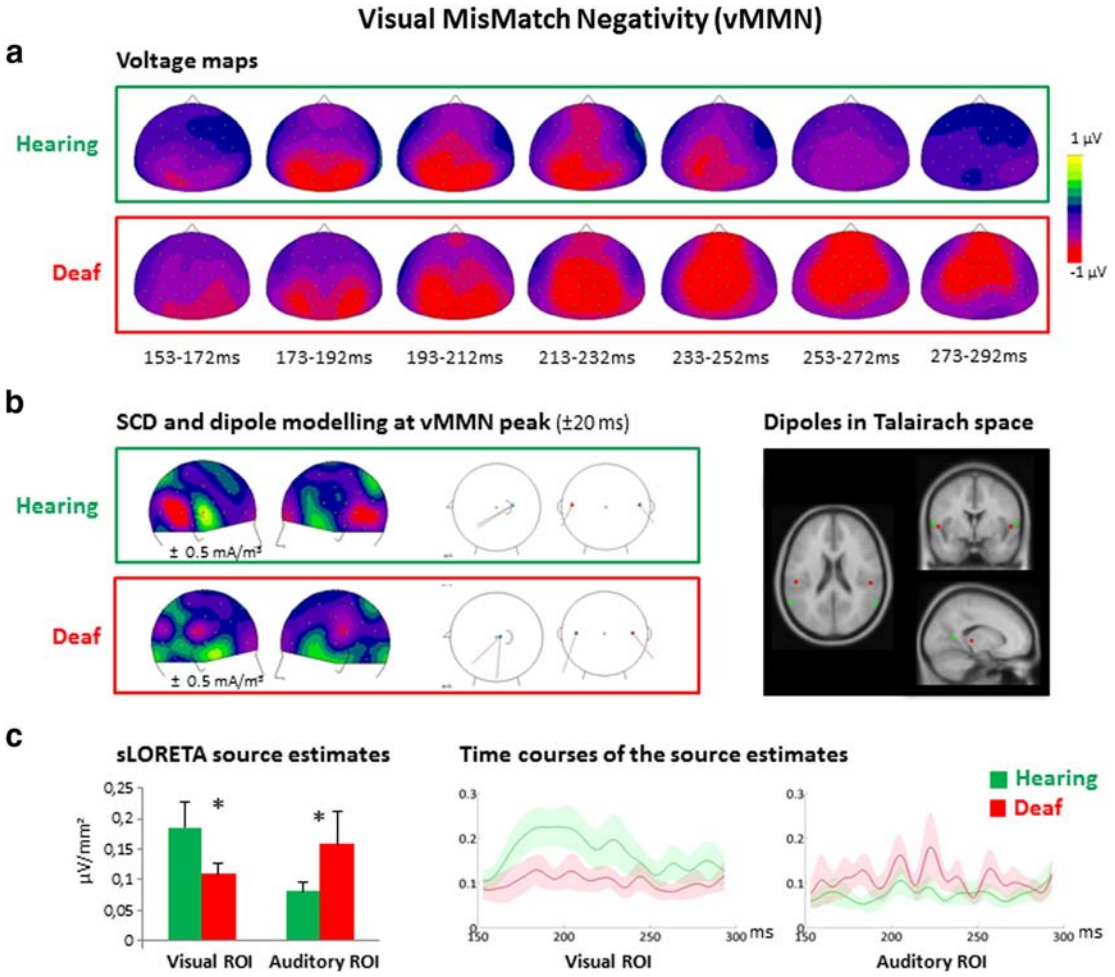

Fig. 6. (a) Potential maps of the vMMN between 153 and 292 ms (7 adjacent 20-ms time windows) in hearing and deaf participants. (b) Left: SCD and dipole modeling results calculated on a 20-ms window around the grand average vMMN peak of each group (hearing controls 193 ms, deaf individuals 252 ms). Right: vMMN dipoles for each group projected on an average MNI brain. Dipole coordinates in deaf participants indicate a more anterior and ventral solution for the vMMN with respect to hearing controls, compatible with auditory cortices (BA41) within the superior and middle temporal gyri. (c) Left: source estimates of vMMN averaged over the whole 140-ms period (153–292 ms) for each group and ROI. While deaf individuals show reduced activation at visual ROI with respect to hearing controls, they show enhanced response at the auditory ROI. Right: time course of the source estimates at visual and auditory ROI within the vMMN 140-ms time window.

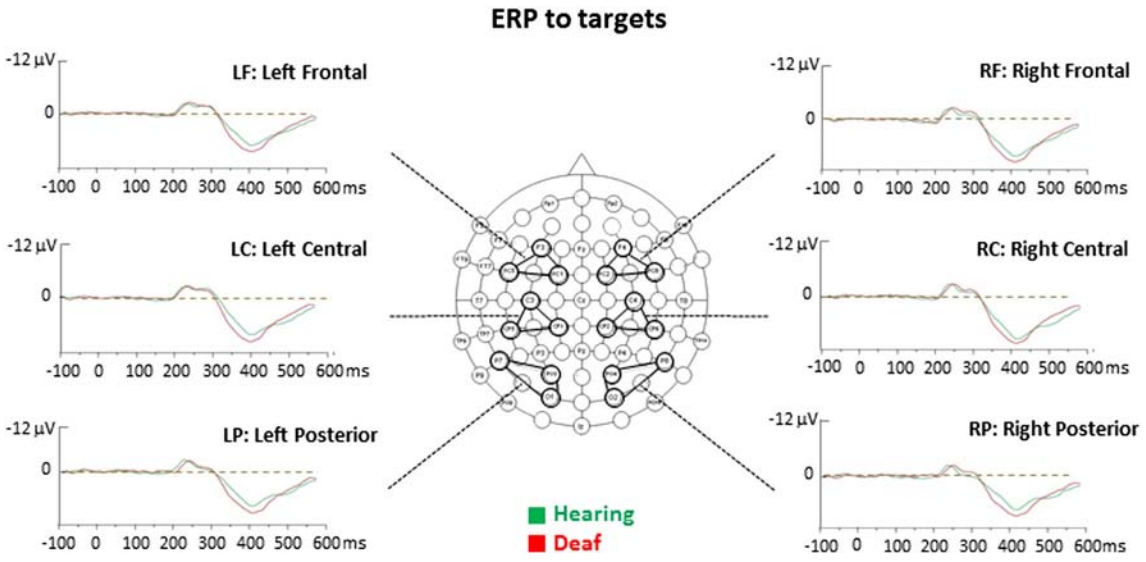

Fig. 7. ERPs to targets (cross disappearance) at each cluster of electrodes and hemisphere. Deaf individuals and hearing control responses do not differ.

trace in the vMMN process – also recruited temporal areas in deaf individuals.

Two main findings emerged. First, we observed a robust vMMN response in deaf people with sources more anterior and ventral than those of hearing controls, and compatible with a localization within the auditory cortices. This was further confirmed by the between-group comparison of source estimates in visual and auditory ROIs. The analysis of the temporal dynamics of this vMMN in deaf people revealed that auditory cortices recruitment occurred with the same timing as the vMMN response (150–300 ms). In addition, the enhanced vMMN response in the auditory ROI measured in deaf individuals with respect to hearing controls was paired with a reduced activation at the visual ROI.

Second, we observed different responses to visual standard stimuli between deaf and hearing participants at early processing stages (P1 component). Importantly also, the source estimates of this early component differed between the two groups of participants: similarly to the vMMN response, the P1 estimated source for deaf people was weaker in visual ROI compared to hearing individuals, and paired with a tendency for being larger at auditory ROI in deaf individuals.

Visual change detection for moving stimuli in the auditory cortices of deaf adults

Dynamic visual events deviating from frequent standard events elicited a robust (about 100 ms long) vMMN in both hearing controls and deaf individuals. The most evident difference between the two subjects' groups emerged in the topographies of the ERPs and in the corresponding SCDs.

Results showed different sources for the vMMN in early deaf adults compared to hearing controls. The voltage maps of the vMMN suggest that in deaf adults the generators of vMMN are more anterior than those of hearing controls, with peak amplitude at central electrodes instead of posterior sites. Similarly the SCD maps strongly suggest different sources of the two vMMNs, with posterior sources in hearing controls but a combination of posterior and more anterior sources in deaf individuals. Dipole fitting analysis confirmed and extended this finding. In hearing controls, the main generators (equivalent current dipoles) were found at posterior sites, compatible with visual cortices; by contrast, in the deaf group the dipole model located the main generators at temporal sites. Clearly, the dipole source modeling could not show the exact location of the vMMN in either group as several generators are probably active simultaneously (early visual cortices certainly contribute as suggested by the SCD topographies). Nevertheless, it shows that the average source (in space) was shifted toward the auditory cortices as result of deafness. The sLORETA source estimates were analyzed to examine more directly the responses within the two ROIs of interest: occipital extrastriate visual areas (BA 18 and BA 19) and auditory cortices (BA 41 and BA 42). This analysis showed that deaf individuals displayed enhanced activity within the auditory cortices, during the whole vMMN time course (140 ms) and simultaneously reduced activity within the visual cortices.

The present results thus showed that the auditory cortices of deaf people participate in the process of monitoring and detecting regularities/changes in the visual stream. It is important to note that the vMMN response in this visual task is elicited only by the presence of "a change" in visual movements (or in shape deformation). The physical characteristics of the stimuli are irrelevant as we averaged the responses to standard events with horizontal and vertical movements, as well as the responses to deviant events with the same logic. Thus, the differential activity that we observed in response to the deviant category can be interpreted as the marker of change detection mechanism. To generate such knowledge a neural system has to extract and store the rule and compare on-line the upcoming information. The present data suggest that cross-modally recruited auditory cortices can reach this level of computation. In support of this partial shift of computation from the visual to the auditory cortices we observed in deaf individuals a reduction of vMMN source estimates at visual cortices with respect to hearing controls.

In three (ERP and MEG) studies, Besle et al. (2005, 2007, 2013) found that rare audio-visual stimuli deviating from standard audio-visual stimuli along the two (auditory and visual) dimensions elicited a significant audio-visual MMN (avMMN), which differed from the two unisensory (auditory and visual) MMNs. With respect to the hypothesis of the involvement of the auditory cortex in deaf individuals' vMMN, it is of interest to compare the topographies we observed in the present study with the ones that Besle and colleagues found using the same visual MMN paradigm but including also an auditory signal, in a group of hearing participants only. In their first ERP study, Besle et al. (2005) measured an avMMN when an auditory signal was also present but not part of the deviance and a genuine avMMN where the response to the change in the stream was triggered by the violation of both the auditory and visual features (Besle et al., 2005, Fig. 3, page 341). It is remarkable to observe a large overlap between the genuine avMMN topography in hearing individuals observed in Besle et al., 2005 and the vMMN in deaf individuals found in the present study. This analogy of the topographies supports the hypothesis that the auditory cortex maintains its property of sensory memory processing even when exposed to a different developmental trajectory, as in the case of early auditory deprivation. Importantly, further support to this hypothesis is also provided by our finding reporting a recruitment of auditory cortices with the same timing as the visual MMN response.

Response to visual motion/shape deformation in auditory cortices in deaf people

The analysis of the responses to standard visual stimuli revealed that deaf individuals and hearing controls display different ERP topographies already at the latency of the P1 component (i.e., around 100 ms after the movement onset). This result extends the findings of our previous work on the detection of attended sudden visual stimuli in deaf people (Bottari et al., 2011). Using a simple detection paradigm we showed that when processing attended static visual events, deaf individuals present a different dynamics in their visual response already at the level of the P1 component, with a prolonged response for stimuli at both central and peripheral visual eccentricities.

The sLORETA source estimates suggested that deaf individuals compared to hearing individuals show decreased activation within visual cortices (BA 18), in analogy with previous findings in deaf people fitted with cochlear implant (Sandmann et al., 2012). Conversely we only found a tendency toward enhanced activation at the auditory ROI in deaf compared to hearing controls at this latency. The present findings extend this previous result in three ways: 1) they replicate it even for an unattended stimulus (attention was drawn to occasional disappearance of central fixation, not to the changing circle); 2) they suggest – at least partly – different generators of the P1 component in hearing controls and deaf adults, with the possible implication of the temporal cortex in the deaf group; and 3) they reveal that the observation made by Sandmann et al. (2012) on postlingually deafened cochlear implant patients can hold true also for early deaf participants who do not use a cochlear implant.

The present study confirmed that in the case of early auditory deprivation, the scalp distribution of the ERP responses associated with visual processing differs from that found in hearing controls (Armstrong et al., 2002; Bottari et al., 2011; Hauthal et al., 2013b; Neville and Lawson, 1987). This topographic difference seems to be related to the involvement of the deafened auditory cortices.

Enhanced response in auditory cortices, but reduced response in visual cortices

As mentioned previously, in the present study the auditory cortices recruitment for visual processing in early deaf adults was accompanied

by reduced activation within the extrastriate visual cortices. Importantly, this phenomenon is remarkably consistent, as it emerged at both P1 and vMMN levels. These results replicate and extend previous findings on postlingually deafened cochlear implanted individuals attending static visual stimuli (Sandmann et al., 2012), for whom recruitment of auditory areas during visual processing was paralleled by decreased extrastriate response at the P1 latency. This reduction of visual extrastriate response would possibly indicate either smaller assembly or reduced synchronization of the activated neurons (Sandmann et al., 2012). While interpreting the effect of cross-modal plasticity (response in auditory cortices for visual stimuli) within a framework of Hebbian competition seems to be a plausible manifestation of brain plasticity, the decreased activity of visual areas in the case of deafness appears less obvious. One possibility is that visual cortices of deaf individuals are more efficient (see Bottari et al., 2011; Hauthal et al., 2013b) when processing visual information and thus need less "visual energy" to process simple visual events. Alternatively, it is also possible to hypothesize that the additional computation performed within auditory areas reduces perceptual load in visual areas. Clearly these two somewhat speculative hypotheses are not mutually exclusive. Interestingly, studies on congenital blind individuals showed that the recruitment of the visual cortices in response to auditory stimuli can be accompanied by weaker responses in primary auditory cortices compared to sighted participants (Gougoux et al., 2009; Klinge et al., 2010). In the literature on blindness, this effect has been interpreted as the consequence of a more widespread auditory network that would include visual areas (Gougoux et al., 2009). Importantly, because these results on blind individuals were obtained using fMRI, the timing of this phenomenon is currently unknown. Conversely, the present data suggest that this is a synchronous "push–pull" effect. To summarize, concurrent but opposite (in terms of neural processing/recruitment) phenomena of cross-modal and intramodal plasticity (visual stimuli and visual cortices) seem to occur in the case of sensory deprivation. Despite being novel evidence, these adaptation mechanisms might represent a general principle as these plastic changes seem to be observable as the result of both deafness and blindness.

Response to static offset

The ERP response to the targets (offset of the fixation cross) did not show differences between the deaf individuals and hearing controls. This lack of difference has interesting implications. On the one hand, the fact that the brain response to the attended target did not differ between the two groups suggests that the differences observed in response to the unattended motion stimuli (standard stimuli) can hardly be ascribed to attentional differences. On the other hand, this result supports the hypothesis put forward by several groups that motion processing is a special context to observe cross-modal plasticity in deaf individuals (Armstrong et al., 2002; see Neville and Bavelier, 2002 for a review).

A multimodal system for change detection in auditory cortices?

The present findings are compatible with the notion that neural plasticity in the brain occurs within the functional constraints of a given brain circuit. Specifically, our results might provide initial evidence toward the existence of a multimodal system for change detection of moving stimuli in the auditory cortices. In hearing people this system operates in response to auditory inputs or in response to audio-visual inputs; instead, in deaf people it seems to operate on visual-only inputs.

Evidence for functional selectivity in the cross-modal recruitment of the auditory cortex was documented in the animal model in the seminal work of Lomber et al. (2010). Studying congenitally deaf cats, these authors demonstrated that the cooling of a selective portion of the auditory areas (specifically DZ, an area located in posterior secondary auditory cortices of cats) causes the decrease of performance in a visual motion discrimination task to a level comparable to hearing cats. In hearing cats, this secondary posterior auditory area is known to be involved in the processing of sound motion (Malhotra and Lomber, 2007). These results are in line with a growing body of evidence in the literature on cross-modal plasticity in blind individuals, reporting the maintenance of functional specialization within the reorganized visual areas (for reviews see Dormal and Collignon, 2011; Meredith et al., 2011). In blind adults, for instance, Collignon et al. (2011) showed in a fMRI experiment that the processing of spatial features in hearing and congenitally blind individuals activates a specialized sub-region of the right dorsal occipital stream, a region that is part of the dorsal stream in sighted people.

Whether the multimodal region for change detection discussed above would be active for any changes, or instead would be selectively recruited for motion changes remains an open issue that could not be addressed with the present data. The predictions of the two alternatives are however clear. In the first case, any type of visual change should recruit auditory cortices in deaf people. In the second case, changing the feature from motion (as here) to – say – color should no longer recruit the auditory cortex of deaf people. Examples of similar dissociations were also given in a previous study (Armstrong et al., 2002). However, irrespective of whether our findings reflect the recruitment of a multimodal circuit for change detection dedicated to all types of discontinuities or for only some types of irregularities, the involvement of the auditory cortices in the vMMN process demonstrates that when a sensory cortex is cross-modally recruited by another modality, its processing level can go beyond simple features. In conclusion, while it was known that the deaf auditory cortex could be activated by tactile and visual stimulation the present findings strongly suggest that the deafened auditory cortices may participate in the process of monitoring and detecting regularities/changes in the visual stream. Remarkably, as the vMMN response in auditory cortices for visual change detection was paired with a reduction of response within the visual system, in the case of early deafness part of the visual computations implied in visual monitoring could shift from visual to auditory cortices.

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