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Visual speech circuits in profound acquired deafness: a possible role for latent multimodal connectivity

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It is commonly held that losing one sense provokes cross-modal takeover of deprived cortical areas, and therefore results in a benefit for the remaining modalities. Using functional magnetic resonance imaging (fMRI), we assessed the impact of acquired deafness on the brain network related to speechreading and teased apart cortical areas with responses showing long-term reorganization, i.e. time-dependent plasticity over 4–48 months of deafness, from those expressing compensation, i.e. performance-related activity. Nine deaf patients (7 women, age; mean ± SE. = 50.2 ± 4.8) and control subjects performed equally well in a visual speechreading task but deaf patients activated the left posterior superior temporal cortex more than controls. This effect correlated with speechreading fluency but not with the duration of sensory deprivation, thus arguing against long-term reorganization as the source of these cross-modal effects. To the contrary, cross-modal activation in the left posterior superior temporal cortex of deaf patients decreased with deafness duration. Our observation that cross-modal effects were most pronounced right after deafness onset is at odds with the classical view on brain reorganization. We suggest that functional compensation of sensory deprivation does not require slowly progressive colonization of superior temporal regions by visual inputs, but can exploit a switch to pre-existing latent multimodal connectivity.

Keywords: deafness; speechreading; fMRI; multimodal; reorganization

Abbreviations: fMRI = functional magnetic resonance imaging; PSNHL = progressive sensorineural hearing loss; STG = superior temporal gyrus; STS = superior temporal sulcus

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Introduction

Thanks to multisensory competition for cortical space, profound deafness provokes brain reorganization that benefits the remaining senses (Levanen and Hamdorf, 2001). Visual capacities in profound congenitally deaf patients, however, appear only enhanced in behavioural domains that would normally benefit from audio-visual integration, and that are under strong attentional top–down control (Bavelier *et al.*, 2006). Two neural mechanisms may account for these behavioural effects. The loss of auditory input in direct projections from auditory to visual areas (Falchier *et al.*, 2002) could possibly enhance susceptibility of visual areas to visual inputs and visual top–down control (Bavelier *et al.*, 2006). Alternatively, the temporal cortex may undergo cross-modal reorganization so that visual stimuli tap into regions that normally respond predominantly to sounds (Calvert *et al.*, 1997; Petitto *et al.*, 2000; Bavelier *et al.*, 2001; Finney *et al.*, 2003; Fine *et al.*, 2005; Pekkola *et al.*, 2006). Accordingly, there is abundant evidence for visual (or tactile) responses in temporal cortex of the deaf, even reaching the primary auditory region (Puce *et al.*, 1998; MacSweeney *et al.*, 2002, 2004; Finney *et al.*, 2003; Fine *et al.*, 2005; Lambertz *et al.*, 2005; Doucet *et al.*, 2006).

Visual responses in superior temporal cortex could result from long-term cross-modal reorganization, but they might also reveal more dynamic functional phenomena. In acquired deafness in particular, cross-modal responses might indicate an immediate change in the relative contribution of auditory/visual/somatosensory input to the temporal cortex. This view implies that superior temporal regions that are classically held as unimodal auditory, are covertly hetero- or multimodal (Beauchamp, 2005; Ghazanfar and Schroeder, 2006; von Kriegstein and Giraud, 2006).

This property may result from converging inputs to the temporal region from different sensory cortices (Ghazanfar *et al.*, 2005), or from low-level functional interactions across associative cortices creating multimodal functional circuits (Beauchamp, 2005; von Kriegstein and Giraud, 2006). These circuits have been shown to become rapidly functional once sensory or task requirements change (von Kriegstein and Giraud, 2006). It is therefore possible that auditory deprivation, rather than inducing slow progressive structural reorganization of temporal cortex, provokes a rapid functional reorganization of latent multimodal circuits, e.g. by disinhibiting far-reaching connectivity with other sensory modalities.

In this study, we pursued the hypothesis that, if cross-modal responses following sensory deprivation result from temporal reorganization relying on new connectivity and synaptogenesis, visual responses in temporal cortex in deaf people should build-up over months to years following sensory deprivation. If, conversely, cross-modal responses result from immediate reorganization, e.g. a takeover of latent multimodal connectivity, visual responses in temporal cortex should be observed rapidly, irrespective of the time elapsed since deafness onset.

This dichotomy has important implications with respect to the behavioural benefit that cross-modal responses may provide. If latent networks are unveiled by auditory deprivation, their participation in global functional network should moderately be disturbed, and they should readily participate in effective compensatory mechanisms, whereas if rewiring and synaptogenesis underpin cross-modal responses, related behavioural effects should build-up over months to years after deafness onset (Giraud *et al.*, 2001; Smirnakis *et al.*, 2005).

Speechreading is the major communication mode in acquired profound deafness, and should thus be under strong reorganization demands. By disentangling neural activity related to deafness duration from that related to speechreading fluency during a speechreading task, we assessed the contribution of long-term versus short-term plasticity to the shift towards exclusive visual communication in deafened adults.

Methods

Participants

Twelve post-lingually deaf adults without known neurological disease were enrolled in a research investigation that was approved by the local ethics committee (CPP, Sud-Est IV, Centre Léon Bérard, Lyon, France). Ten of them had bilateral sensorineural hearing loss since childhood yielding profound deafness in adulthood. The other two patients were deafened by ototoxic drug and head trauma. None of them used sign language, and they all relied on speechreading and reading for communication.

Three deaf patients were excluded from the functional magnetic resonance imaging (fMRI) analysis because they could not perform the tests correctly during scanning, leaving data from 9 deaf patients 7 women and 2 men, age; mean ± SE = 50.2 ± 4.8, see Table 1 for demographic data) and 15 controls (7 men and 8 women, age; mean ± SE = 32.2 ± 3.4). To minimize age effect in group comparisons, the latter were performed only with the 9 oldest control subjects (3 men and 6 women, age; mean ± SE = 39.3 ± 4.2). Data from the other six controls, however, were included in a conjunction analysis that assessed effects that were consistently present in deaf subjects and absent in controls. All controls had normal hearing and no audiological or neurological antecedents. Written informed consent was obtained from all participants. All of them had normal or corrected-to-normal visual acuity, and were right-handed according to a modified version of Edinburgh Inventory of Handedness (Oldfield, 1971).

Assessment of clinical parameters

In deaf patients, speechreading fluency was assessed before the fMRI investigation by a standard French sentence test with visual cue only. A list composed of 100 three-phoneme words (Lafon, 1964) was presented by a speech therapist and the correctly identified words were scored.

Duration of deafness was determined as the number of months elapsed since onset of profound hearing loss. Onset of profound deafness is defined as the time point when oral communication was fully compromised even with the best-fitted hearing aid. Clinical details are provided in Table 1 for the 9 deaf subjects who participated in the fMRI data analysis.

FMRI experiment

Stimuli

During fMRI, subjects performed a speechreading task (SR) from short videos of a French native male pronouncing French numbers from 1 to 9. In the original version of the videos, the speaker pronounced the numbers aloud and the sound track was subsequently removed. In a baseline condition (CO), the same speaker made closed-mouth gurning movements at the same rate as the pronounced numbers. The stimuli were edited by iMovie version 4.0.1 on Macintosh (Apple computer, Inc., USA). Each video lasted 2 s. Duration, onset time and magnitude of mouth movements were equated across conditions.

Experimental protocol and design

To ensure that all participants had understood the instructions correctly and could identify numbers by speechreading, subjects were trained with a computer program using the same stimuli as those used during scanning. After training, all participants showed over 80% correct identification rate during the speechreading task. During fMRI, subjects either covertly repeated the number pronounced by the speaker (SR) or counted the number of face videos (always the same person) that appeared on the screen (CO). In both tasks, subjects pressed a response button with the right index finger whenever the identified number or the count was even. The two tasks were thus overall balanced with respect to motor output as even numbers occurred as often as even counts. The internally represented linguistic content and the task-related semantic judgement were also balanced across conditions in this way. Videos were presented randomly to prevent anticipation and force speechreading in the SR condition. In the control task however, randomization of gurning faces did not determine the

Table 1 Clinical data

Patient number	Sex	Age	Cause of deafness	Duration of deafness (months)	Duration of bilateral hearing loss (years)	Hearing threshold without/with hearing aid (dB HL)		Word perception test score (%)	
						Right ear	Left ear	Auditory-only condition	Visual-only condition (Speechreading)
1	F	64	PSNHL	36	46	107/65	95/no HA	0	50
2	M	41	PSNHL	4	26	100/58	100/73	0	52
3	F	28	PSNHL	12	10	88/63	98/73	0	56
4	M	58	PSNHL	16	41	93/81	82/72	0	60
5	F	59	PSNHL	36	11	98/82	68/63	0	82
6	F	52	PSNHL	36	23	103/no HA	87/no HA	0	44
7	F	29	Head trauma	5	1	112/no HA	100/no HA	0	52
8	F	52	PSNHL	48	22	90/65	90/63	0	52
9	F	69	PSNHL	8		77/48	97/80	0	60

output and subjects counted regularly from 1 to 9. Hence, there remained an inevitable difference between conditions in predictability of content and task response.

The fMRI experiment comprised two runs, with five 2-min SR blocks and five CO blocks, randomly presented using Presentation software version 9.90 (Neurobehavioral system, Inc., Albany, CA, USA). This experiment was part of a larger protocol designed to test various aspects of visual language in deaf people (see Supplementary Material). Each block began with a screen showing instructions for 2 s, and 9 stimuli followed in random order with a 1 s black screen in between stimuli. A fixation cross was presented at the beginning and the end of each block and a 25 s black screen followed each block.

The stimuli were back-projected onto a screen positioned at the base of the scanner by a Canon Xeed SX50 LCD projector, and viewed via a mirror angled above the subject's head in the scanner.

Imaging parameters

Gradient echo-planar MRI data were acquired with a 1.5T magnetic resonance scanner (Siemens Sonata) with standard head coil to obtain volume series with 27 contiguous transverse oblique slices of 4 mm thickness (voxel size $3.4 \times 3.4\,mm^2$ in transverse plane, no gap, TR 2.6 s, TE 60 ms) covering the whole brain. Earplugs and earmuffs were provided both to controls and deaf subjects to equate experimental environment. In each run, 244 functional images were acquired. The first four images from each run were discarded to reach a steady state—each experimental session thus yielded 480 scans per subject.

Data analysis

We analysed the fMRI data using SPM2 (Wellcome Department of Cognitive Neurology, London, UK, http://www.fil.ion.ucl.ac.uk/spm) in a Matlab 7.1 (Mathworks, Sherborn, MA, USA) environment. Standard preprocessing procedures (realignment and unwarping, normalization and spatial smoothing with an 8-mm full width at half-maximum Gaussian kernel) were performed and statistical block-design analyses including two conditions were modelled for individual data analysis. The output of these individual analyses was entered into three second-level, random-effect (RFX) analyses.

Group analyses: main effects and group differences

An ANOVA with two groups per condition examined the main effects of condition in each group. We report, in Table 2, the effects that are significant at $P = 0.05$, FWE-corrected for multiple comparisons. We used a statistical threshold of $P = 0.001$ ($T = 3.37$), for group comparisons (Table 3).

Speechreading effects common to all deaf subjects relative to controls

We ran another ANOVA including contrast images of the speechreading condition from each of the 9 deaf subjects and all 15 hearing controls, and performed a conjunction analysis (deaf 1 > controls and deaf 2 > controls up to deaf 9 > controls) to identify regions that were consistently overactivated in every deaf patient compared to the control group.

Multiple regression analysis of deafness duration and speechreading performance factors

Effects due to duration of deafness and lipreading fluency in deaf patients were assessed in the fMRI dataset. Contrast images of the speechreading condition in deaf patients were entered into a multiple regression analysis with the two clinical factors as covariates to identify brain regions correlating with each factor. Age was additionally entered in this analysis to remove its possible influence on the experimental variance. In a further step, findings on speechreading effects from the deaf patients' group were used to define an inclusive mask (corrected $P = 0.05$), and the results were thresholded using a voxel height threshold of $P < 0.01$ ($T = 3.14$), uncorrected.

Results

Clinical measures

We first analysed how deafness impacted on the performance of visual speech by assessing the correlation between deafness duration and lipreading fluency measured during an open set clinical test. In the group of 12 subjects, the two factors appeared independent of each another (Fig. 1, $P = 0.167$, correlation coefficient $r = -0.431$), indicating that

Table 2 Main effects (FWE-corrected $P = 0.05$) and speechreading-specific activations ($P = 0.005$ uncorrected)

	R/L	Brain region (BA)	MNI coordinates (T)					
			Deaf patients			Hearing controls		
			Speechreading	Counting	Speechreading > Counting	Speechreading	Counting	Speechreading > Counting
Temporo parietal, *lateral*	L	Planum temporale, posteromedial (42)	−44 −38 14 (8.52)		−38 −36 10 (2.85)			
	L	STG anterior	−50 −4 −10 (5.79)					
	L	STG posterior (22)	−54 −36 12 (9.51)	−54 −38 12 (6.90)		−62 −38 14 (6.52)		−62 −36 14 (2.71)
	L	MTG (21)	−64 −30 2 (10.50)		−64 −30 2 (4.03)			
	L	MTG posterior (21)	−52 −48 10 (6.83)					
	L	Temporooccipital junction (21)	−52 −62 10 (6.30)	−48 −66 6 (6.71)		−50 −56 4 (6.45)		
				−50 −64 8 (6.68)				
	L	Inferior parietal lobule (40)	−46 −50 50 (8.41)	−46 −50 52 (9.43)		−42 −50 52 (6.66)		
	R	STG anterior (22)	52 12 −10 (8.16)					
	R	STS posterior (22)	58 −42 10 (9.90)	58 −42 12 (7.85)		58 −42 10 (7.23)		
			52 −38 6 (9.21)			52 −38 6 (6.88)		
	R	STS middle (22)	64 −24 −2 (7.02)			56 −26 −4 (5.98)		
	R	MTG (21)	54 −28 −12 (7.52)			56 −26 −4 (5.98)		
	R	MTG posterior (21)	52 −54 2 (7.57)	52 −54 0 (8.66)		60 −48 0 (6.29)		
	R	Temporooccipital junction (21)				48 −62 4 (6.69)	46 −62 4 (7.12)	
	R	Temporoparietal junction (40)	52 −40 26 (8.61)	62 −40 24 (9.37)				
			50 −44 36 (6.64)					
	R	Inferior parietal lobule (40)	40 −48 48 (6.08)			36 −56 56 (6.03)		
Temporal, *ventral*	L	Fusiform gyrus (37/19)	−50 −48 −16 (6.71)	−40 −50 −16 (6.52)		−42 −64 −20 (5.75)		
			−38 −68 −20 (7.47)	−40 −66 −20 (7.37)				
	R	Fusiform gyrus (37)	42 −50 −14 (6.23)	42 −50 −14 (7.40)		46 −52 −14 (6.98)	44−52−16 (5.6)	
			−1246 −50 (6.21)			44 −56 −24 (6.46)		
Insula	L	Insula	−26 20 4 (12.67)	−38 12 4 (8.71)	−26 20 −6 (3.66)	−46 18 −4 (12.62)	−40 12 2 (6.21)	−46 18 −4 (5.44)
			−28 20 −4 (11.59)		−24 20 6 (3.40)	−26 20 2 (9.38)		−30 18 −6 (4.70)
			−34 14 4 (10.52)			−30 18 −6 (9.24)		
	R	Insula	34 20 0 (8.15)	34 20 2 (8.45)		32 20 0 (8.45)		30 18 −14 (2.99)
						34 26 −10 (6.92)		
Frontal, *lateral*	L	Middle frontal gyrus, frontopolar (10)				−36 52 8 (6.01)		−34 56 10 (2.72)
	L	Middle frontal gyrus (9)	−40 20 30 (6.83)			−42 18 42 (7.18)		−38 16 28 (4.36)

			−48 22 30 (6.74)					−44 18 40 (4.11)
	L	Inferior prefrontal gyrus (44)	−56 10 16 (7.06)			−60 6 10 (8.57)		
	L	Precentral gyrus (6)	−44 −4 42 (8.40)	−46 −2 42 (7.23)		−52 4 36 (7.78)		
	R	Inferior frontal gyrus, pars orbitalis (47)	50 26 −12 (6.44)					
	R	Middle frontal gyrus (9/46)	36 36 38 (7.53)	36 36 38 (7.15)		44 32 38 (5.96)		
						36 42 22 (7.44)		
	R	Inferior frontal gyrus (44)	60 14 24 (9.74)	48 8 16 (8.54)		54 16 30 (7.83)		
			50 16 28 (9.09)					
			48 8 18 (8.47)					
	R	Precentral gyrus (6)	50 4 42 (7.56)	50 4 42 (7.48)		44 8 26 (6.76)		
			50 −2 56 (7.39)			48 4 40 (6.62)		
			42 0 46 (6.87					
Frontal, *medial*	L/R	Medial frontal gyrus (6)	−6 −4 60 (9.55)	−6 −6 60 (7.43)		−6 −4 60 (9.07))	−6 −6 60 (6.33)	−6 4 58 (3.77)
			8 12 50 (7.83)					−4 14 50 (3.36)
			6 −6 68 (7.26)					
	L	Cingulate sulcus (32)				10 16 38 (5.92)		
Occipital	L	Inferior occipital gyrus (18)	−24−100−2 (12)	−24−100−2 (12.8)		−24−100−2 (8.55)	−24−99 0(8.04)	
			−10−92−14 (9.72)	−44 −80 −2 (6.77)			−24−92−1(6.46)	
	R	Inferior occipital gyrus (18)	16 −104 8 (9.41)	14−102 14 (9.53)		30 −90 −10 (7.82)	28−88−10(7.17)	
			14 −100 −8 (8.28)					
			22 −96 −6 (7.01)					
Basal ganglia	R	Putamen	20 −8 16 (8.02)		20 −8 16 (2.85)	24 6 2 (6.04)		
	R	Thalamus				6 −20 6 (6.54)		4 −20 6 (3.46)
	L	Putamen	−20 2 12 (9.63)	−20 2 12 (7.26)	−26 8 4 (3.11)	−24 0 6 (7.16)		
	L	Thalamus	−12 −8 6 (6.98)			−10 −14 4 (7.38)		−10 −14 6 (3.53)
Cerebellum			24 −62 −28 (6.75)		10 −64 −26 (2.99)	12 −76 −18 (6.13)		
			8 −74 −11 (6.32)					

BA = Brodmann area; STS = superior temporal sulcus; STG = superior temporal gyrus; MTG = middle temporal gyrus.

Table 3 Deaf patients > controls during speechreading (uncorrected $P = 0.001$, $T = 3.37$)

	L/R	Region	BA	MNI coordinates	Cluster size	Voxel T
Temporoparietal, *lateral*	L	STG posterior	42	−42 −36 12	47	4.71
	L	Inferior parietal lobule	40	−60 −44 42	18	3.77
	L	STS middle	22	−64 −30 2	19	3.82
	L	STS middle	22	−38 −28 −4	4	3.52
	L	STG anterior	22	−48 −6 −10	1	3.40
	R	STG anterior	22	54 4 −12	18	3.76
	R	Temporoparietal junction	22	58 −40 24	4	3.53
Prefrontal	R	Inferior frontal gyrus	44	62 16 22	19	3.87
	L	Posterior orbital gyrus	47	−26 32 −4	26	4.09
	L	Middle frontal gyrus	8	−32 30 54	1	3.40
Occipital	L	Lingual gyrus	18	−6 −92 −12	62	4.86
	L	Cuneus	17	−16 −98 −6	4	3.38
	R	Middle occipital gyrus	17	14 −104 12	33	4.59
	R	Calcarine gyrus	17	22 −78 4	5	3.63
Basal ganglia	L	Putamen		−24 −22 22	18	3.97

STG = superior temporal gyrus; STS = superior temporal sulcus.

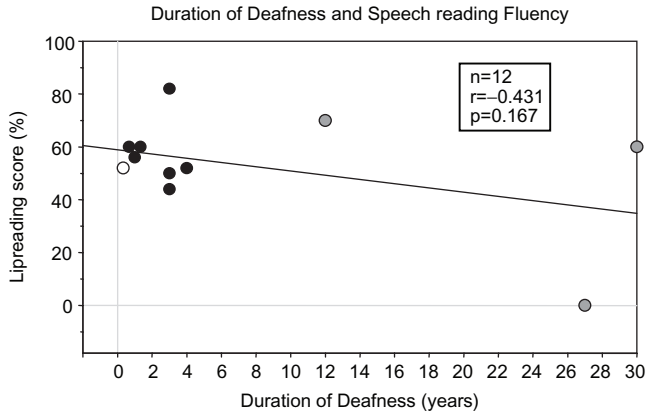

Fig. 1 Relationship between deafness duration at the moment of the fMRI experiment and speechreading fluency, as measured during a clinical test prior to fMRI experiment in 12 post-lingually deaf patients. Black circles indicate those subjects who participated in the fMRI experiment (progressive sensorineural hearing loss). The white circle indicates the patient with abrupt deafness, who was enrolled in the fMRI protocol.

those subjects with longer deafness duration were not the ones with the best speechreading scores. Although all deaf patients had higher scores in our closed set task, their performance in open set showed a larger variance (40–85% among those patients enrolled in the fMRI protocol).

Behavioural effects in deafs and controls

We analysed the fMRI dataset to compare the neural circuits involved in speechreading in post-lingual deaf and normal-hearing subjects, relative to counting the occurrence of gurning faces, a task that controlled for semantic and most of the executive aspects of the speechreading task. By using a closed set of stimuli, we ensured that the task was easy to perform in both groups, and that it entailed no advantage for the deaf group. Accordingly, both groups obtained over 90% correct rate on target trials in both conditions (Fig. 2) and performance showed no statistical difference across groups (Mann–Whitney U-test, $P = 0.345/0.753$ for percent correct trials and $P = 0.294/0.401$ for response time RT in speechreading/counting conditions). However, accuracy was lower and RT longer during speechreading in controls than during counting in both groups (Wilcoxon signed rank test, $P = 0.043/0.106$ for percent correct trials and $P = 0.017/0.012$ for RT in deaf patients/hearing controls). This difference across tasks reflects the difference between a predictable (counting) versus a stimulus-dependent (speechreading) motor output.

Neural activity in deaf patients—cross-modal effects

During speechreading relative to rest, patients and controls activated common areas including bilateral visual cortex, fusiform, middle temporal, angular gyrus, the insula and the lateral prefrontal cortex (Fig. 3), in keeping with previous data (Calvert *et al.*, 1997; Calvert and Campbell, 2003; Hall *et al.*, 2005). When counting gurning faces, activation levels throughout this network dropped in controls but remained elevated in patients (Fig. 3, Table 2), revealing increased automaticity in dynamic face processing, irrespective of whether faces express meaningful speech or not. Enhanced automaticity in deaf patients relative to controls also showed through an increase in basal ganglia (putamen) activity during both tasks (Booth *et al.*, 2007). All moving faces activated regions that classically process auditory speech stimuli (Scott and Johnsrude, 2003), which confirms that auditory deprivation entails reorganization. In addition, we observed a significant task-by-group interaction in the left posterior superior temporal cortex, indicating that speechreading activated this region more than viewing gurning faces, in patients relative to controls (Fig. 4, green spot).

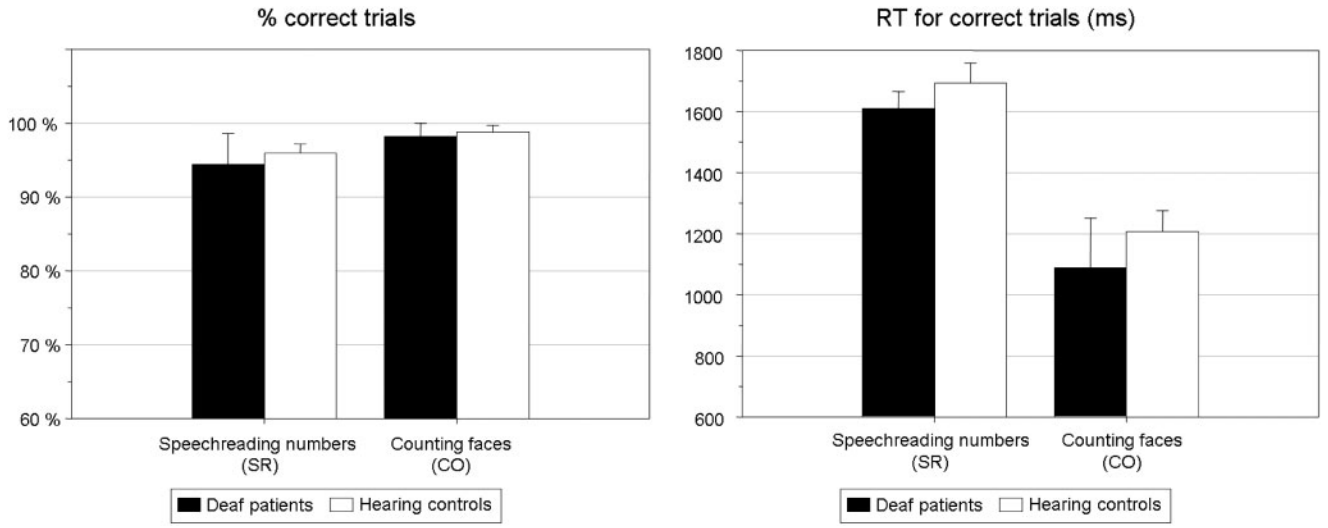

Fig. 2 Behavioural results show a significant effect of condition (speechreading versus counting) except for accuracy in controls ($P = 0.105$), but no group effect. Error bars represent SE.

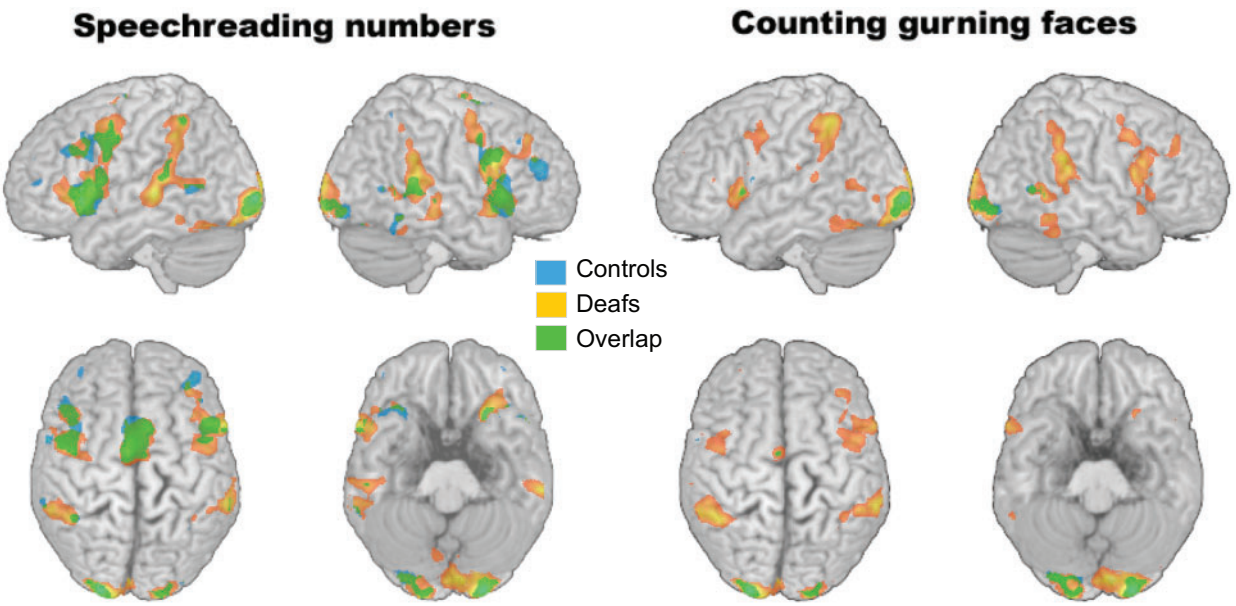

Fig. 3 Main activation effects for speechreading numbers and counting faces in Deafs (orange) and Controls (blue) ($P = 0.05$ FWE-corrected, $T = 5.77$).

Directly contrasting brain responses between deafs and controls in the speechreading condition confirmed that many brain regions shifted their main input modality, i.e. they express a cross-modal effect. Deaf patients had higher activity levels in all auditory association regions including bilateral superior temporal gyrus (STG) and superior temporal sulcus (STS), anterior temporal, visual, ventral prefrontal cortices and bilateral temporoparietal junctions, extending to the inferior parietal lobule on the left side (Fig. 4, Table 3). The left posterior superior and the right anterior temporal cortices were activated more during speechreading than gurning (Fig. 4a and e), but only the left posterior superior temporal cortex (−40, −38, 12) was consistently more active in each single deaf patient relative to the hearing controls (Fig. 4, red spot, and 4b). It was at this location that we also observed a significant group-by-task interaction. Visual cortices (Fig. 4d), bilateral angular gyrus (Fig. 4c, activation profile was similar in both angular gyri) and right prefrontal regions (Fig. 4f) were overactivated in patients during both speechreading numbers and counting gurning faces, indicating that these regions were not directly relevant to speech communication after deafness, but possibly contributed to enhance globally the visual processing of speech sources, i.e. faces (Womelsdorf *et al.*, 2006; Moradi *et al.*, 2007).

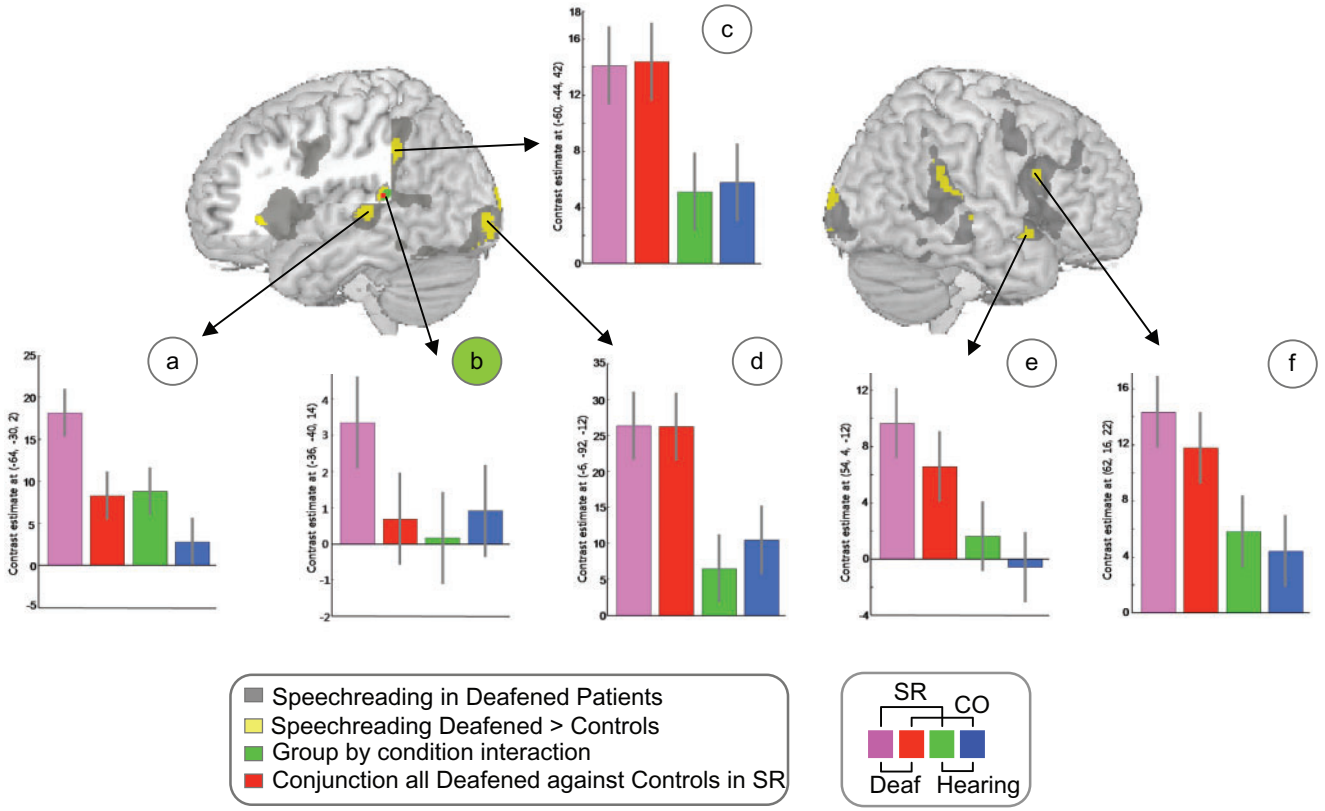

Fig. 4 Brain regions where activity is higher in patients than controls during SR are displayed in yellow over the SR main effect in the deaf group (grey, from Fig. 3) ($P = 0.005$, $T = 2.74$, $k = 20$). Each of the nine individual deaf patients activated the left planum temporale ($-40\ -38\ 12$, red spot) more than all 15 controls during SR relative to CO. Plots **a** to **f** depict the relative effect size across groups and conditions in those regions where patient exhibit an overactivation relative to controls. Speechreading-specific effects were seen in the left posterior superior temporal gyrus/sulcus (STG/STS, plots **a** and **b**). Note the group by condition interaction in posterior STG (green spot).

In contrast, hearing participants had higher activations than deaf patients in medial and lateral frontopolar regions (BA46 and BA10), but no interaction with task was observed.

Multifactorial analysis

Regions where the activity level during speechreading varied as a function of (i) deafness duration, and (ii) speechreading fluency measured prior to the fMRI experiment were assessed in a multivariate analysis. The possible influence of age was discarded by including age as a nuisance variable in the analysis. Positive correlation with speechreading fluency was found in bilateral dorsal and ventral prefrontal cortex, and bilateral insula, in bilateral posterior superior temporal cortex with a left predominance, bilateral STS and bilateral visual association areas (with a left predominance), and in the right fusiform region ($40\ -52\ -14$) (Fig. 5). Speechreading activity negatively correlated with deafness duration in bilateral posterior superior temporal regions and visual cortices, in overlap with regions where activity reflected speechreading scores. In addition, deafness duration correlated negatively with activity in the left angular gyrus, the left prefrontal (Broca's area) and bilateral fusiform cortices. Positive correlation with deafness duration was limited to restricted parts of both prefrontal cortices, and the right temporoparietal junction.

As our intention was to assess the contribution of deafness duration and its possible interaction with performance for regions that are cross-modally activated after deafness, we focus on the examination of the multivariate analysis to the subset of regions that were overactivated in deaf patients relative to controls (the yellow network in Fig. 4). In the region of left posterior STG that showed speech-specific effects, we observed a positive correlation with speechreading fluency and a negative correlation with deafness duration (Fig. 5d) that extended posteriorly (Fig. 5e). The same profile was observed in the visual cortex (Fig. 5f), and in the right posterior STG (Fig. 5c, note that the latter was not more activated in patients than controls). This profile hence dominated across the cross-modal network; whereas only the right angular gyrus and Broca's area showed a positive correlation with deafness duration (Fig. 5a and b). Activity in the latter region also correlated positively with speechreading fluency, which suggests progressive compensatory mechanisms that

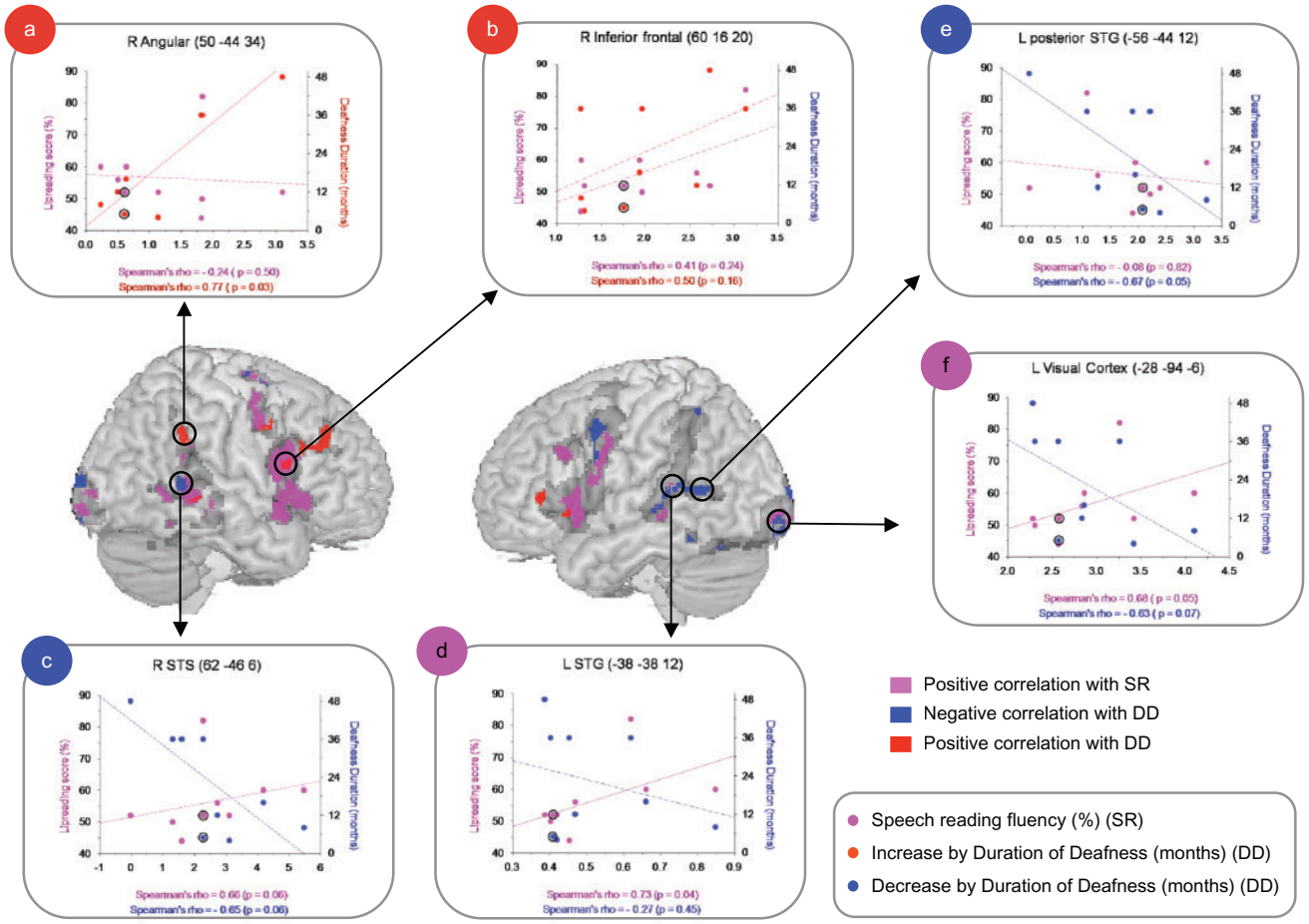

Fig. 5 Results from a multiple regression analysis are displayed on a rendering indicating in grey regions that respond during speechreading in deaf patients (SR main effect in deaf as an inclusive mask set at corrected $P = 0.05$). Regression results are thresholded at uncorrected $P = 0.05$ for display purpose. Pink—Positive correlation with Speechreading fluency. Red—Positive correlation with deafness duration. Blue—Negative correlation with deafness duration. Plots **a** to **f** permit to appreciate speechreading fluency effect in all regions showing an effect of deafness duration (statistic for regressions are indicated below each plot). Circles highlight the only patient with abrupt deafness.

improve visual speech processing. For the angular gyrus, however, the correlation of activity with deafness duration did not appear to be behaviourally relevant as there was no associated correlation with speechreading fluency.

Discussion

Cross-modal reorganization in speech regions

Relative to normal-hearing subjects, deaf patients exhibited enhanced responses to dynamic faces in left STG/STS speech regions. In deaf patients, regions that normally receive predominant auditory input have thus reorganized to process visual input that potentially contains speech information. Neural responses to speechreading in these regions correlated with individual speechreading fluency scores, which confirm previous observations in normal-hearing subjects (Ludman *et al.*, 2000; Hall *et al.*, 2005). In these same regions, we did not detect a positive correlation with deafness duration between 4 and 48 months of sensory deprivation. Even though speechreading does not usually improve with time and effort (Andersson *et al.*, 2001*a*), it could be argued that the strong cognitive and motivational components attached to speechreading in profound deafness should enhance neural plasticity (Blake *et al.*, 2006). We take this negative result to indicate that visual responses in auditory speech regions do not reflect *progressive* takeover of auditory regions by visual input. This interpretation is in line with recent animal studies of adult brain plasticity showing only limited reorganization in visual regions that were deprived of their main sensory input within a time span similar to the one we investigated here (Smirnakis *et al.*, 2005). Our data rather suggest that cross-modal reorganization in speech regions was already present 4 months after deafness onset, which speaks for changes occurring close to deafness onset. Our findings additionally

show that these changes are immediately relevant for human communication.

In keeping with our observations, rapid reorganization following deprivation is observed in the motor cortex of hand amputees (Reilly *et al.*, 2006). In contrast with the classical view that other representations progressively colonize deprived cortical area, EMG response profiles during phantom hand movements (Reilly *et al.*, 2006) indicate that the hand representation immediately *utilizes* the arm motor territory to *express itself*, thanks to pre-existing overlapping/redundant representations. In the domain of audio-visual speech processing, this novel take on cortical plasticity suggests that what we used to interpret as cross-modal reorganization after deafness reflects the opposite tendency of cerebral dynamics, i.e. the resilience of audio-visual speech networks to sensory perturbations. Only the maintenance of stable speech representations, and their access through partially redundant audio-visual connectivity could potentially preserve communication in acquired deafness.

Plasticity of the audio-visual speech network

Audio-visual speech networks are established early in life, and rapidly take an irreversible configuration. Schorr *et al.* (2005) showed that children born deaf and fitted with cochlear implants are susceptible to the McGurk effect if auditory restoration occurs before the age of 30 months. After this age, their perception of audio-visual syllables is visually biased. These findings speak for a sensitive period in audio-visual speech, after which auditory input can no longer be fused with visual input, with the positive consequence that the established network becomes resilient to subsequent sensory damage. In adults, speechreading performance varies widely across individuals, and neither explicit training nor visual communication demands appear to greatly enhance performance (Bernstein *et al.*, 2000, 2001, Andersson *et al.*, 2001*b*; Summerfield, 1992). Given that speechreading ability is grounded in audio-visual binding, and that visual phonological forms are ambiguous by nature, the possibilities to improve speechreading in a unimodal visual setting are limited. Disambiguation of visual phonological forms thus requires additional sensory information, i.e. added hand gestures as in Cued Speech (Charlier and Leybaert, 2000).

If deafness occurs after such a critical period for learning audio-visual associations, phonological memory will progressively deteriorate (Lyxell *et al.*, 1994). This process is slow and the effect subtle, thus preserving the capacity for re-learning audio-visual matching if hearing is subsequently restored by cochlear implant. The more intact the pre-established audio-visual phonological network remains during deafness, the more immediate the efficiency of oral speech comprehension in case of reafferentation. Previous functional neuroimaging data in cochlear implantees support this idea by showing that progressive increase in auditory and visual responses to speech over years after auditory restoration parallels an improvement in speechreading performance (Tyler *et al.*, 1995; Giraud *et al.*, 2001). This effect was observed in good implant users only, but not in a larger cohort including patients with all possible speech scores (van Dijk *et al.*, 1999; Rouger *et al.*, 2007). This suggests that either deaf patients with poor auditory speech scores had already very high speechreading skills, which thus cannot further improve after restoration of audio-visual matching, or more likely, that those patients with poor auditory gain had poor phonological representations and poor speechreading performance before deafness, limiting a possible improvement of audio-visual phonological matching and speechreading skills after implantation. It is also probable that those hearing subjects with poor audio-visual fusion are likely to become poor speechreaders in case of acquired deafness. At any rate, the dynamics of reorganization in speech regions that we observed here, is compatible with the idea that reactivity to sensory impairment is both determined and constrained by the latent connectivity that has developed during childhood, and has (moderately) been tuned throughout life.

Underlying audio-visual connectivity of speech regions at the time of deafness

Cross-modal responses to visual speech are underpinned by a robust audio-visual network (Campbell, 1998). Perisylvian temporal regions respond to both auditory and visual inputs, with predominant auditory responses (MacSweeney *et al.*, 2004), indicating that neurons in these regions are at least bimodal. The early effects we detect here as increased responses relative to controls and a negative correlation with deafness duration most probably reflect and express the effective connectivity that is structurally present at the moment of deafness. Eight of the 9 patients enrolled in the fMRI study became deaf due to progressive sensorineural hearing loss (PSNHL). This condition could have slowly modified the audio-visual balance in speech communication. Our results indicate that speech regions express their underlying potential to process visual speech right at the onset of deafness. That this potential has been tuned by increased reliance on vision in the context of PSNHL is possible, and would agree with the principle of inverse effectiveness, which presupposes that multimodal compensation is maximal when one sense is the least effective (Meredith and Stein, 1996). That the only patient with abrupt hearing loss had a lower speechreading score than the other patients with the same deafness duration is compatible with this view (circled data in Fig. 5). To test this hypothesis, we correlated speechreading responses with the duration of progressive hearing loss. However, we did not detect any statistically significant effect, and no subthreshold effect overlapped with the left posterior STG/STS region. It is to be noted that the assessment of the onset of progressive hearing loss is rather unreliable.

Invalidation of this hypothesis would therefore also require studying speechreading in PSNHL at various stages preceding deafness. Our data do not prove an explicit effect of PSNHL, although this type of aetiology is likely to play a role. They rather point to the resilience of the audio-visual speech network to perturbation of sensory balance. Our findings show that over and above any potential effect of PSNHL, cross-modal reorganisation of speech regions occurs rapidly once auditory input is no longer available, without progressing towards visual colonization of auditory speech regions.

What type of reorganization?

Audio-visual speech processing is underpinned by two possible connectivity principles. The classical view is that auditory and visual inputs converge on the same neural substrate. This indeed could account for many fusion effects, e.g. that visual information from speaker's face enhances speech intelligibility even though lipreading is not necessary for speech comprehension, (Erber, 1969; Summerfield, 1979; MacLeod and Summerfield, 1987; van Wassenhove *et al.*, 2005). This convergence principle, however, lacks the flexibility to account for the more general observation that new multimodal combinations can become rapidly effective even in adults (Sheffert and Olson, 2004; Lehmann and Murray, 2005). Flexible multimodal binding could operate via small networks of distributed sensory representations (Beauchamp, 2005). Thus, even sparse unimodal sensory input, as in speechreading, could tap into a broad range of sensory associations (von Kriegstein *et al.*, 2005, 2006; von Kriegstein and Giraud, 2006). This associative principle would (i) enhance the overall detectability of unimodal events by engaging more cortical substrate, and (ii) increase speed and accuracy of unimodal stimulus identification by eliciting specific predictive loops between association areas.

Regarding plasticity, the distinction between associative loops and association by convergent inputs is important, as reorganization of inputs to a common region should continuously evolve towards complete visual dominance in the absence of competitive auditory input, while reorganization of associative loops would lose efficiency with time in the absence of mutual reinforcement from each input modality. By showing a negative correlation with deafness duration in speech regions, our results are compatible with reorganization of associative loops between auditory and visual regions. These results align with others indicating that efficient functional rearrangements are very likely to occur immediately after sensory deprivation (days to months) in regions displaying complex and far-reaching connectivity, such as speech regions, and that they reflect a reorganization within pre-existing connectivity patterns rather than synaptic rearrangements (Smirnakis, 2005).

Phonological processing after deafness

Although post-lingually deaf patients critically rely on lipreading in everyday communication, our results confirmed that speechreading skills saturate. They do not improve, or even slightly deteriorate over time (Lyxell *et al.*, 1994), which is congruent with the intuition that, in the absence of feedback from speech input, phonological representations progressively fade out (Andersson and Lyxell, 1998). In our fMRI dataset, phonological deterioration could account for the negative correlation of deafness duration with activity in the left posterior temporal cortex (Fig. 5d and e), and in dorsal Broca's area (-52 6 44 $P = 0.001$; Gold and Buckner, 2002). The largest effect in phonological regions was thus observed in those patients who deafened most recently, which underlines the potential of these regions to rapidly readjust their receptive properties. Moreover, it also appears that long-term effects counteract the potential of these regions to access phonological representations from visual inputs. This observation contradicts the classical view that visual inputs should progressively invade regions that are normally under strong auditory influence.

Further findings (beyond the scope of this report) demonstrated a specific deterioration of phonological processing with time elapsed since deafness onset. In 8 of the 9 patients enrolled in the speechreading fMRI experiment, we performed an additional fMRI experiment involving a visual phonological memory task (Supplementary Material). Contrary to speechreading, this visual phonological task did not elicit higher left posterior STG responses in patients than in controls, but a negative correlation between phonology-related neural activity and deafness duration was observed in this region (Wernicke's area and left supramarginal gyrus, Supplementary Fig.; Jacquemot *et al.*, 2003; Vigneau *et al.*, 2006). This effect was associated with increased reaction times and increased activity in other brain regions, which further support that deaf patients progressively lose efficiency in phonological processing and attempt to compensate otherwise.

Long-term global compensatory effects

Together, our results show that after onset of deafness, auditory speech regions rapidly enhance their receptiveness to visual inputs. With progressive duration of deafness, the phonological processing carried out by these regions becomes less and less accurate. Long-term compensation mechanisms might partly counteract degraded phonological representations by enhancing active phonological search and attentional control.

The long-term effects that we detected as positive correlation with deprivation duration are compatible with such compensatory cognitive strategies. These effects were limited and not lateralized to the left hemisphere although the task targeted language. Time-dependent effects were detected in bilateral prefrontal cortex and in the right

angular gyrus. Activity in bilateral inferior prefrontal regions correlated positively with both deafness duration and speechreading fluency, and the right angular gyrus with deafness duration only. The prefrontal cortex integrates inputs from several sensory modalities and controls behaviour (Duncan and Owen, 2000; Koechlin *et al.*, 2003), and the right inferior parietal region is involved in spatial attention (Hillis *et al.*, 2005) and action awareness (Farrer *et al.*, 2007). Both regions are likely to mediate conscious compensatory strategies to palliate a progressive phonological deficit. Yet, activity in the right angular gyrus did not correlate with speechreading fluency, and was equal for speechreading and gurning faces (Fig. 4c). This region might thus govern aspects of the speechreading task that did not directly determine speechreading performance, possibly through global visual task monitoring.

Interestingly, activity levels in these prefrontal and parietal regions increased linearly within the 4–48 months time window of our study without reaching a plateau effect. In other words, these effects had not yet saturated 4 years after deafness onset, and may thus express reorganization for instance via synaptogenesis or other slow but long-term plastic mechanisms (Baker *et al.*, 2005).

Conclusions

Comparison of functional speechreading circuits in hearing controls and deaf subjects delineated brain regions that cross-modally reorganize after auditory deprivation. We did not observe an increase in neural activity in these regions between 4 and 48 months after deafness onset. On the contrary, speech regions showed a progressive decrease in activity with the time elapsed since deafness, and their activity correlated positively but independently with speechreading fluency scores. We take these results to indicate that auditorily deprived speech regions do not undergo progressive cross-modal reorganization, but rather express their underlying potential to respond to visual speech right at the onset of deafness. We propose that cross-modal reorganization relevant to speechreading reflects the configuration of latent multimodal circuits that have been tuned by multisensory experience preceding deafness.

Supplementary Material

Supplementary material is available at *Brain* online.

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