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Conduction aphasia, sensory-motor integration, and phonological short-term memory – An aggregate analysis of lesion and fMRI data

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ABSTRACT

Conduction aphasia is a language disorder characterized by frequent speech errors, impaired verbatim repetition, a deficit in phonological short-term memory, and naming difficulties in the presence of otherwise fluent and grammatical speech output. While traditional models of conduction aphasia have typically implicated white matter pathways, recent advances in lesions reconstruction methodology applied to groups of patients have implicated left temporoparietal zones. Parallel work using functional magnetic resonance imaging (fMRI) has pinpointed a region in the posterior most portion of the left planum temporale, area Spt, which is critical for phonological working memory. Here we show that the region of maximal lesion overlap in a sample of 14 patients with conduction aphasia perfectly circumscribes area Spt, as defined in an aggregate fMRI analysis of 105 subjects performing a phonological working memory task. We provide a review of the evidence supporting the idea that Spt is an interface site for the integration of sensory and vocal tract-related motor representations of complex sound sequences, such as speech and music and show how the symptoms of conduction aphasia can be explained by damage to this system.

1. Introduction

Conduction aphasia, first described in 1874 by Carl Wernicke, is a syndrome that is characterized by frequent phonemic paraphasias (sound-based speech errors) with attempts at self-correction, impaired verbatim repetition, and naming difficulties in the presence of otherwise fluent and grammatical speech output. In contrast to these deficits in speech production, patients with conduction aphasia have relatively spared auditory comprehension (Baldo, Klostermann, & Dronkers, 2008; Benson et al., 1973; Damasio & Damasio, 1980; Goodglass, 1992).

The phonological production deficits in conduction aphasia are linked to articulatory planning load, so that picture naming or repetition of multi-syllabic words, sentences, and phrases is especially impaired (Goodglass, 1992). Due to their relatively preserved auditory comprehension, conduction aphasics are capable of accurately monitoring – and attempting to correct – their own errors in speech output. This self-correcting behavior often results in repeated unsuccessful efforts to correct a phonological speech error (e.g., "baselaw, lacelaw, basecall, casecall ..." for baseball), a behavior that is sometimes referred to as *conduite d'approche* (Goodglass, 1992).

The main symptoms of conduction aphasia point to a deficit at a phonological level of processing. The paraphasic errors made by patients with conduction aphasia are overwhelmingly of a phonemic variety (Bartha & Benke, 2003; Goodglass, 1992), the repetition deficit appears to reflect a deficit in phonological short-term memory (Baldo & Dronkers, 2006; Baldo et al., 2008), and the naming deficit is frequently associated with tip-of-the-tongue states and is benefitted by phonemic cueing (Goodglass, Kaplan, Weintraub, & Ackerman, 1976). Of course, there is considerable syndromic variation among patients with conduction aphasia; however, in the current study, we use the diagnostic label "conduction aphasia" to refer to aphasic patients that share a similar pattern of language deficits that include relatively preserved auditory comprehension in the presence of fluent but paraphasic speech output and dramatically impaired repetition.

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Neurological tradition has attributed conduction aphasia to damage to a white matter tract, the arcuate fasciculus that connects the two major language centers, Wernicke's and Broca's area (Geschwind, 1965). Thus, conduction aphasia has often been referred to as a "disconnection syndrome" because the lesion to the arcuate fasciculus is assumed to interrupt communication between the sensory and motor modules of the classically defined speech language system. More recent evidence, however, indicates that damage to the arcuate fasciculus is not a prerequisite of conduction aphasia (Dronkers, 2000; Shuren et al., 1995). Moreover, electrical stimulation of the left superior temporal gyrus (STG) in an epileptic patient has been shown to induce phonemic paraphasias and auditory-verbal repetition deficits, a finding suggesting that cortical dysfunction alone is sufficient to produce the symptoms of conduction aphasia (Anderson et al., 1999). Finally, most of the available anatomical evidence suggests that conduction aphasia is most often caused by damage to the left superior temporal gyrus and/or the left supramarginal gyrus, that is, a region centered around the posterior portion of the Sylvian fissure (Axer, von Keyserlingk, Berks, & von Keyserlingk, 2001; Baldo & Dronkers, 2006; Damasio & Damasio, 1980; Green & Howes, 1977; Turken et al., 2008).

Functional neuroimaging research in the last 15 years has indicated an important role for this temporoparietal zone in tasks of phonological short-term memory and speech production. Studies of object naming (Hickok et al., 2000; Okada, Smith, Humphries, & Hickok, 2003), single-word repetition (Price et al., 1996), silent reading (Buchsbaum, Olsen, Koch, Kohn, et al., 2005), and covert articulation of syllables (Paus, Perry, Zatorre, Worsley, & Evans, 1996; Wildgruber, Kischka, Ackermann, Klose, & Grodd, 1999) have shown elevated activation in the posterior superior temporal area, even in the absence of auditory input or feedback (e.g. during covert production). Moreover, several studies have shown that during speech production tasks, posterior superior temporal activity is modulated by phonological variables such as word length and frequency (Graves, Grabowski, Mehta, & Gordon, 2007; Graves, Grabowski, Mehta, & Gupta, 2008; Levelt, Praamstra, Meyer, Helenius, & Salmelin, 1998; Okada et al., 2003). Studies examining phonological short-term memory that have used auditory-verbal stimuli and a relatively extended covert maintenance period have consistently shown that a region in the posterior portion of the planum temporale, area Spt (Sylvian-parietal–temporal), activates during both perception (stimulus encoding) and convert rehearsal in tests of phonological memory (for a review, see Buchsbaum and D'Esposito (2008), Chein, Ravizza, and Fiez (2003) and Jacquemot and Scott (2006)). Examination of single subject activations in these studies (Buchsbaum, Hickok, & Humphries, 2001; Hickok, Buchsbaum, Humphries, & Muftuler, 2003) reveals that the area of maximal activity during phonological rehearsal tasks is most often located in planum temporale at the posterior end of the left Sylvian fissure, inferior to the supramarginal gyrus. Finally, the peak Talairach coordinate reported for delay period activity in studies of phonological working memory is nearly identical to the coordinates reported in basic speech production tasks (Buchsbaum, Olsen, Koch, Kohn, et al., 2005; Graves et al., 2007, 2008). In short, Spt activates during the performance of behaviors that are impaired in conduction aphasia (repetition, naming, verbal short-term memory), and is located in an area of cortex often damaged in patients with the disorder.

One hypothesis that has recently been advanced (Hickok & Poeppel, 2004, 2007) is that Spt functions as an interface site for the integration of sensory and vocal tract-related motor representations of complex sound sequences, including speech and music (Hickok, Okada, & Serences, 2009; Pa & Hickok, 2008). According to this hypothesis, conduction aphasia is a deficit that is caused by damage to Spt and surrounding tissue, which disrupts the interaction of sensory and motor systems during speech production and phonological short-term memory. Thus, the phonemic paraphasias and repetition difficulties that characterize conduction aphasia are the result of a damaged sensory-motor integration circuit that leads to an impairment in the capacity for auditory representations of speech to constrain and guide the corresponding articulatory representations thought to be stored in the inferior frontal gyrus and ventral premotor cortex (Hickok & Poeppel, 2000, 2004; Wise et al., 2001). To date, the correspondence between the functionally defined area Spt and the lesion sites associated with conduction aphasia has been indirect, approximate, and anatomically imprecise. With the emergence of methods such as voxel-based lesion mapping, however, and the ability to place functional neuroimaging results in the same normalized anatomical space as lesion data, we now have the ability to directly test whether indeed conduction aphasia involves damage to area Spt.

Simple tests of phonological working memory that involve an unfilled delay period interposed between speech stimulus presentation and a memory test require that a subject can both accurately register and encode an input stimulus sequence and can maintain an internal representation of that code during the delay. We have hypothesized that area Spt is critical for the transformation from an auditory "input" code to an articulatory, or "output", code that must occur during tests of simple repetition as well as phonological working memory. In the context of functional neuroimaging, however, the temporal separation between encoding, maintenance, and response components of the task allows for a separate assessment of brain activation for each phase. Thus, as a practical matter, phonological working memory tasks offer a useful way of isolating regions that are active during stimulus perception, short-term maintenance – or both.

In the present study, we have gathered 105 single subject fMRI scans on a phonological working memory paradigm, collected across five studies and three laboratories, and performed an aggregate analysis on the entire set of statistical activation maps (see Yarkoni, Barch, Gray, Conturo, & Braver, 2009). To compare the spatial pattern of activation in the fMRI phonological working memory analysis with the lesion distribution of conduction aphasia, we performed a conjunction analysis of the aggregate fMRI map with the lesion distribution in stereotaxically normalized space of 14 patients with chronic conduction aphasia (Turken et al., 2008). We predicted and confirmed that the area of maximum lesion probability among patients with conduction aphasia would contain within its bounds area Spt, as defined in fMRI studies of phonological working memory. In the Discussion, we review evidence supporting the idea that conduction aphasia is a disorder of sensory-motor integration that is caused by damage to area Spt.

2. Methods

2.1. Aggregate fMRI analysis

The data entered in the aggregate fMRI analysis were taken from five verbal working memory studies carried out by the authors in the last 5 years. The studies were selected because each involved auditory presentation of verbal material followed by a delay period involving verbal rehearsal, and because relevant contrasts for these "encoding" and "delay" phases of the task were available (in non-normalized "native" image space) for each of the 105 individual subjects (age range: 8–42) that took part in the studies. We did not include two studies (Buchsbaum et al., 2001; Hickok et al., 2003) from our laboratories because the data sets were relatively small (13 subjects combined) and because the data had been archived and were not readily available. Aside from these two omissions, we included all studies that were

carried out in our collective laboratories and met our inclusion criteria (further detailed below).

Two of these studies have been previously published (Buchsbaum, Olsen, Koch, & Berman, 2005; Hickok et al., 2009), and three others (Finn et al., in preparation; Buchsbaum et al., submitted for publication; Buchsbaum et al., in preparation) are currently unpublished (see Table 1). Each of the studies used a similar task paradigm and shared the following features: (1) auditory presentation of phonological stimuli during a short stimulus encoding period, (2) at least an 8 s delay period in which subjects were instructed to covertly rehearse to the to-be-remembered items, and (3) a response phase in which memory for the presented items were tested via serial recall or a recognition memory probe requiring a yes/no response. In each study, then, an estimate of both stimulus-related and maintenance-related activation during phonological working memory could be assessed by separately modeling the encoding and delay phases of the trial (see Fig. 1). For more detail on the task and scanner parameters for the studies, see Supplementary appendix 1.

The analysis of delay-period activation was performed on the contrast maps representing the difference in activation during the retention interval and a baseline estimate of BOLD activity. In each of the five studies, the full trial was modeled with a set of temporally shifted hemodynamic regressors (see Fig 1.), one for each phase (encoding, delay, recall/probe). The activation magnitude for any of the three phases corresponds to the beta estimate for the appropriate term in the multiple regression model. These raw magnitude estimates of the activity in each phase of the trial were then assessed for statistical significance using one-tailed *t*-tests. In each study entering the analysis, the first level (i.e. within-study) statistical analyses were computed in native (non-spatially normalized) image space and then were transformed to stereotaxically normalized space (using the MNI template).

To ensure consistency in the method of normalization across studies, where available (93 out 105 subjects), high resolution structural MRIs were normalized to the same stereotaxic reference space using the MNI atlas. A nonlinear volumetric image registration program (FNIRT; http://www.fmrib.ox.ac.uk/fsl/fnirt) was used to warp each subject's MRI to normalized MNI space. The nonlinear registration parameters were then used to transform all of the native space statistical contrast maps to normalized template space. This normalization procedure was applied even for the set contrasts that had come from previously published data sets (Buchsbaum, Olsen, Koch, et al., 2005; Hickok et al., 2009) – which had applied slightly different normalization routines – so as to maximize the degree of cross-study consistency. For the 12 subjects for which the original high-resolution MRIs were not available, a low resolution EPI image was instead used for linear (12 parameter affine model) spatial normalization to the MNI template using the program FLIRT (Jenkinson & Smith, 2001).

Because the studies differed in a variety of ways including the field strength (3T or 4T) of the MRI scanner, the number of trials in the session, the precise imaging sequence used, etc., we did not compute a typical parametric one-sample *t*-statistic to access statistical significance of the contrast at the group level. Instead, for each voxel in normalized space, we counted the number of subjects with a *z*-statistic for the contrast of interest that exceeded 2.05 ($p < 0.01$, one-tailed). The threshold was chosen to be relatively lenient so as to increase sensitivity to smaller effects. Note that this threshold was not used to determine statistical significance at the group level (see resampling procedure described below) but only for the purposes of counting the number of subjects with an above-threshold activation at each voxel. This raw count was then converted to a percentage, and a corrected significance threshold was determined through a permutation-based resampling procedure (Nichols and Holmes, 2002). To derive a null distribution for the expected proportion of activated voxels given the empirical distribution of the *z*-statistics in the set of 105 contrasts, the spatial indices (excluding non-brain voxels) of each of the 105 normalized contrast maps were randomly permuted. The proportion of *z*-statistics exceeding 2.05 was computed for each voxel in the set of spatially permuted contrast maps, and the maximum value across the entire reshuffled volume was recorded. This was repeated for 100 iterations and the threshold for statistical significance was determined to be the 5th largest (for a corrected $p < .05$ alpha) value in the set. Thus, the end result was a whole-brain (or family-wise) correction for multiple comparisons that was based on the empirical distribution of *z*-statistics in the set of 105 contrast maps. Because the empirical distributions were slightly different for the encoding and delay contrasts, this method yielded different thresholds for the two contrasts, namely 37% for the encoding maps and 28% for the delay maps. Thus, a voxel

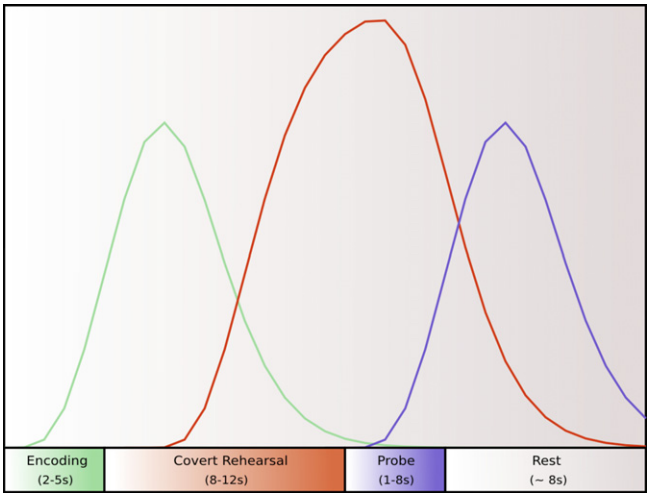

Fig. 1. General task structure and fMRI modeling of Phonological Short-term memory paradigms. Each of the five fMRI tasks included in the aggregate analysis followed the same basic structure: there was an *encoding* phase during which auditry-verbal stimuli were presented; this was followed by a delay period in which subjects were instrcuted to *covertly rehearse* the just-presented items; this was followed by a test phase, in which subjects were instructed to either overtly recall the items in order, or to make a recognition judgment.

Table 1
Basic information about studies that entered fMRI meta-analysis.

Study	*N*	Stimuli	Encoding (s)	Delay (s)	Probe
Buchsbaum, Olsen, Koch, et al. (2005) and Buchsbaum, Olsen, Koch, Kohn, et al. (2005)	12	Two words	4	10	Recall
Buchsbaum et al. (submitted for publication)	10	Six letters	6	10	Recognition
Finn et al. (in preparation)	23	Seven letters	6	8	Recall
Buchsbaum et al. (in preparation)	34	Nine letters	8	12	Recall
Hickok et al. (2009)	22	Jabberwocky sentences	3	12	Oddball detection

Study = the study contributing the fMRI contrast maps; *N* = number of subjects; Stimuli = the type of verbal stimuli used in working memory task; Encoding = the duration of the stimulus encoding phase; Delay = the duration of the delay phase; Probe = the manner in which memory was tested following the delay period.

was declared significant across the group if 37% or 28% of subjects had a *z*-statistic exceeding 2.05 in the encoding and delay contrasts, respectively. A null hypothesis distribution for the conjunction of encoding and delay phases – i.e. the percentage of subjects with significant values ($p < 0.01$) for both encoding and delay phases – was generated using the same resampling scheme and yielded a threshold of 22%. Analyses of the test phase (probe or recall) periods were not performed due to the heterogeneity in the tasks employed and because the main point of theoretical interest was the encoding and delay phases.

2.2. Lesion overlap mapping

2.2.1. Participants

The sample consisted of fourteen patients (4 women and 10 men) who suffered a single, left hemisphere (LH) middle cerebral artery stroke resulting in a chronic conduction aphasia. Patients were selected from a large pool of patients at the Center for Aphasia and Related Disorders, VA Northern California Health Care System (VANCHCS) based on the following criteria: Native English speakers in the chronic phase of stroke (>12 months post-onset), with no pre-morbid history of psychiatric illness, dementia or neurologic illness. The diagnosis of conduction aphasia was made based on the pattern of performance on the Western Aphasia Battery (WAB) (Kertesz, 1982), namely, impaired repetition (0–69% correct), moderate to preserved fluency (50–100% correct), and relatively good comprehension (70–100% correct). Spontaneous speech and naming errors in this group were primarily phonemic in nature. Patients' overall languages scores on the WAB ranged from 40 to 88 (out of 100), which were all below the cut-off for normal language performance (i.e., all patients were aphasic). Patients' mean age was 61.6 (SD = 9.3; range 48–77); mean education was 13.0 (SD = 3.7; range = 5–18); and number of months post-stroke was 56.1 (SD = 55.8; range = 13–247). See Table 2 for performance scores for each subject on the subtests of the WAB. All patients were pre-morbidly right-handed, except for one patient who was left-handed and one patient who reported being ambidextrous.

2.4. Lesion overlay analysis

Patient lesions were reconstructed from CT and MRI scans acquired around the time of testing. For cases where digital MRI images were available ($N = 7$), lesions were traced directly onto T1 scans using MRIcro software (Rorden & Brett, 2000). A neurologist, who was blind to the patients' diagnoses and goals of the study, reviewed the lesion reconstructions for accuracy. Next, the scans were non-linearly transformed into MNI space (MNI-152 template) using SPM5 running on Matlab software (Mathworks, Natick, MA). Lesion masks were used for each reconstruction so that the SPM normalization procedure was not affected by the presence of the lesion (i.e., cost function masking) (see Brett, Leff, Rorden, & Ashburner, 2001). When digital images were not available ($N = 7$), the same board-certified neurologist reconstructed the lesions onto an 11-slice template based on the atlas of DeArmond et al. (see Friedrich, Egly, Rafal, & Beck, 1998; Knight, Scabini, Woods, & Clayworth, 1988). These 11-slice reconstructions were digitized and then non-linearly transformed into MNI space using SPM5. To do this, the two templates were aligned using 50 control point pairs to match anatomical features. The slices were then aligned using a local weighted mean transformation implemented by the *cpselect*, *cp2tform* and *imtransform* functions in Matlab 6.5. These algorithms were applied to warp the lesion reconstructions from the 11-slice template into MNI space. All 14 patients' reconstructed lesions were then overlaid to reveal regions of maximal lesion overlap.

3. Results

3.1. Aggregate fMRI analysis

As can be seen in Fig. 2, activation during both the encoding and delay periods was most prominent in frontal, lateral temporal, and parietal cortices, bilaterally. During encoding, almost the entire extent of the auditory and multisensory cortex of the bilateral superior temporal lobe was active. During rehearsal, however, activity in the superior temporal area was confined only to a few areas, including the left posterior planum temporale (Area Spt), the left posterior superior temporal sulcus (pSTS), and the middle superior temporal sulcus (mSTS), bilaterally. Activation in the frontal cortex during both encoding and delay was most robust in the left hemisphere along the dorso-ventral axis comprising premotor cortex, the inferior frontal gyrus, and the anterior insula (see Fig. 2). The supplementary motor area on the medial wall and superior parietal cortex, bilaterally, were also reliably active during rehearsal. The single most consistent region of rehearsal-related activity in the brain, with 77% of subjects showing a significant effect in the peak voxel (MNI: $x = 51$, $y = -12$, $z = 41$), was located in the dorsal portion of the precentral gyrus.

Table 2
Characterization of individuals with conduction aphasia.

Subject	AQ	Rep.	Naming	Comp.	Digit span	Word span single syll.	Word span multi syll.
1063	75	56	72	78	2	2	–
0806	40	9	2	70	0	0	0
1138	65	16	68	80	2	1	0
1133	70	56	76	90	3	1	1
1015	67	49	80	84	2	2	2
1137	73	51	83	89	–	–	–
0736	74	48	64	98	2	2	2
0626	88	61	79	98	2	4	4
0694	83	64	82	96	3	2	3
0822	67	52	36	88	3	2	2
0718	74	64	79	84	2	2	2
0820	72	49	71	82	2	2	2
0639	80	58	87	82	–	–	–
0655	78	53	76	82	–	–	–

Notes. AQ = aphasia quotient (out of 100); Rep. = WAB repetition percentage; Naming = WAB naming percentage; Comp. = WAB auditory comprehension percentage; Word Span Single Syll. = maximum number of single-syllable words repeated aloud; Word Span Multi Syll. = maximum number of multi-syllabic words repeated aloud; – = data not available.

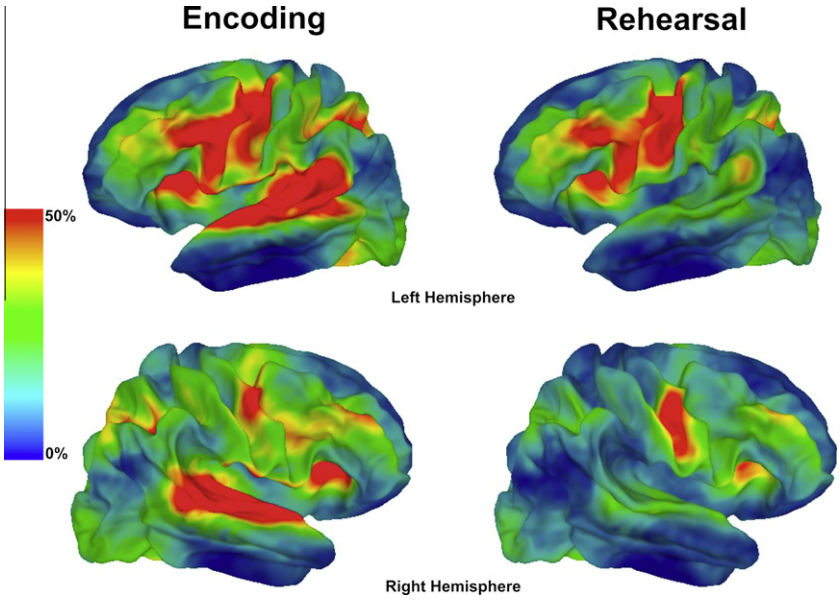

Fig. 2. Proportion of subjects showing reliable activation during encoding and delay phases. Each of the four brain surfaces shows the percentage of subjects showing significant activity ($p < 0.01$ at single subject level) for the either encoding > baseline or the delay > baseline contrasts. These images are unthresholded so that entire cortical pattern can be visualized. Whole-brain thresholds corrected for multiple comparisons were 37% (for encoding) and 28% (for delay).

3.2. Conjunction of encoding and delay contrasts

To examine regions that were active both during stimulus encoding and verbal memory maintenance, we performed a conjunction analysis between the delay and encoding meta-contrasts. The purpose of this contrast was to distinguish regions that are active both during auditory-perceptual encoding and during covert rehearsal from those that are active exclusively in one of the two task phases (e.g. pure sensory or pure rehearsal-related areas). We have previously used this contrast to identify area Spt (e.g. Hickok et al., 2003), which activates robustly both to auditory input and during covert rehearsal. Thus, if Spt has sensory-motor properties it should therefore show robust activity both during auditory-verbal perception *and* during covert speech or covert rehearsal.

As can be seen in Fig. 3, a number of regions were active during both task phases, including dorsal and ventral portions of the prefrontal cortex the intraparietal sulcus, STS bilaterally, and area Spt in the left planum temporale (see Table 3 for MNI coordinates of activated clusters; maximum coordinate: −51, −43, 20). Neither right nor left auditory cortex was reliably activated during rehearsal, a finding that is consistent with previous reports (e.g. Buchsbaum, Olsen, Koch, et al., 2005; Hickok et al., 2003). In addition, while area Spt and a region in the posterior STS were

Table 3
MNI coordinates of activated clusters for encoding ∩ delay conjunction.

Cluster size	Max%	*X*	*Y*	*Z*	Label
1701	69	−51	−9	42	Precentral sulcus
580	46	−30	−54	39	Intraparietal sulcus
529	62	0	9	51	Supplementary motor area
503	43	27	−63	−21	Cerebellum
467	33	57	−6	42	Precentral sulcus
255	44	45	−36	45	Intraparietal sulcus
178	33	36	18	6	Anterior insula
103	44	−21	6	0	Putamen
70	33	−51	−42	21	Posterior planum temporale
33	27	−60	−30	3	Superior temporal sulcus
30	29	63	−24	0	Superior temporal sulcus
28	28	30	−3	51	Middle frontal gyrus
9	23	12	6	0	Caudate nucleus

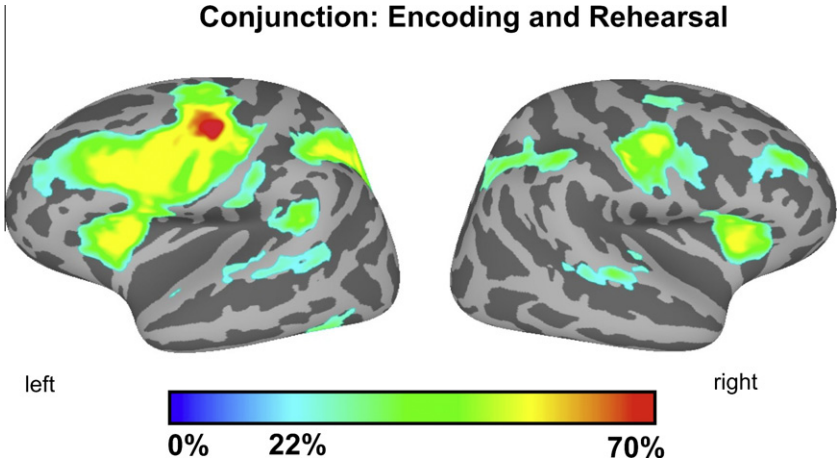

Fig. 3. Proportion of subjects showing reliable activation for the conjunction of encoding and delay phases. Left and right brain inflated surfaces showing areas active both during encoding and during the delay period. The surfaces are thresholded at 22%, corresponding to a whole-brain corrected *p*-value of 0.05.

active only in the left hemisphere in the conjunction analysis, the middle portion of the STS was reliably active in both hemispheres (see Fig. 3).

The peak percentage of overlap in Spt was what might appear to be a relatively modest value of 33% (35 out of 105 subjects). This number, however, reflects to some extent the neuroanatomical variability of the posterior portion of the planum temporale, which is difficult to align across subjects due to individual differences in the trajectory of the Sylvian fissure (Westbury, Zatorre, & Evans, 1999). Visual inspection of single subjects activation reveals a cluster of activity in the posterior planum temporale. To illustrate this, a single slice (in MNI space; $z = 24$) showing single-subject encoding ∩ delay activation for all 14 subjects from study #2 (as numbered in Table 1) is displayed in Supplementary Fig. 1. Each subject's activation is overlaid on the subject's own MRI after nonlinear registration to MNI space. Although all of the 14 subjects have a cluster of activation in the posterior planum temporale, the peak value in the group overlap map is only 50%. Thus, even with 100% of subjects in a group showing activation in the immediate vicinity of planum temporale, anatomical variation (as well as variation in the mapping between function and structure) is sufficiently large that it cannot be fully corrected by nonlinear registration.

3.3. Comparison with conduction aphasia

To directly compare the area of activation associated with phonological working memory in the temporal lobe with the distribution of lesions in conduction aphasia (Baldo et al., 2008), the fMRI conjunction (encoding + delay) analysis was compared with a lesion overlay map based on 14 patients with chronic conduction aphasia. In the left two panels of Fig. 4, the two overlap maps are shown on a left hemisphere cortical surface. The region most often compromised in this group of patients with conduction aphasia was centered in left temporoparietal cortex, in an area encompassing parts of the supramarginal and angular gyri as well as the posterior superior temporal cortex. The area of maximum overlap (85% lesion overlap *and* significant encoding/delay fMRI conjunction) between the lesion overlap map and the fMRI conjunction map is displayed in the rightmost panel of Fig. 4, where it can be seen that the peak area of cross-study overlap is located squarely in the posterior portion of the planum temporale (area Spt).

4. Discussion

The main result of our analysis was that the maximum overlap between the distribution of lesions in conduction aphasia and the activated regions in fMRI studies of phonological short-term memory was in the left posterior planum temporale region, area Spt, a site which has been argued to support sensory-motor integration for vocal tract actions (Hickok et al., 2009). In what follows, we will summarize the arguments for Spt as a sensory-motor integration area and discuss how this claim can illuminate the symptom complex of conduction aphasia.

4.1. Sensory-motor integration in area Spt

By sensory-motor integration, we mean the mechanism by which sensory information can be used to guide action. Visually guided reaching/grasping is a canonical example. Visual information about the location and shape of a coffee cup can be used to guide a reach toward and grasp of that cup. To achieve this task, location and shape information from the visual system must be transformed from visual representations into some representation that can inform action. During the action itself, sensory feedback is also critical to ensure accuracy of the movement. Thus sensory-motor integration is critical for motor control (Kawato, 1999; Shadmehr & Krakauer, 2008). In the speech domain, there is unequivocal evidence for sensory involvement of speech production. For example, delayed auditory feedback of one's own voice disrupts speech fluency (Stuart, Kalinowski, Rastatter, & Lynch, 2002; Yates, 1963). Other forms of altered speech feedback have similar effects: shifting the pitch or first formant (frequency band of speech) in the auditory feedback of a speaker results in rapid compensatory modulation of speech output (Burnett, Senner, & Larson, 1997; Houde & Jordan, 1998). Altered auditory feedback has been found to activate the posterior planum temporal region relative to unaltered speech (Tourville, Reilly, & Guenther, 2008), suggesting that Spt is involved in this form of sensory-motor integration. Levelt (1983) has also documented the importance of feedback monitoring in speech production.

The functional properties of sensory-motor systems have been studied extensively in the context of the visual system. For example, it has been found that in the parietal lobe of the macaque, the intraparietal sulcus (IPS) contains a constellation of functional regions that support sensory-motor integration (Andersen, Snyder, Bradley, & Xing, 1997; Colby & Goldberg, 1999; Grefkes & Fink, 2005). These regions are organized around motor effector systems (for a recent review see (Grefkes & Fink, 2005).

Spt has been shown to exhibit functional properties characteristic of sensory-motor areas in the macaque IPS. Spt shows sensory-motor response properties responding both during the perception and production of speech (Buchsbaum, Olsen, Koch, Kohn, et al., 2005; Buchsbaum, Olsen, Koch, et al., 2005;

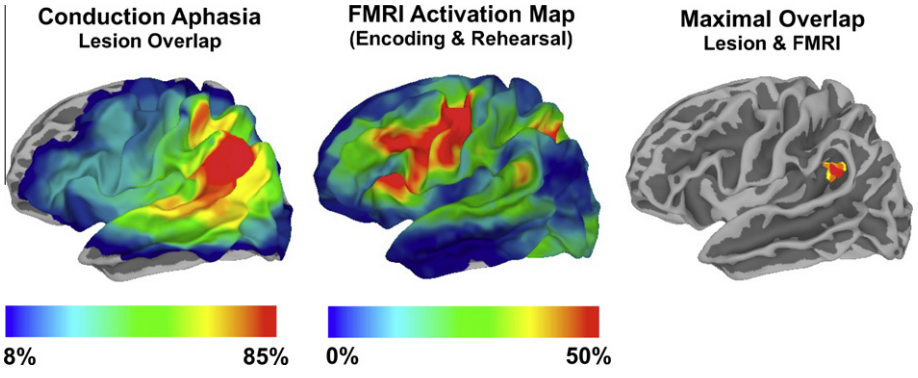

Fig. 4. A comparison of conduction aphasia, phonological working memory in fMRI, and their overlap. The uninflated surface in the left panel shows the regional distribution lesion overlap in patients with conduction aphasia (max is 12/14 or 85% overlap). Middle panel shows the unthresholded conjunction of encoding and delay maps in the aggregate fMRI analysis (see Fig. 3 for thresholded and inflated view of the same data). The right panel shows the area of maximal overlap between the lesion and fMRI surfaces (lesion >85% overlap and significant fMRI activity for conjunction of encoding and delay).

Buchsbaum et al., 2001; Hickok et al., 2003) as well as for non-speech vocal tract actions such as humming melodic stimuli (Hickok et al., 2003; Pa & Hickok, 2008). A recent study has demonstrated that the pattern of activity across voxels in Spt is different during the sensory and motor phase of such tasks indicating partially distinct populations of cells, some sensory-weighted and some motor-weighted (Hickok et al., 2009). A similar distribution of cell types has been found in monkey IPS sensory-motor areas (Grefkes & Fink, 2005). Spt activity is tightly correlated with activity in frontal speech-production related areas, such as the pars opercularis (BA 44) (Buchsbaum et al., 2001) suggesting that the two regions are functionally connected. Spt activity is motor-effector selective, responding with greater magnitude when the output task involves the vocal tract compared to the manual effectors (Pa & Hickok, 2008). Finally, Spt includes cortex on the posterior planum temporale. The planum temporale is often considered an auditory region. However, human cytoarchitectonic studies (Galaburda & Sanides, 1980) and comparative studies in monkeys (Smiley et al., 2007) indicate that the posterior PT region is not part of unimodal auditory cortex. Consistent with this, recent functional studies have found that Spt responds also to visual input that is relevant to vocal tract actions such as visual speech (lip reading) (Okada & Hickok, 2009). Taken together, this constitutes strong evidence for Spt as a sensory-motor integration area for the vocal tract.

4.2. Sensory-motor integration and conduction aphasia

It has been proposed that deficits in repetition and speech production typical of conduction aphasia may be viewed as a sensory-motor integration deficit resulting from damage to area Spt (Hickok & Poeppel, 2000, 2004; Hickok et al., 2003). This hypothesis explains paraphasic speech output fairly straightforwardly on the assumption that speech production relies on auditory guidance (e.g., auditory feedback control) at least under some circumstances. It has already been established that the auditory system guides speech production rapidly and automatically in cases of altered auditory feedback at the phonetic level (an example of auditory guidance from self-monitoring). It is also clear that higher-level auditory information (e.g., sequences of sounds) can guide speech output. For example, it is trivial for healthy individuals to listen to and then parrot back a sequence of nonsense syllables (e.g., ba-da-ga) or even a non-speech stimulus such as a novel melody or sequence of tones (an example of auditory guidance from external input). Because nonsense syllables or tones are not represented in conceptual-semantic memory, the repetition of such sequences requires some form of auditory-motor interface capable of translating a sequence of sounds into a sequence of motor commands. In short, it is clear that at least under some circumstances, including both self- and other-generated speech, acoustic information guides speech production.

Conduction aphasics typically have difficulty with verbatim repetition of speech, and this difficulty is exacerbated by length and decreased familiarity and/or decreased semantic constraint (such as "He is the one who did it", or nonwords) (Goodglass, 1992). These situations require the patient to rely more extensively on the auditory-phonological trace for successful repetition (Howard & Nickels, 2005). If Spt is damaged, the auditory-motor translation system will be dysfunctional, leading to more errors in situations that require increased reliance on the auditory trace of the input. Indeed a recent study found precisely that conduction aphasics fail in situations that require retention of the auditory-phonological trace of a perceived utterance (Baldo et al., 2008). Several case studies have also been presented in which patients with conduction aphasia-like symptoms have disproportionate difficulty in repeating nonwords relative to intact speech perception and relatively preserved speech production (some errors were noted as is typical in conduction aphasia) (Howard & Nickels, 2005; Jacquemot, Dupoux, & Bachoud-Levi, 2007). This pattern of sparing and loss was interpreted in one report as evidence for a disconnection between phonological input and output codes (Jacquemot et al., 2007), which is in line with our proposed explanation of conduction aphasia. Interestingly, there is also evidence that conduction aphasics are less affected by delayed auditory feedback than controls as would be expected if the repetition deficit results from disruption of sensory-motor integration systems (Boller, Vrtunski, Kim, & Mack, 1978).

This explains the repetition deficit, but what about paraphasias in spontaneous speech output where there is not a sensory trace to maintain? There is reason to believe that speakers rely to some extent on an auditory-phonological memory of words they are attempting to produce, as Wernicke proposed, or in modern motor control terminology that the targets of a motor speech act are auditory in nature as argued explicitly by Guenther, Hampson, and Johnson (1998). If motor control of speech production is driven by auditory speech targets and if the link between auditory and motor systems is disrupted, one expects an increase in the error rate in speech production, i.e., paraphasias in spontaneous speech. Further, one expects that the error rate would increase as a function of processing load as is the case with conduction aphasics. Consistent with this, functional imaging studies have shown word length and word frequency effects in auditory-related areas, including Spt and STS, during speech production: longer or lower frequency words yield greater activation than shorter or higher frequency words in picture naming tasks (Graves et al., 2007; Okada et al., 2003; Wilson, Isenberg, & Hickok, 2009). Thus the present model provides an explanation for phonemic paraphasias in speech production.

A prevalent view in the literature is that conduction aphasics have a phonological short-term memory deficit (Baldo et al., 2008; Caramazza, Basili, Koller, & Berndt, 1981; Shallice & Warrington, 1977), specifically affecting the *phonological store* component (Baddeley, 1992). This component is assumed to be a temporary store for phonological information that is distinct from auditory-phonological processing systems involved in speech comprehension. The contents of the phonological store can be kept active via articulatory rehearsal. The phonological store, together with the articulatory rehearsal mechanism, constitutes the *phonological loop* (Fig. 5A). This model explains not only the poor repetition via disruption of the phonological store, but also the preserved

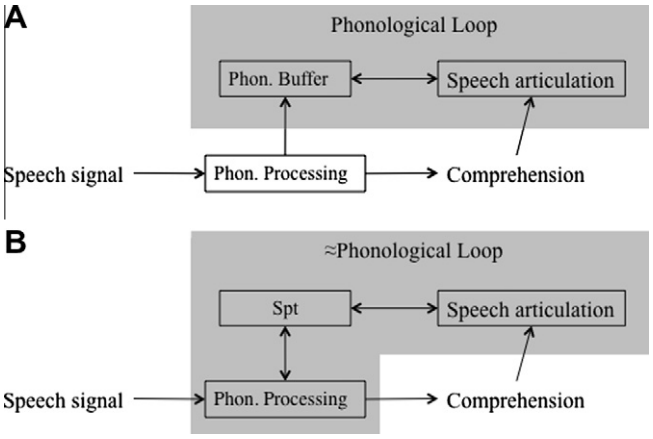

Fig. 5. Schemetic diagram showing the role of Spt in the phonological loop. (A) A diagram showing the architecture of the phonological loop as typically presented (e.g. Baddeley, 1992). (B) Reinterpretation of the phonological loop as emerging from sensorimotor interaction between perceptual and motor speech centers, which is mediated by Spt.

comprehension via the preserved auditory-phonological processing system. The sensory-motor account is not incompatible with this view. In fact it promotes a similar architecture, although with different functional operations associated with the various architectural components.

According to the sensory-motor account, phonological short-term memory is an emergent property of the sensory-motor circuit (Fig. 5B) (Buchsbaum & D'Esposito, 2008; Buchsbaum, Olsen, Koch, et al., 2005; Hickok & Poeppel, 2007; Hickok et al., 2000, 2003; Postle, 2006). On this view, the articulatory rehearsal mechanism is the same as in the phonological loop model, but the "phonological store" is not a distinct, specialized buffer, but the same auditory-phonological system that is involved in processing speech for comprehension. A recent large-scale lesion study ($N = 210$) has provided evidence for this claim by demonstrating an association between STM function (digit span) and lesions involving the left STG/STS (Leff et al., 2009). In addition, the linkage between sensory and motor components is mediated by a sensory-motor interface component. The functional deficit in conduction aphasia, then, involves damage to this sensory-motor interface, i.e., area Spt. This model (i) explains the phonological short-term memory deficit because the articulatory mechanism can no longer refresh the contents of the "phonological store" (the auditory-phonological processing system), (ii) explains phonemic paraphasias because auditory-phonological information cannot be used normally to support speech production, and (iii) explains preserved comprehension because auditory-phonological processing systems are not damaged. Thus, the sensory-motor account not only explains phonological short-term memory parsimoniously as an emergent property of a system that is needed independently for speech processing, and links this circuit to a class of sensory-motor circuits known to exist in the primate parietal lobe, but also explains the co-occurrence of phonological STM deficits and phonemic paraphasias in conduction aphasia.

One complication for this view is the existence of a small number of cases in the literature of nominal conduction aphasics ("short-term memory patients" or "repetition conduction aphasics") that have difficulty with repetition and verbal STM and yet have little or no impairment in naming and spontaneous speech production. The first thing to note is that none of the present sample of 14 patients with conduction aphasia could be classified as "repetition conduction aphasics" as all had significant naming impairments and were well below the normal cut-off for performance on a language battery. This is consistent with the fact that only a very small number of patients with "pure" verbal STM deficits in the presence of unimpaired spontaneous speech[1] have been reported in the literature (reviewed in Shallice & Vallar, 1990). The rarity of these STM cases suggests that they reflect an atypical functional organization (or reorganization Nadeau, 2001). One possibility is that repetition conduction aphasia is associated with lesions that involve the lateral superior temporal cortex but spare the more dorsally situated Spt, leading to deficits in memory for acoustic–phonetic material but sparing speech production processes that depend on auditory-motor integration (Buchsbaum, Olsen, Koch, et al., 2005; Leff et al., 2009). To address the functional–anatomical puzzle presented by repetition conduction aphasia, however, it will be necessary to conduct large sample studies that involve a mixture of symptom severity on relevant indices of speech production and STM, combined with high-resolution MRI and fMRI activation paradigms.

Consistent with other recent studies, we found that lesions resulting in conduction aphasia sometimes involve the posterior lateral superior temporal gyrus (STG) and posterior superior temporal sulcus (STS). These regions have also been implicated in phonological processing on the basis of their activation in a range of tasks including speech production (Indefrey & Levelt, 2000, 2004; Price et al., 1996; Wilson et al., 2009), speech perception (Binder et al., 2000; Hickok & Poeppel, 2007; Liebenthal, Binder, Spitzer, Possing, & Medler, 2005; Okada & Hickok, 2006; Scott & Wise, 2004; Vaden, Muftuler, & Hickok, 2010), and auditory-phonological short-term memory (present result and (Buchsbaum, Olsen, Koch, et al., 2005; Hickok et al., 2003)). We suggest that these STG/STS regions correspond to the "phonological processing" system in Fig. 5B. This raises a question: if left STG/STS regions support phonological processing for comprehension and if these same regions are damaged in at least some conduction aphasics, why is speech comprehension preserved in these patients? The answer is that speech recognition is bilaterally organized such that the right STG/STS can support speech sound perception sufficiently well to allow for good comprehension (Hickok & Poeppel, 2004, 2007; Hickok et al., 2000). Evidence for this view comes from chronic stroke studies which indicate that damage to the STG/STS is not associated with substantial phonemic perception deficits in speech recognition tasks (Baker, Blumstein, & Goodglass, 1981; Miceli, Gainotti, Caltagirone, & Masullo, 1980), from acute stroke studies which show that phonemic perception deficits are mild even in acute stages of disruption (Rogalsky, Pitz, Hillis, & Hickok, 2008), and from Wada studies which indicate that even deactivation of the entire left hemisphere fails to substantially impair the ability to distinguish subtle phonemic contrasts in auditory comprehension (Hickok et al., 2008). Thus, damage to auditory-phonological systems in the left hemisphere STG/STS only partially disrupts this bilateral system, leaving comprehension minimally impaired.

Another question raised by the STG/STS involvement in conduction aphasia is whether damage to this region results in distinct deficits compared to damage to Spt. From a functional standpoint, one might expect conduction aphasia-like symptoms to result from damage to either the left hemisphere auditory-phonological systems or to the network (Spt) that interfaces these systems with the articulatory systems (assuming that it is primarily the left hemisphere auditory-phonological networks that interface with the motor system). Damage to either component could produce paraphasic errors and deficits in repetition and phonological STM either because intact auditory-phonological systems cannot interface with the motor system (Spt damage) or because the auditory-phonological system itself – that is, the portion of the system that interfaces with the motor system – is disrupted (STG/STS damage). There is one symptom of conduction aphasia that may distinguish the contributions of these two components: word-finding. Word-finding deficits are common in conduction aphasia and these deficits typically manifest as tip-of-the-tongue-like states (Goodglass et al., 1976), which imply a deficit in accessing phonological forms for production (Vigliocco, Antonini, & Garrett, 1998). There is no obvious reason why a pure sensory-motor integration deficit should cause failures to access phonological forms. That is, one would expect that phonological forms should be activated accurately but that attempts to produce such forms may result in paraphasic errors. Thus, we propose that damage to auditory-phonological networks in the STG/STS is primarily responsible for word-finding deficits in conduction aphasia. There is some suggestive evidence for this view. A study comparing conduction aphasics with supra- versus infra-Sylvian lesions found that infra-Sylvian cases had more difficulty naming than supra-Sylvian cases (Axer et al., 2001). That same study found that infra-Sylvian cases also performed worse on comprehension measures, as one might expect if auditory-phonological systems are partially damaged. Somewhat surprisingly, infra-Sylvian cases were also more impaired on the repetition test; damage to the sensory-motor

[1] One indication of the latter group's rarity is that its representatives are widely known by their initials (e.g. Shallice & Butterworth, 1977; Vallar & Baddeley; 1984).

interface should disrupt repetition at least as much as damage to phonological processing systems. However, the repetition test involved semantically meaningful items that may have caused the effect as follows. Repetition can be achieved via a semantic route as long as subtle semantic distinctions are not required (Baldo et al., 2008). It may be that the supra-Sylvian group was able to rely on the semantic route more effectively than the infra-Sylvian group who had (partial) damage to auditory-phonological networks and slightly depressed comprehension scores. We would predict comparable deficits on a nonword repetition task. Overall, these findings are suggestive of functional differences between STG/STS and Spt in terms of their involvement in the various symptoms of conduction aphasia, but more research is needed with larger sample sizes and more precise localizations to draw any firm conclusions.

It was noted above that Spt is not speech specific, activating equally well when tonal stimuli are involved (Hickok et al., 2003). This is consistent with the claim that Spt supports sensory-motor integration for the vocal tract as the sensory-motor task for the tonal stimuli involved a vocal tract behavior: humming. This finding predicts that conduction aphasics should have deficits not only in repeating speech, but also in "repeating" (humming back) novel melodies or tone sequences. This behavior is not typically assessed, but two studies suggest that conduction aphasics indeed have deficits involving immediate serial recall of tonal stimuli (Gordon, 1983; Strub & Gardner, 1974). One of these even reports that conduction aphasics are worse in immediate recall of binary sequences of tones (e.g., high–low–low–high–high) than recall of binary sequences of digits (e.g., 1–2–2–1–1) (Gordon, 1983).

5. Summary and conclusions

Drawing on data from a range of sources including the present finding, we have argued that conduction aphasia is, in large part, a "dorsal stream disorder" that results from damage to area Spt, a sensory-motor integration area for the vocal tract action system (Hickok & Poeppel, 2004, 2007; Hickok et al., 2003, 2009; Pa & Hickok, 2008). This account of conduction aphasia not only explains the occurrence of phonemic paraphasia and repetition deficit, but also explains the phonological short-term memory deficit in the syndrome (Baldo et al., 2008) on the assumption that short-term memory capacity is an emergent property of sensory-motor circuits (Buchsbaum & D'Esposito, 2008; Hickok et al., 2003; Postle, 2006; Ruchkin, Grafman, Cameron, & Berndt, 2003; Wilson, 2001) rather than a system with a dedicated memory buffer. Partial damage to left hemisphere auditory-phonological processing systems may exacerbate all of these symptoms in some cases and may account for word-finding difficulties. Auditory comprehension is relatively preserved in conduction aphasia due to the bilateral organization of auditory-phonological processing systems (Hickok & Poeppel, 2007; Hickok et al., 2000) or to the complete sparing of these systems when the lesion is restricted the posterior planum temporale/supramarginal gyrus region.

The proposed sensory-motor account of conduction aphasia is very much in the spirit of Wernicke's original proposal which also appeals to auditory-motor disruption in explaining the syndrome. Our modern account however, proposes that auditory-motor interaction is mediated by a cortical network rather than simply a white matter pathway, and appeals to bilateral organization of auditory-phonological processing systems to explain preserved comprehension.

Finally, the proposal that auditory-motor integration for speech and other vocal tract actions is organized similarly to sensory-motor integration systems in other domains situates speech processing networks (and conduction aphasia) in a broader context of research on the cortical organization of sensory and motor systems. The division of sensory processing streams into two broad streams, one with a tight connection to the motor system and the other with only indirect, perhaps semantically-mediated connections to action systems, appears to be an organizational property of cortical sensory systems generally (Hickok & Poeppel, 2007). This result is not only a satisfying example of convergence between traditionally distinct domains of research, but also opens the door to cross-fertilization between fields.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.bandl.2010.12.001.

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