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Pure word deafness and the bilateral processing of the speech code

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Abstract

The analysis of pure word deafness (PWD) suggests that speech perception, construed as the integration of acoustic information to yield representations that enter into the linguistic computational system, (i) is separable in a modular sense from other aspects of auditory cognition and (ii) is mediated by the posterior superior temporal cortex in both hemispheres. PWD data are consistent with neuropsychological and neuroimaging evidence in a manner that suggests that the speech code is analyzed bilaterally. The typical lateralization associated with language processing is a property of the computational system that acts beyond the analysis of the input signal. The hypothesis of the bilateral mediation of the speech code does not imply that both sides execute the same computation. It is proposed that the speech signal is asymmetrically analyzed in the time domain, with left-hemisphere mechanisms preferentially extracting information over shorter (25–50 ms) temporal integration windows and right mechanisms over longer (150–250 ms) windows. © 2001 Cognitive Science Society, Inc. All rights reserved.

Keywords: Speech perception; Neural basis of speech; Imaging; Temporal processing; Hemispheric asymmetry; Lesions; Auditory agnosia

1. Introduction

As is true for the analysis of practically all cognitive systems, research on speech perception illustrates the tension between invoking specialized processing mechanisms and relying on general principles available for processing any auditory pattern, whether generatives

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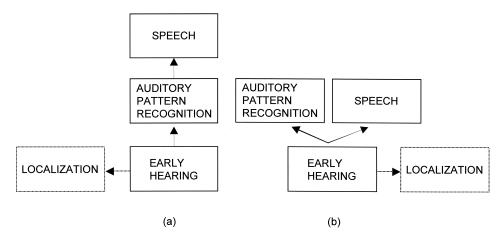


Fig. 1. Two ways in which general auditory pattern recognition and speech perception may differ.

ated by a human vocal tract, a bird, a dump truck, or an electronic function generator. Borrowing from Farah's (Farah, 1995) analysis contrasting object and face recognition, we assume two possible relations between a specialized speech perception system and a generalized auditory object recognition system (Fig. 1). The systems could build on one another such that speech perception is a special case of auditory recognition (a). Speech, on this view, is distinct from generalized auditory processing because it deals with familiar, frequent, 'overlearned' signals. Predictions of this view include (i) that all signals are initially processed by the generalized mechanisms before being elaborated as speech percepts and (ii) that damage to general pattern recognition mechanisms necessarily leads to compromised speech perception.

Alternatively, speech perception and nonspeech recognition may function in parallel (b). On this view, the systems rely on shared mechanisms early in the processing stream but make use of specialized architecture in the construction of the relevant percepts. Moreover, they can be damaged selectively. Whichever cartoon boxology turns out to be (more) right, both proposals are consistent with the idea that the main computational goals for the auditory system are the localization of sounds, the recognition of conspecific vocalizations such as speech, and recognition of other auditory patterns occurring in the acoustic environment. It is argued here that neuropsychological data are more consistent with the view illustrated in (b), a position initially articulated by Liberman and colleagues (e.g., Liberman & Mattingly 1985). Moreover, the neuropsychological (as well as neuroimaging) data support a functional anatomic model of speech perception that implicates both left and right hemispheres, notwithstanding "left-hemisphere imperialism" in the context of speech and language studies. Finally, a model that explores hypotheses about how information is organized in the time domain is argued to lead to a more psychophysically and biologically motivated interpretation of hemispheric symmetry and asymmetry in speech perception.

2. Pure word deafness

Prior to the extensive use of hemodynamic imaging (positron emission tomography - PET, functional magnetic resonance imaging - fMRI) and electromagnetic recording (electroencephalography - EEG, magnetoencephalography - MEG) techniques, the deficit-lesion correlation literature constituted the major source of evidence on the neural basis of speech perception. That body of work has revealed that many different brain lesions and their associated deficits lead to speech perception deficits (Blumstein 1995). For example, Wernicke's aphasics and conduction aphasics are well known for presenting with speech perception problems.

Among the syndromes that are associated with significant speech perceptual problems, pure word deafness (PWD) stands out because of its remarkable specificity. PWD is a rare form of auditory dysfunction in which the integrity of the speech comprehension system is selectively compromised. "Early" hearing such as frequency discrimination (as assessed by audiometry) is intact, but subsequent to a lesion (typically in the posterior superior temporal lobe, bilaterally), the comprehension of spoken language is dramatically impaired. In contrast, the abilities to speak, read, and write remain intact. Crucially, the ability to process nonspeech auditory information, including music, also remains relatively more intact than spoken language comprehension (Fig. 1). What makes word deafness a compelling syndrome, then, is that its existence demonstrates a dissociation between speech perception, nonspeech auditory processing, and central (supramodal) language processing.

The descriptions of the subjective experience of PWD are intriguing. "(Spoken language) is like a great noise all the time . . . you think you can catch it and it fades away, like foreign folks speaking in the distance. . . . When people speak loudly or quickly the words just run together" (Klein & Harper 1956). One patient experienced speech as "meaningless noise, garbled sound, or a foreign language" (Mendez & Rosenberg 1991). The patient studied by Saffran et al. (1976) pointed out that "it's as if there were a bypass somewhere, and my ears were not connected to my voice." These investigators reported that the patient was "unable to describe his auditory experience beyond the frequent complaint that speech sounds did not 'register,' or that they would not 'come up' (Saffran et al. 1976).

To appreciate the relevance of PWD for the functional architecture of auditory cognition, three issues require discussion. First, to what extent are such patients able to process nonspeech information? Second, what is the psychoacoustic or psycholinguistic nature of the deficit? Third, what is the pattern of lesions leading to the deficit?

2.1. The relation of word deafness to other auditory deficits

There are several auditory deficits in which the ability to process *language* remains intact while the ability to analyze *auditory information* is severely compromised. In particular, cortical deafness, auditory agnosia, and PWD are syndromes that have in common that language per se remains intact whereas auditory processing is disturbed to varying degrees. Building on reviews by Buchman et al. (1986), Poeppel (1995), Polster and Rose (1998), and Bauer and Zawacki (2000), Table 1 summarizes the clinical findings that distinguish these three syndromes.

	D
Auditory disorders following cortical	and/or subcortical lesions
Table 1	

	Pure word deafness	Auditory agnosia	Cortical deafness
Speech comprehension	impaired	+	impaired
Speech repetition	impaired	(or mildly impaired) +	impaired
Recognition of familiar non-speech sounds	+	(or mildly impaired) impaired	impaired
Recognition of music	+	+/-	impaired
Hearing sensitivity (audiometry)	+	+	impaired
Language I:	+	+	+
Spontaneous speech Language II: Reading comprehension	+	+	+
Language III: Writing	+	+	+

⁺ indicates adequate performance in a given domain.

The three categories of deficits have in common that patients perform adequately on tasks requiring an intact 'central' (supramodal) language system. In patients with cortical deafness, all aspects of auditory cognition are profoundly impaired. This syndrome is therefore somewhat uninformative with regard to the status of speech perception proper. An important contrast, however, is provided by the deficits categorized as *pure word deafness* versus *auditory agnosia*. The critical dissociation comes from the fact that patients with (nonverbal) auditory agnosia have a relatively more intact speech comprehension system despite their impaired recognition of nonspeech sounds. The comparison of these with PWD patients therefore provides evidence for a neuropsychological double dissociation between recognition of speech and nonspeech sounds.

An interesting special case of auditory agnosia is *amusia*. Amusia (typically associated with right temporal lobe lesions) often occurs independently, that is, the patients perform adequately on speech tasks *and* other auditory pattern recognition tasks but not on tasks requiring pattern recognition in music (Peretz et al. 1994). Again, these observations suggest clear functional segregation in auditory cognition. Taken together, the PWD and auditory agnosia data suggest an architecture in which there exists an initial sound-based representation that is 'stimulus-neutral' and that subsequent to this generic representation there is a segregation into functional streams that are responsible for different types of information. Recent reviews of the neuropsychological literature are consistent with this characterization, and in some cases argue explicitly for modularity in auditory cognition (Polster and Rose 1998).

2.2. Psycholinguistic assessments of word deafness

Several studies have characterized patients' speech perception deficits using psycholinguistic measures (Saffran et al. 1976; Auerbach et al. 1982; Miceli 1982; Tanaka et al. 1987;

Table 2 PWD patients' performance on phonetic tasks

	Consonant ID		Vowel	VOT
	voicing	place	ID	boundaries
Saffran et al.	<<<	<	OK	normal
Auerbach et al.	<	<<<	100%	normal
Miceli	<	<<<<	100%	normal
Tanaka et al.	<<<	<<<	47%	-
Yaqub et al.	<	<<<<	97%	-
Praamstra et al.	<<<<	<<<	43-97%	abnormal

Key: < minor impairment; <<< medium impairment; <<<< severe impairment. ID: identification. VOT: voice onset time judgment.

Yaqub et al. 1988; Praamstra et al. 1991). These studies unfortunately revealed no uniform performance, as illustrated in Table 2; therefore it is not possible to attribute specific phonetic or phonological deficits to the syndrome. However, all patients manifested dramatic speech perceptual disorders despite relatively full competence in speaking, reading, and writing. Note, also, that there was agreement across cases: whereas phoneme identification and discrimination appeared to be severely impaired, consonantal tasks such as voice-onset time (VOT) judgments and place-of-articulation judgments were considerably more affected than vowel-based tasks. In addition, information such as metrical (rhythmic) structure appeared to cause fewer problems for PWD patients. Typically, patients were able to satisfactorily execute tasks such as detecting the number of syllables in a word.

2.3. Lesion patterns in word deafness

Data on PWD range from the earliest case reports in the latter half of the 19th century (Wernicke 1874; Kussmaul 1877; Wernicke and Friedlaender 1883; Freud 1891) to recent imaging studies (Engelien et al. 1995). There are several reviews of the disorder (Buchman et al. 1986; Poeppel 1995; Polster and Rose 1998) and a recent collection of the available literature with review (Griffiths et al. 1999), so only a summary of the lesion patterns will be given here. Sufficient data are available to broadly assess the lesion in at least 59 cases. Of those 59 cases, 42 have bilateral temporal lobe lesions (cortical and subcortical) and 17 have unilateral lesions (mostly subcortical). Of the 17 unilateral lesion cases, 16 are in the left temporal lobe and only one is in the right temporal lobe (Roberts et al. 1987). The pattern of lesions leading to PWD are therefore (a) bilateral lesions in the superior temporal cortex or bilateral lesions affecting the auditory radiations projecting to posterior superior temporal gyrus and (b) left unilateral lesions that compromise the auditory radiations, or the transcallosal projections from the right temporal cortex to left superior temporal cortex. In cases of PWD consequent to unilateral lesions, the lesion site includes left posterior temporal subcortical tissue and, crucially, the lesions interrupt the ipsilateral auditory projections as well as transcallosal projections from the right hemisphere, thereby effectively deafferenting the posterior superior temporal gyrus from inputs from both hemispheres (Takahashi et al.

1992; Poeppel 1995). Thus, the unilateral cases as well as the bilateral cases implicate both hemispheres in auditory speech perception.

Many of the *bilateral* cases share one essential feature: after the first (unilateral) lesion, speech perception was typically intact after an initial period of disturbance. Only after the second lesion (in the other hemisphere) were permanent speech perception problems the consistent outcome. The fact that most of these patients only manifested PWD subsequent to the *second* lesion supports the hypothesis that both sides of the superior temporal gyrus are necessary. Importantly, the temporal order of left and right lesions for a number of cases has been analyzed (Ulrich 1978). No robust pattern has been observed, including the possibility that PWD *only* becomes manifest after a second lesion on the left - in which case one might argue that it is really only left posterior superior temporal gyrus that is implicated. Rather, both left-before-right and right-before-left lesion patterns are attested.

The central point is the finding that in the majority of cases pure word deafness is caused by *bilateral lesions of the superior temporal cortex*, including the posterior superior temporal gyri. The left unilateral cases can be explained as falling into two groups. Characteristic of one group is that the lesion is deep subcortical and affects both left and right STG by compromising the left ipsilateral projections to posterior STG as well as the transcallosal projections from and to left and right STG (Takahashi et al. 1992). Although the lesion is left unilateral, it implicates left and right STG. The second group of left unilateral PWD cases might be associated with lesions that include extensive portions of cortex. In those cases aspects of language comprehension beyond speech perception proper are also compromised.

An important issue concerns the role of primary auditory cortex and its role in PWD. Some researchers have argued that a lesion that includes primary auditory cortex is the necessary precondition for PWD (Coslett et al. 1984; Phillips and Farmer 1990). We disagree with this reading of the literature: primary auditory cortex is not always implicated in the etiology of PWD. A possible model for PWD, then, is that while primary auditory cortex can still be intact, one or more cortical fields in the posterior and lateral aspect of the superior temporal gyrus are compromised (either through direct lesion or underlying white matter lesion). Interestingly, Freud developed a similar proposal in his 1891 *Zur Auffassung der Aphasien*, where he speculated that word deafness is probably a consequence of incomplete bilateral lesions of auditory cortex (Freud 1891).

2.3.1. Are there subtypes of pure word deafness?

Given that there are different types of lesions leading to PWD, one might consider the existence of PWD subtypes. The concept of pure word deafness subtypes has been articulated by Auerbach et al. (1982). Auerbach and others (Yaqub et al. 1988) argued that word deafness exist in two forms, one being a prephonemic temporal processing disorder associated with bilateral temporal lobe lesions and the second a disorder in phonemic discrimination that cannot be attributed to a difficulty in temporal auditory acuity and is associated with left unilateral lesions.

There is some agreement in the literature that this view represents a sensible fractionation of PWD. For example, Albert and Bear (1974) argued that the PWD they attested in their patient was a consequence of a disorder in temporal processing. They found that their patient had a rate-dependent speech perception deficit: when spoken to at an abnormally slow rate,

this patient's understanding improved significantly. In a neurobiologically motivated review, Phillips and Farmer (1990) also argue for PWD as a temporal processing disorder. They point out that speech perception requires intact time-resolution mechanisms in the time domain of milliseconds to tens of milliseconds. Not only does speech perception require temporal sequencing of segments (or syllables) in time, but because critical stimulus information about the identity of speech stimuli is represented on the order of tens of milliseconds (notably formant transitions), a compromised cerebral time resolution mechanism will lead to severely impaired speech recognition.

I propose the following modification of this model. The factors that come into play are (i) whether the lesion is bilateral versus unilateral, (ii) whether the lesion is cortical versus subcortical, and (iii) whether the lesion includes primary auditory cortex or is restricted to posterior STG. In maintaining the central hypothesis that speech sound processing is mediated by the posterior superior temporal gyri in both hemispheres, the predicted patterns are as follows:

- a. If the lesion includes *primary* ("core") auditory cortex and pSTG bilaterally, the disorder will be cortical deafness or auditory agnosia including word deafness. This type of disorder has been called 'prephonemic' and is consistent with the notion of impaired high temporal resolution mechanisms. The word deafness occurring in this case is associated with a deficit in analyzing many types of auditory information.
- b. If the lesion is restricted to *pSTG bilaterally*, the patient presents with PWD. Speech perception is fully compromised and speech sounds are perceived as noise.
- c. If the lesions are *subcortical with cortical sparing* (due to a unilateral or bilateral lesions), the syndrome is the one commonly designated 'disconnection syndrome.' Speech sounds are perceived as belonging to a foreign language. The recognition of speech as speech may be mediated by residual projections (thalamo-cortical or corticocortical) that are still intact, but the access to the areas is severely compromised (deafferenting of pSTG). This would be an instance of associative agnosia.

There does not yet exist sufficient information to distinguish systematically among the possible subtypes and to correlate lesion type with the deficits, so a discussion about possible subtypes must remain speculative. It is clear, however, that predicted subtypes will only be identified with subtle psycholinguistic testing. The major clinical distinction is predicted to be a consequence of whether posterior superior temporal cortex is destroyed bilaterally or not. If pSTG is destroyed bilaterally, speech sound processing is predicted to be profoundly compromised.

The mere existence of PWD is suggestive of some degree of specialization for speech because it is possible to selectively affect speech perception. Of course, the PWD evidence alone does not warrant that conclusion. Although word deafness is reported reasonably often, it is rarely *pure*. Rather, there can be mild but identifiable impairments of nonspeech sound processing. It has been argued that speech perception is relatively more impaired because it is an example of the processing of complex sound patterns (PWD patients often do better with simple auditory tasks such as frequency, intensity, and duration discrimination with tones and nonspeech sounds). Indeed, one might maintain that speech recognition is an instance of pattern recognition of highly overlearned stimuli, and to effectively recognize the

patterns, the integrity of the entire neural system is essential. This view is the one illustrated in Fig. 1a. However, when considering cases of agnosia and amusia (cited above in 2.1), the position for relatively independent processing of speech and nonspeech signals (Fig. 1b) is strengthened. Analogous to some PWD patients, there are agnosic and amusic patients that are successful at low-level tasks while failing at the relevant pattern recognition tasks. To exclude the possible explanation of the documented dissociations in terms of complexity effects one would need a set of studies in which PWD and agnosia patients are tested on items of equal acoustic complexity in the spectral and time domains. While this type of study has not been executed (these patients are rare), the existing evidence suggests that word deafness patients deal effectively with nonspeech signals that are spectrally broadband and complex, including music, and agnosic patients effectively decode speech while failing on various spectrally rich nonspeech tokens (Table 1). In summary, the neuropsychological double dissociation evidence is consistent with a modular functional architecture in auditory cognition (see also Polster & Rose 1998).

3. Supporting evidence for bilaterality: lesions in monkeys and imaging in humans

Experiments on auditory processing in monkeys following unilateral and bilateral ablation of temporal cortex also provide a source of information relevant to the issue of unilateral versus bilateral mediation of auditory processing. While drawing from comparative anatomy can be problematic in the speech domain, some of the central findings are suggestive and converge with the data from PWD.

There is consensus that in macaque monkeys, unilateral superior temporal cortex lesions have relatively small effects on the discrimination of frequency, intensity, duration, or of complex spectral differences. In contrast, bilateral lesions of the same areas have considerable effects on the execution of the same tasks (Elliott & Trahiotis 1972; Iversen & Mishkin 1973; Colombo et al. 1990). Some lesion experiments have directly investigated the neural basis of the auditory processing of complex sounds such as species-specific vocalizations. In one study, squirrel monkeys' ability to discriminate between conspecific vocalizations and nonconspecific but acoustically equally complex sounds (e.g., the vocalizations of other species) was observed (Hupfer et al. 1977). Only medium and large bilateral lesions led to difficulties in executing previously learned discrimination tasks. Importantly, testing after the first (unilateral) lesion, whether it was right or left, revealed only minor deficits. Only subsequent to bilateral lesions were the deficits profound. In one series of experiments, Japanese macaques' ability to discriminate between different types of species-specific coo vocalizations after unilateral and bilateral lesions of the superior temporal cortex was tested (Heffner & Heffner 1984; Heffner & Heffner 1986a; Heffner & Heffner 1986b; Heffner & Heffner 1989). The macaques were tested after unilateral and bilateral ablations. Only following bilateral lesions were the animals permanently impaired on the discrimination of species-specific vocalizations. In summary, the animal lesion data support a model that posits bilateral processing of complex auditory stimuli: only bilateral superior temporal cortex

lesions lead to the permanent inability to discriminate among and process complex auditory signals.

Turning to activation studies, the initial work on 'phonological processing' using PET showed that many different cortical areas were implicated in speech sound processing. However, the interpretation of the findings led to considerable controversy (Demonet et al. 1996; Poeppel 1996). Based on recent evidence obtained by PET, fMRI, and MEG, there is now growing consensus that the prelexical processing of speech implicates both left and right superior temporal cortices. (The extent to which auditory word recognition is also mediated bilaterally is less well understood.) This pattern of data are particularly robust when subjects passively listen to speech—as is typical in spoken language recognition (Poeppel et al. 1996; Hickok & Poeppel 2000; Norris & Wise 2000). Other (nonauditory) areas such as parts of Broca's area (e.g., Brodmann's area 44) and the supramarginal gyrus show activation when subjects are asked to execute tasks that require them to explicitly manipulate certain representations. For example, when subjects make judgments on the segmental content of speech, the left inferior frontal gyrus is implicated (Burton et al. 2000). However, the basic finding that has been reinforced by a range of recent PET and fMRI studies is that both left and right nonprimary auditory cortices are the main areas of activation (Belin et al. 2000; Binder et al. 2000; Mummery et al. 1999; Scott et al. 2000).

Taken together, the PWD data and imaging data from humans (and the animal lesion data) support the hypothesis that complex sound processing, particularly speech sound processing, is carried out by auditory cortex in both hemispheres, not just the presumed 'dominant' hemisphere.

4. Maintaining left-right asymmetries: speech and the time domain

So far it was argued that the substrate for speech perception is the superior temporal gyrus in both hemispheres. Of course, the extensive lateralization that is characteristic of language processing is also well established. The observations discussed here are consistent with the view that *language processing beyond the input signal is lateralized*. In other words, the computations that are part of the speech perceptual interface are mediated bilaterally, but the 'central' computational system that we associate with phonological, morphological, syntactic, and semantic computation, is for the most part, lateralized to the dominant hemisphere.

A question motivated by the model of bilateral speech perception is this: if both hemispheres, specifically both posterior superior temporal gyri, play critical roles in the analysis of speech signals, do both areas mediate the same processes or do they compute different aspects of the acoustic speech signal? It is proposed here that the crucial hemispheric difference derives from the manner in which information relevant to speech is quantized in the time domain.

The hypothesis that is proposed, Asymmetric Sampling in Time (AST), builds on three observations. (1) Psychophysical and physiological results show that information that unfolds over time is "chunked." In particular, *temporal integration windows* provide the logistical framework to organize temporally developing information (for reviews see Yost et al. 1993; Pöppel 1997; Warren 1999). The temporal windows for which there exists a body

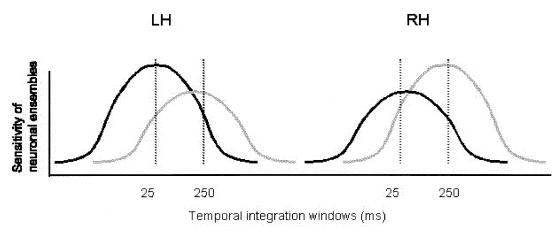
of psychophysical evidence (across methods and sensory systems) have durations on the order of 25–40 ms and 200–300 ms. (2) Neuropsychological and imaging data suggest that left hemisphere mechanisms are suited for the execution of tasks that require high temporal resolution, for example, tasks in which rapidly changing (30–40 ms) acoustic transients must be identified (Nicholls 1996; Johnsrude et al. 1997; Belin et al. 1998). (3) Speech phenomena occur on several different time scales. For example, acoustic-phonetic components such as formant transitions occur over short time scales, say 25–50 ms. In contrast, there are perceptually robust phenomena on the temporal order of syllables (150–250 ms). Previous work on speech perception, for example by Nusbaum and Henly (1992), has suggested that because these time constants are relevant for different kinds of tasks there must be adaptive windows of analysis sensitive to the given perceptual requirements. Similarly, vowels and consonants, which we associate with different time-scales have been shown to be treated differently by the perceptual system (Liberman et al. 1967; Pisoni 1973).

The AST hypothesis (Fig. 2) holds that left nonprimary auditory areas preferentially extract information from short 20-50 ms temporal integration windows. The right hemisphere homologue preferentially extracts information from long 150-250 ms integration windows. By assumption, the input speech signal has a neural representation that is bilaterally symmetric at the primary cortical representational level. Beyond the initial representation, however, the signal is elaborated asymmetrically in the time domain. Another way to characterize AST is to say that the sampling rate of nonprimary auditory areas differs, with LH sampling the spectro-temporal cortical representation built in A1 at high frequencies (~40 Hz; gamma band) and RH at low frequencies (4–10 Hz; theta and alpha bands).

AST exemplifies functional segregation and multiresolution analysis, signal processing strategies common in other domains. Recent work by Ivry and Robertson (1998) and Ivry and Lebby (1998) goes in a similar direction, attempting to account for the lateralization of early perceptual phenomena in speech and vision. In their "double-filtering-by-frequency" (DFF) model each stimulus is initially characterized by it spectral content. In any given perceptual task, the attentional system delimits the relevant spectral range (filter 1). Subsequently, the spectral representation becomes asymmetrically elaborated in the two hemispheres. The spectral point defined by the attentional system acts as a frequency center point around which the information is filtered. High-pass information is passed to left hemisphere areas, low-pass information to the right (filter 2). The model does not posit absolute frequency ranges that are distributed to the two hemispheres but predicts that higher versus lower frequency portions of a stimulus are relative to the attentionally defined point. The AST hypothesis differs from DFF in several ways, most crucially in that the AST model does not assume that there is an initial 'attentional capture' stage in which the relevant parts of the signal are identified. The present model assumes that integration windows of different sizes are simply a part of the architecture of the processing system independently of the attentional system.

Fig. 2 illustrates how to conceptualize how different information types relate to the different proposed timing mechanisms. There are some obvious consequences of the AST proposal: shorter temporal integration windows are associated with higher time resolution but lower spectral resolution; longer windows, in contrast, have lower time but higher spectral resolution. (Importantly, the windows are hypothesized to be temporally based, not category-based, e.g., vowels vs. consonants.) This means that the same input signal will be

a. Physiological lateralization



b. Functional lateralization

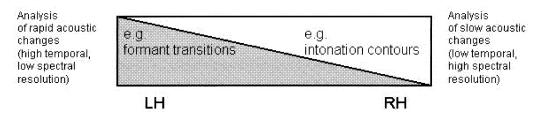


Fig. 2. The upper panel shows possible distributions of neuronal ensembles with different temporal integration times. In the left hemisphere (LH) cell ensembles with a preference for short integration times dominate; vice versa in RH. The lower panel illustrates how such physiological sensitivities may be associated with preferences in processing on or the other type of information.

subjected to two types of analysis that yield complementary information types. Insofar as rapidly changing information is relevant, left cortical regions provide the more appropriate substrate; more gradually changing information or information that requires fine-grained spectral distinctions will be predominantly analyzed by right cortical mechanisms. The AST concept also connects in natural ways to the notion of global versus local processing in that there is a global, lower time-resolution analysis at (~syllabic level) and a local, high temporal resolution analysis (~subsyllabic level).

Empirical predictions for AST include: (1) always bilateral activation in *natural* speech tasks (Norris & Wise 2000; Hickok & Poeppel 2000); (2) linguistic and affective prosody (at the level of intonation contour) should both be associated with right hemisphere mechanisms; (3) the analysis of formant transitions should be left lateralized (Liberman et al. 1967); (4) phonetic phenomena that occur at the level of syllables should be more driven by right hemisphere mechanisms; (5) music perception should lateralize to the right for most musical attributes (including pitch) (Zatorre et al. 1994); (6) rapid FM sweeps should lateralize to the left, slow (e.g., 300ms) sweeps should drive the right temporal lobe; (7) if temporal

integration windows are physiologically reflected as oscillatory brain activity, then the shorter time windows associated with the left hemisphere should yield oscillations in the gamma band that have more power in the left.

Some of the predictions are clearly borne out (e.g., 1, 3, 5), others are controversial (e.g., 2). However, new data from hemodynamic (PET) and electromagnetic (MEG) recordings support some of the specific predictions (e.g., 6 and 7). For example, the connection between temporal integration windows and oscillatory activity is explored in a whole-head MEG study (Poeppel et al. 2000). If activity in specific frequency bands is associated with the functional activation of each hemisphere (e.g., phonetic segmentation in the left vs. prosodic analysis in the right), then the relevant frequency bands might be differentially salient in the two hemispheres during different functional states. We tested this hypothesis in MEG recordings during the presentation of auditory stimuli of varying spectral complexity: ripples (dynamic broadband stimuli). High-frequency responses were robustly different for left and right regions, with gamma activity (25–60 Hz) being more pronounced in left temporal cortex. The data are consistent with the view that sensory input is analyzed on different time-scales by the two hemispheres.

5. Conclusions

Speech perception is the process of extracting information from an acoustic signal and constructing the appropriate representations that can interface with the mental lexicon and the linguistic computational system (Blumstein 1995; Chomsky 1995). If one separates the process of speech perception from further language processes in this way and examines its functional anatomy, deficit-lesion and neuroimaging evidence suggest that the process is mediated bilaterally, in the superior temporal gyri. The compelling lateralization of language processing that we are accustomed to seeing reflects linguistic processing that is independent of the input modality. Insofar as one observes lateralization in the domain of auditory cognition and speech, it reflects the asymmetric treatment of signal in the time domain.

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References

Albert M., & Bear D. (1974). Time to understand: A case study of word deafness with reference to the role of time in auditory comprehension. *Brain*, 97, 373–384.

Auerbach S., Allard T., Naeser M., Alexander M., & Albert M. (1982). Pure word deafness: analysis of a case with bilateral lesions and a defect at the prephonemic level. *Brain*, 105, 271–300.

- Bauer R. M., & Zawacki T. (2000). Auditory agnosia and amusia. In M. J. Farah T. E. Feinberg (Eds.) *Patient-based approaches to cognitive neuroscience* Cambridge MA: MIT Press.
- Belin P., Zatorre R. J., Lafaille P., Ahad P., & Pike B. (2000). Voice-selective areas in human auditory cortex. *Nature*, 403 (6767):309–312.
- Belin P., Zilbovicius M., Crozier S., Thivard L., Fontaine A., Masure M. C., & Samson Y. (1998). Lateralization of speech and auditory temporal processing. *J Cogn Neurosci*, 10, 536–540.
- Binder J. R., Frost J. A., Hammeke T. A., Bellgowan P. S., Springer J. A., Kaufman J. N., & Possing E. T. (2000). Human temporal lobe activation by speech and nonspeech sounds. *Cereb Cortex*, 10, 512–528.
- Blumstein S. (1995). The neurobiology of the sound structure of language. In M Gazzaniga (Ed.), *The Cognitive Neurosciences 1st ed.*. Cambridge, MA: MIT Press.
- Buchman A., Garron D., Trost-Cardamone J., Wichter M., & Schwartz M. (1986). Word deafness: one hundred years later. J Neurol Neurosurg Psychiatry, 49 (5):489–499.
- Burton M. W., Small S., & Blumstein S. E. (2000). The role of segmentation in phonological processing: an fMRI investigation. J Cogn Neurosci, 12, 679–690.
- Chomsky N. (1995). The minimalist program. Cambridge MA: MIT Press.
- Colombo M., D'Amato M., Rodman H., & Gross C. (1990). Auditory association cortex lesions impair auditory short-term memory in monkeys. Science, 247, 336–339.
- Coslett H., Brashear H., & Heilman K. (1984). Pure word deafness after bilateral primary auditory cortex infarcts. *Neurology*, *34*, 347–352.
- Demonet J. F., Fiez J. A., Paulesu E., Petersen S. E., & Zatorre R. J. (1996). PET Studies of Phonological Processing: A Critical Reply to Poeppel. *Brain Lang*, 55 (3):352–379.
- Elliott D., & Trahiotis C. (1972). Cortical lesions and auditory discrimination. *Psychological Bulletin*, 77, (3):198–222.
- Engelien A., Silbersweig D., Stern E., Huber W., Doring W., Frith C., & Frackowiak R. S. (1995). The functional anatomy of recovery from auditory agnosia: A PET study of sound categorization in a neurological patient and normal controls. *Brain*, 118 (Pt 6):1395–1409.
- Farah M. (1995). Dissociable systems for recognition: visual cognition. In S. M. Kosslyn D. N. Osherson (Eds.) *An invitation to cognitive science* Cambridge MA: MIT Press.
- Freud S. (1891). Zur Auffassung der Aphasien. Wien: Deuticke.
- Griffiths T. D., Rees A., & Green G. (1999). Disorders of human complex sound processing. *Neurocase*, 5, 365–378.
- Heffner H., & Heffner R. (1984). Temporal lobe lesions and perception of species-specific vocalizations by macaques. *Science*, 226, 75–76.
- Heffner H., & Heffner R. (1986). Effect of unilateral and bilateral auditory cortex lesions on the discrimination of vocalizations by Japanese macaques. *Journal of Neurophysiology*, 56 (3):683–701.
- Heffner H., & Heffner R. (1986). Hearing loss in Japanese macaques following bilateral auditory cortex lesions. *Journal of Neurophysiology*, 55 (2):256–271.
- Heffner H., & Heffner R. (1989). Cortical deafness cannot account for the inability of Japanese maxaques to discriminate species-specific vocalizations. *Brain and Language*, 36 (2):275–285.
- Hickok G., & Poeppel D. (2000). Towards a functional neuroanatomy of speech perception. Trends Cognitive Sciences, 4, 131–138.
- Hupfer K., Jürgens U., & Ploog D. (1977). The effect of superior temporal lesions on the recognition of species-specific calls in the squirrel monkey. *Experimental Brain Research*, 30, 75–87.
- Iversen S., & Mishkin M. (1973). Comparison of superior temporal and inferior prefrontal lesions on auditory and non-auditory tasks in rhesus monkeys. *Brain Research*, 55, 355–367.
- Ivry R., & Lebby P. (1998). The neurology of consonant perception: specialized module or distributed processors? In M. Beeman C. Chiarello (Eds.) *Right hemisphere language comprehension: perspectives from cognitive neuroscience* (pp. 3–25). Mahwah, NJ: Erlbaum.
- Ivry R. B., & Robertson L. C. (1998). The two sides of perception. Cambridge MA: MIT Press.
- Johnsrude I. S., Zatorre R. J., Milner B. A., & Evans A. C. (1997). Left-hemisphere specialization for the processing of acoustic transients. *Neuroreport*, 8, 61–65.

- Klein R., & Harper J. (1956). The problem of agnosia in light of a case of pure word deafness. *J. Ment. Sci, 102*, 112–120.
- Kussmaul A. (1877). Disturbances of speech. In H. von Ziemssen (Ed.), *Cyclopedia of the practice of medicine* (pp. 581–875). New York: William Wood.
- Liberman A. M., Cooper F. S., Shankweiler D. P., & Studdert-Kennedy M. (1967). Perception of the speech code. *Psychological Review*, 74, 431–461.
- Liberman A. M., & Mattingly I. G. (1985). The motor theory of speech perception revised. *Cognition*, 21, 1–36.
 Mendez M., & Rosenberg S. (1991). Word deafness mistaken for Alzheimer's Disease: differential characteristics. *Journal of the American Geriatrics Society*, 39, 209–211.
- Miceli G. (1982). The processing of speech sounds in a patient with cortical auditory disorder. *Neuropsychologia*, 20, 5–20.
- Mummery C. J., Ashburner J., Scott S. K., & Wise R. J. (1999). Functional neuroimaging of speech perception in six normal and two aphasic subjects. *J Acoust Soc Am*, 106 (1):449–457.
- Nicholls M. (1996). Temporal processing asymmetries between the cerebral hemispheres: evidence and implications. *Laterality*, *1*, 97–137.
- Norris D., & Wise R. (2000). The study of prelexical and lexical processes in comprehension: psycholinguistics and functional neuroimaging. In G. M (Ed.), *The new cognitive neurosciences* Cambridge: MIT Press.
- Nusbaum H. C., & Henly A. S. (1992). Constraint satisfaction, attention, and speech perception: implications for theories of word perception. In M. E. H. Schouten (Ed.), *The auditory processing of speech: from sounds to* words Berlin: Mouton.
- Peretz I., Kolinsky R., Tramo M., Labrecque R., Hublet C., Demeurisse G., & Belleville S. (1994). Functional dissociations following bilateral lesions of auditory cortex. *Brain*, *117*, 1283–1301.
- Phillips D., & Farmer M. (1990). Acquired word deafness, and the temporal grain of sound representation in the primary auditory cortex. *Behavioural Brain Research*, 40, 85–94.
- Pisoni D. B. (1973). Auditory and phonetic memory codes in the discrimination of consonants and vowels. *Perception & Psychophysics*, 13 (2):253–260.
- Poeppel, D. (1995). The neural basis of speech perception. Unpublished Doctoral thesis, MIT, Cambridge
- Poeppel D. (1996). A critical review of PET studies of phonological processing. Brain & Language, 55, 317–351.
- Poeppel, D., Boemio, A., Simon, J., Sauvé, K., Depireux, D., Ribary, U., & Llinas, R. (2000). *High-frequency response asymmetry to auditory stimuli of varying spectral complexity*. Paper presented at the Society for Neuroscience, New Orleans
- Poeppel D., Yellin E., Phillips C., Roberts T. P. L., Rowley H. A., Wexler K., & Marantz A. (1996). Task-induced asymmetry of the auditory evoked M100 neuromagnetic field elicited by speech sounds. *Cognitive Brain Research*, 4, 231–242.
- Polster M. R., & Rose S. B. (1998). Disorders of auditory processing: evidence for modularity in audition. *Cortex*, 34, 47–65.
- Pöppel E. (1997). A hierarchical model of temporal perception. Trends Cognitive Sciences, 1 (2):56–61.
- Praamstra P., Hagoort P., Maassen B., & Crul T. (1991). Word deafness and auditory cortical function: a case history and hypothesis. *Brain*, 114, 1197–1225.
- Roberts M., Sandercock P., & Ghadiali E. (1987). Pure word deafness and unilateral right temporo-parietal lesions: a case report. *J Neurol Neurosurg Psychiatry*, 50 (12):1708–1709.
- Saffran E., Marin O., & Yeni-Komshian G. (1976). An analysis of speech perception in word deafness. *Brain and Language*, 3, 209–228.
- Scott S. K., Blank C. C., Rosen S., & Wise R. J. (2000). Identification of a pathway for intelligible speech in the left temporal lobe. [In Process Citation]. *Brain*, 123 (Pt 12):2400–2406.
- Takahashi N., Kawamura M., Shinotou H., Hirayama K., Kaga K., & Shindo M. (1992). Pure word deafness due to left hemisphere damage. *Cortex*, 28, 295–303.
- Tanaka Y., Yamadori A., & Mori E. (1987). Pure word deafness following bilateral lesions: a psychophysical analysis. *Brain*, 110, 381–403.
- Ulrich G. (1978). Interhemispheric functional relationships in auditory agnosia: an analysis of the preconditions and a conceptual model. *Brain and Language*, 5, 286–300.

Warren R. M. (1999). Auditory perception. Cambridge UK: Cambridge University Press.

Wernicke C. (1874). Der aphasische Symptomenkomplex. Breslau: Cohn and Weigart.

Wernicke C., & Friedlaender C. (1883). Ein Fall von Taubheit in Folge von doppelseitiger Läsion des Schläfenlappens. Fortschritte der Medizin, 1, 177–185.

Yaqub B., Gascon G., Al-Nosha M., & Whitaker H. (1988). Pure word deafness (acquired verbal auditory agnosia) in an Arabic speaking patient. *Brain*, 111, 457–466.

W. A. Yost A. N. Popper R. R. Fay (Eds.) (1993). Human psychophysics New York: Springer.

Zatorre R. J., Evans A. C., & Meyer E. (1994). Neural mechanisms underlying melodic perception and memory for pitch. *J Neurosci*, 14, 1908–1919.