DISORDERS OF AUDITORY PROCESSING: EVIDENCE FOR MODULARITY IN AUDITION

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Abstract

This article examines four disorders of auditory processing that can result from selective brain damage (cortical deafness, pure word deafness, auditory agnosia and phonagnosia) in an effort to derive a plausible functional and neuroanatomical model of audition. The article begins by identifying three possible reasons why models of auditory processing have been slower to emerge than models of visual processing: neuroanatomical differences between the visual and auditory systems, terminological confusions relating to auditory processing disorders, and technical factors that have made auditory stimuli more difficult to study than visual stimuli. The four auditory disorders are then reviewed and current theories of auditory processing considered. Taken together, these disorders suggest a modular architecture analogous to models of visual processing that have been derived from studying neurological patients. Ideas for future research to test modular theory more fully are presented.

Key words: auditory processing, modularity, review

Introduction

Neuropsychological investigations of patients suffering from brain damage have flourished in recent years and helped to produce more detailed and neuroanatomically plausible models of several aspects of cognitive function. For example, models of language processing are often closely aligned with studies of aphasia (e.g., Caplan, 1987; Goodglass, 1993) and models of memory draw heavily upon studies of amnesia (e.g., Schacter and Tulving, 1994; Squire, 1987). Most of this research has relied on visually presented materials, and as a result visual processing disorders tend to be more well-documented and better understood than their auditory counterparts. Indeed, whereas detailed functional and neuroanatomical models have been proposed for visual processing (e.g., Marr, 1980, 1982; Young, 1988; Van Essen, Anderson and Felleman, 1992), similar accounts of auditory processing have not yet been conceived (Peretz, 1993). This general disparity between the sophistication of models of visual and auditory processing can also be observed at a more specific level within the study of language. Thus, for example, disorders associated with visually presented linguistic stimuli (the acquired dyslexias) have been the subject of intensive investigation and have helped make theoretical advances regarding the normal reading process (Patterson, Coltheart and Marshall, 1985; Coltheart, Patterson and Marshall, 1987), but disorders associated with auditorily presented linguistic stimuli (pure word deafness) have been less well investigated and had less impact on theories of normal speech comprehension. The limited research in auditory processing and its close ties to language are apparent from a cursory

inspection of contemporary neuropsychology textbooks, which reveals that several chapters are dedicated to visual processing of objects and faces, but auditory processing is often subsumed in chapters on language processing (e.g., Ellis and Young, 1988; McCarthy and Warrington, 1990).

Despite, or perhaps because of, the limited attention that has been paid to auditory processing, the last few years has seen increased research activity involving the auditory domain (c.f., McAdams and Bigand, 1993). For example, two areas of memory research have begun to focus on the processing of auditory stimuli. In the implicit memory arena, the specificity of priming effects for auditorily presented words (Schacter and Church, 1992, 1995) and environmental sounds (Chiu and Schacter, 1995) has been investigated; and experiments on source memory have used voice as a source of information (e.g., Glisky, Polster and Routhieaux, 1995). In addition, studies of voice recognition have emerged in the neuropsychological (e.g., Van Lancker, Cummings, Kreiman et al., 1988; Van Lancker and Kreiman, 1987) and cognitive (e.g., Bull and Clifford, 1984; Van Wallendael, Surace, Hall Parsons et al., 1994) literatures. Finally, research into variables that affect recognition and identification of sounds has begun to occur (e.g., Ballas and Mullins, 1991; Ballas, 1993), and more detailed examinations of auditory processing disorders have been conducted (e.g., Peretz, 1993; Peretz, Kolinsky, Tramo et al., 1994; Schnider, Benson, Alexander et al., 1994). Given this increased interest in auditory stimuli, it seems timely to review the literature on disorders of auditory processing, and examine theoretical questions regarding the functional architecture of the auditory system. Before presenting the review, however, three possible reasons for the delayed emergence of research on auditory processing – the nature and incidence of auditory impairments, terminological confusions, and technical/methodological limitations – are identified and discussed in turn.

The first factor that appears to have limited cognitive neuropsychological investigations of auditory processing is that disorders of auditory processing are quite rare, especially when compared to the incidence of disturbances in visual processing. There appear to be two related reasons for this disparity in incidence. First, the auditory system has a neuroanatomical redundancy built into it that does not exist in the visual system. Similar to the eye, each ear projects information to both hemispheres. However, unlike the visual system, which has a direct mapping from one half of the visual field to the contralateral visual cortex, the auditory system transmits information about sound in all parts of space to both hemispheres. Although projections to the contralateral hemisphere are more prominent than projections to the ipsilateral hemisphere (Celesia, 1976), the non-specific nature of the ipsilateral connections has two consequences for studying auditory processing. First, bilateral lesions are often required to produce auditory disorders. Indeed, unilateral lesions in auditory cortex are not uncommon, but they may remain undiagnosed because they do not produce significant impairments (Michel, Peronnet and Schott, 1980). Moreover, because auditory cortex is located in the lateral temporal lobes, two separate neurological events are often required to produce bilateral lesions. Second, the generalised ipsilateral connections in the auditory system, as opposed to the defined mappings of contralateral space in the visual system,

make it more difficult to draw firm conclusions from auditory studies that use dichotic listening tasks (see Hugdahl, 1988) than from visual studies that use visual-half field presentation, regardiess of whether commissurotomy patients (e.g., Sparks and Geschwind, 1968; Corballis and Ogden, 1988) or normal subjects (e.g., Carmen and Nachson, 1973; Kimura, 1967) are tested.

A second aspect of auditory processing that might account for the low incidence of selective auditory disorders regards diagnostic difficulty caused by the close relationship between speech comprehension and auditory processing. In cases in which an auditory disorder occurs independently of a language impairment, the disorder may not be clinically conspicuous and thus may remain either unreported by the patient or undiagnosed by the neurologist (Vignolo, 1982). By contrast, when an auditory disorder occurs in conjunction with a language disorder, the auditory impairment may remain undiagnosed, be viewed as secondary to a primary disorder or thought to be unimportant. For example, pure word deafness, has at times been presented as a disorder indistinguishable from the more general language impairment of Wernicke's aphasia (e.g., Wernicke and Friedlander, 1883; Rubens, 1979). In short, the dominance of linguistic processing has overshadowed what may be an important and independent auditory processing impairment.

The second factor that appears to have limited the study of auditory processing is a series of terminological confusions that began soon after auditory processing was first investigated. The first confusion occurred when Wernicke (1874) and Kussmaul (1877) disagreed regarding whether or not auditory language comprehension deficits (i.e., pure word deafness) could occur independently of speech production deficits. Wernicke used the term "sensory aphasia" to encompass an auditory comprehension deficit that he believed was inextricably linked to an impairment in speech production and took exception when Kussmaul (1877) used the phrase "word deafness", suggesting that the comprehension deficit could occur independent of any impairment in production. This debate reflects two aspects of the terminological confusions that have persisted in the literature: (a) the relationship between language and auditory processing; and (b) the use of different terms to describe similar symptoms. Thus, confusion has arisen both from the labels assigned to clinical disorders and from the manner in which the disorders have been defined. For example, the term auditory agnosia has been used in a restrictive sense to refer to defective recognition of nonverbal sounds and noises (e.g., Vignolo, 1969; Bauer, 1993), and in a more general sense to refer to a global impairment in the processing of all types of auditory information (e.g., Buchtel and Stewart, 1989). As a result, patients with the same clinical disorders have not always presented with the same symptoms.

A second, more general confusion has occurred when "verbal processing" has been conflated with "auditory processing", perhaps because the distinction between verbal and visual stimuli has been misappropriately applied to processing domains. Thus, although visual stimuli necessarily require visual presentation, verbal stimuli can be presented and processed either visually (i.e., reading) or auditorily (i.e., speech), or even tactually (i.e. Braille). Because of this conflation, auditory processing has been intimately and unnecessarily tied to

language in a fashion that never occurred with visual processing; instead, verbal stimuli (i.e., printed words) have always been recognised as a specific type of visual information. Recently, it has been recognised that verbal (or linguistic) stimuli represent one type of auditory information, and that non-linguistic sounds represent a different type of auditory information in the same way that pictures and words represent different types of visual information. Even in the case of auditorily presented verbal stimuli, spoken words only represent the linguistic component of an auditory signal that is rich with other information such as the speaker's voice, intonation and affect that can apparently be processed independently of the content of the message (Yaqub, Gascon, Alnosha and Whitaker, 1988; Klein and Harper, 1956; Miceli, 1982; Heilman, Scholes and Watson, 1975). Thus, spoken language includes information about the speaker such as sex, age, region of origin and affective state in the speech signal that either are not part of, or certainly not as easily deduced from, written language. In short, avoiding the conflation between verbal and auditory processing makes it evident that auditory processing encompasses a wider range of stimuli than just spoken words, and may allow independent sub-components of the processing system to be identified.

Technical and methodological factors comprise a third reason for the relative shortage of studies investigating disorders of auditory processing. The conflation between "auditory" and "verbal" processing might suggest that studies of language have focused on the auditory modality, but most research into language processing has used written words because of the relative ease of studying stimuli in the visual domain compared to the auditory domain (Frauenfelder and Tyler, 1987; Pisoni and Luce, 1987). For example, visual stimuli can be presented for varying durations without changing the stimulus, but the duration of auditory stimuli cannot be altered without also changing some important aspect of the stimulus. Similarly, visual stimuli that are presented simultaneously can be identified relatively easily, but simultaneous presentation of auditory stimuli makes it difficult to identify the individual sounds (see Bregman, 1990, 1993, for discussion of "auditory scene analysis"). The ability to process visual stimuli simultaneously also allows meaningful reaction times to be measured, whereas the more serial nature of auditory processing makes reaction times difficult to measure and interpret. Finally, high-quality authentic reproductions of visual stimuli (i.e., photographs) have been readily available for many years, but until recently, recording voices or sounds reduced the quality and authenticity of the stimuli.

Having identified three reasons why neuropsychological investigations of auditory processing have been slow to emerge, it now remains to review the literature on disorders of auditory processing and address theoretical questions regarding the functional organisation of the auditory system. The next section presents brief reviews of four auditory processing disorders – cortical deafness, auditory agnosia, word deafness, and phonagnosia.[1]

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[1]Amusia, or an impairment in musical processing, has generally been construed as an auditory processing disorder (see Critchley and Henson, 1978). However, given that expressive and receptive examples of the disorder that appear to correspond to Broca's and Wernicke's aphasia, respectively, have been reported in the literature (e.g., Polk and Kertesz, 1993), and that music has the characteristics of language (Henson, 1978; Jackendoff and Lerdahl, 1982), it may be better viewed as analogous to aphasic disorders. For these reasons, this review does not consider amusia to be an auditory processing disorder.

Disorders of Auditory Processing

As the previous section indicates, initial interest in auditory processing primarily involved testing for language comprehension deficits in the auditory modality. However, it was not long before descriptions of deficits in other aspects of auditory processing appeared in the literature, and by the end of the 19th century, three of the disorders that will be reviewed in this paper had been identified: cortical deafness (Wernicke and Friedlander, 1883); auditory agnosia (Freud, 1891); and pure word deafness (Bastian, 1897; Kussmaul, 1877; Lichtheim, 1885). Only phonagnosia remained undiscovered until very recently (Van Lancker and Canter, 1982).

These early clinical descriptions of auditory disorders set the foundation for an ongoing debate regarding the underlying nature of the auditory system; that is, whether the disorders reflect quantitative differences in the severity of damage to the auditory processing system or qualitative differences between independent modules of that system that are susceptible to selective impairment. In the former case, the disorders are said to reflect more or less severe examples of impairments to a single auditory processing system that operates serially. For example, Mendez and Geehan (1988) concluded that "the different clinical syndromes resulting from lesions in the cortical auditory areas constitute a spectrum of related auditory processing disorders with similar clinical features and common underlying psychoacoustic abnormalities" (p. 7). By contrast, in the latter case, the disorders would represent impairments in unordered, independent processing modules. For example, Peretz et al. (1994) concluded that "the perception of speech and the perception of nonspeech patterns are subserved by distinct neural systems, hence invalidating the claim that all auditory agnosia result from the disruption of a single general-purpose mechanism" (p. 1295). Although this conclusion is restricted to auditory agnosia, it can be applied to auditory processing in general.

Examining this debate more closely, reveals that it consists of two questions that are often considered together, but that can be addressed independently. There is a neuroanatomical question to be addressed: are the disorders associated with damage to different brain regions? And, there is a psychological question to be addressed: do the disorders reflect the operation of independent processing modules in the auditory system? Because most research into disorders of auditory processing has focused on patients with discrete brain lesions, the anatomical and psychological questions have generally been addressed simultaneously. Disentangling them, however, may enable a clearer picture of the functional architecture of the auditory system to emerge. Therefore, in the following review, a clinical description of each deficit is provided first, followed by sections that address anatomical and theoretical considerations. Because the goal of this paper is to provide a general conceptual framework for understanding disorders of auditory processing, these reviews focus on the main features of each disorder; detailed consideration of specific aspects of each disorder can be found in the review articles cited in each section.

Cortical Deafness

Wernicke and Friedlander (1883) described a patient who was unable to hear any sounds, but had no apparent damage to the hearing apparatus and labelled the disorder cortical deafness. Henschen (1916; cited in Kanshepolsky, Kelly and Wagner, 1973) presented sixteen further cases with post-mortem examination to establish the validity of the disorder, but relatively few cases continue to be reported. One review of the literature, reported "only 12 cases of significant deafness due to purely cerebral pathology" (Graham, Greenwood and Lecky, 1980, p. 43) and suggested that Jerger, Weikers, Sharbrough et al. (1969) were the first to document "unequivocal deafness due to clearly demonstrated cortical lesions with no evidence of a peripheral lesion" (p.45).

Identifying cases of cortical deafness has proved to be difficult for several reasons. First, patients rarely suffer bilateral lesions in the critical region of auditory cortex in the lateral temporal lobes (Clark and Russell, 1938). Second, strict criteria have been adopted to accept that a patient is cortically deaf (Kanshepolsky et al., 1973; Graham et al., 1980). For example, in addition to unawareness of auditory stimuli and abnormal audiograms, it has been proposed that cortical deafness requires an EEG or evoked potential pattern that shows normal brain stem activity but impaired cortical activity (Coslett, Brashear and Heilman, 1984; Rapin, 1985). Additionally, Oppenheimer and Newcombe (1978) suggested that some patients believed to suffer from cortical deafness may, in fact, suffer from auditory inattention or neglect (e.g., Heilman and Valenstein,1972; Beaton and McCarthy, 1993, 1995; Hugdahl, Westert and Asbjørsen, 1991; and Hugdahl and Wester, 1994). Finally, cortical deafness is often transient, resolving to a less severe, or more specific, auditory processing disorder such as pure word deafness or auditory agnosia. For example, Mendez and Geehan (1988) presented a patient who behaved as if he was totally deaf initially, but after two months was able to recognise environmental sounds and after three months was able to recognise music, leaving a residual impairment in auditory word recognition. For all of these reasons, then, the incidence of cortical deafness remains relatively low.

Although the term "cortical deafness" suggests that the impairment must be cortical in origin, there is some evidence that subcortical lesions can produce the same complete loss of hearing (e.g. Clark and Russell, 1938). However, the disorder is most commonly associated with extensive bilateral damage to the auditory cortex in the lateral temporal lobes including Brodmann's areas 41 and 42 that destroys auditory sensory input areas (e.g., Bramwell, 1927; Jerger et al., 1969; Lhermitte et al., 1971; Graham et al., 1980; Tanaka et al., 1991; Earnest et al., 1977). In the case of subcortical lesions, hearing loss appears to result from a disconnection between sensory and perceptual processing. In either instance, the impairment is so complete that it obviates the need for theoretical analysis.

Auditory Agnosia

Because visual agnosia is a deficit restricted to object recognition that dissociates from deficits in processing of written language (see Farah, 1994), by

analogy, auditory agnosia is probably best defined as an inability to recognise auditorily presented sounds independent of any deficit in processing spoken language. This category-specific deficit has at times, however, been replaced by a more general definition in which auditory agnosia refers to a deficit involving any type of auditory stimuli. As a result, the exact boundaries of auditory agnosia seem to be established on a case-by-case basis, and have at times included patients whose disorder encompasses linguistic stimuli (e.g., Buchtel and Stewart, 1989; Goldstein, Brown and Hollander, 1975; Marshall, Rappaport and Garcia-Bunuel, 1985; Oppenheimer and Newcombe, 1978; Reinhold, 1950; Rosati et al., 1982), and other times excluded patients whose disorders encompass linguistic stimuli (Spreen, Benton and Fincham, 1965; Fujii et al., 1990; Vignolo, 1969, 1982). Understandably then, auditory agnosia has been subject to attacks as confusing, and the very existence of the disorder has even been doubted. For example, in a review of the early literature, Vignolo quoted Dejerine that "true auditory agnosia has neither a clinical autonomy nor a definite pathogenesis" and noted that Marie did not even discuss auditory agnosia in a paper on neurological cases with auditory processing disorders (Vignolo, 1969, p. 177). In early neurological accounts, then, auditory agnosia did not share the attention that was paid to visual agnosia (e.g., Lissauer, 1890). Instead, the absence of auditory agnosia in cases of pure word deafness merely served to demonstrate that language could be selectively impaired. By contrast, when disorders in processing nonlinguistic auditory information, were observed, their significance was rarely discussed (Vignolo, 1969).

Although the concept of auditory agnosia dates back to Freud (1891) the first case of 'pure' auditory agnosia appears to have been reported only thirty years ago (Spreen et al., 1965). They presented a patient who was severely impaired at identifying a variety of sounds such as coughing, whistling and a baby crying, but showed no evidence of impaired speech comprehension. Although audiometric testing revealed that the patient suffered moderate loss at high frequencies, there was no evidence that his sound recognition impairment was restricted to high frequency stimuli, so the authors concluded that the disorder was one of recognition and not perception. More recently, both single case reports (Lambert, Eustache, Lechevalier et al., 1989) and group studies of patients with auditory agnosia have been reported (Schnider et al., 1994).

Despite the lack of clear evidence for selective auditory agnosia, Kleist (1928, 1934, cited in Spreen et al., 1965) provided the first theoretical analysis of auditory agnosia by drawing a distinction between the ability to perceive isolated sounds or sequences of them, and the ability to understand their meaning. He also suggested that the two disorders were associated with impairments to different cortical regions: the perceptual disorder was associated with damage to Brodmann's areas 41, 42 and 52 and the associative disorder with damage to areas 37 and 20. Several decades later, Vignolo and his colleagues (e.g., Spinnler and Vignolo, 1966; Vignolo, 1969, 1982) suggested these disorders might be analogous to the apperceptive and associative forms of visual agnosia proposed by Lissauer (1890), and therefore, that patients could either suffer from a perceptual-discriminative or an associative-semantic impairment. Recent observations that patients with left hemisphere lesions tend

to make semantic errors on auditory tasks and patients with right hemisphere lesions tend to make perceptual errors on auditory tasks has provided strong support for this general hypothesis (Faglioni, Spinnler and Vignolo, 1969; Spinnler and Vignolo, 1966; Vignolo, 1969, 1982; Schnider et al., 1994), even if specific localisation within the hemispheres has been more elusive.

Pure Word Deafness

Kussmaul (1877) coined the term "pure word deafness" to refer to an inability to comprehend spoken words despite intact hearing, speech production and reading ability. For example, one patient complained that speech sounded "like a great noise all the time . . . like a gramophone, boom, boom, boom, jumbled together like foreign folks speaking in the distance" (Klein and Harper, 1956, p 113), and another captured the phenomenology of the disorder by saying: "I can hear you talking but I can't translate it" (Kanshepolsky et al., 1973). The experience of word sounds appears to undergo a qualitative change, and some word deaf patients cannot judge the length of a word (Klein and Harper, 1956). However, some patients appear able to extract information about the speaker from their voice (i.e., sex, age, region of origin, or affective information) despite being unable to comprehend the spoken message (Klein and Harper, 1956; Miceli, 1982; Michel et al., 1980). Despite this impairment in speech comprehension, these patients can be unimpaired at identifying non-linguistic sounds.

Similar to auditory agnosia, the existence of pure word deafness as a selective disorder has not always been readily accepted. Freund (1895; cited in Goldstein, 1974) suggested that it was merely a symptom of partial cortical deafness and Marie (1906) wrote that he had never observed a case that even approached the classic description. As a result a controversy regarding pure word deafness developed regarding whether the term "pure" should indicate that the disorder only impairs auditory processing of *language* (and not other auditory stimuli), or that the disorder only impairs *auditory processing* of language (and other aspects of language are intact; see Kussmaul vs. Wernicke debate). Most patients who have been labelled as word deaf appear also to have suffered from auditory agnosia (e.g., Auerbach, Allard, Naeser et al., 1982; Gazzaniga, Velletri Glass, Sarno et al., 1973; Oppenheimer and Newcombe, 1978; von Stockert, 1982), amusia (e.g., Tanaka, Yamadori and Mori, 1987; Denes and Semenza, 1975; Shoumaker, Ajax and Schenkenberg, 1977), or both (e.g., Wohlfart, Lindgren and Jernelius, 1952), providing evidence against the former interpretation. Indeed, in a review of the first 100 years of research on word deafness, Buchman, Garron, Trost-Cardmone et al. (1986) concluded that all patients diagnosed with pure word deafness suffer from additional auditory processing impairments and even suggested that the word "pure" be dropped from the name of the syndrome. However, more recently, cases of pure word deafness without other auditory impairments have been reported (Yaqub et al., 1988; Takahashi, Kawamura, Shinotou et al., 1992; Metz-Lutz and Dahl, 1984), providing support for the former interpretation. Therefore it seems important and

appropriate to reserve "pure word deafness" for patients who process other types of auditory information normally and have intact linguistic skills in other domains, regardless of how rare such cases might be.

Early researchers (e.g., Kussmaul, 1877; Bastian, 1897; Barrett, 1910; Hemphill and Stengel, 1940) generally agreed that pure word deafness was associated with damage in the temporal convolution of the left hemisphere, but that it was most commonly observed after bilateral lesions. For example, Mills (1891) described a woman who had two apoplectic attacks: one in the left hemisphere that rendered her word deaf and then a second one in the right hemisphere that left her cortically deaf. These observations lead Mills to conclude that a lesion in the posterior two thirds of the first and second left temporal convolutions will produce complete, or almost complete word deafness, whereas destruction of these areas in both hemispheres would be necessary for cortical deafness.

However, more recent investigations have suggested a more compelling anatomical explanation of word deafness in terms of a disconnection of Wernicke's area from auditory input.

This disconnection could occur after two types of lesions: (a) bilateral damage to auditory cortex or (b) a subcortical lesion in the left hemisphere that severs both ipsilateral and contralateral projections to Wernicke's area (Ziegler, 1952; Geschwind, 1965; Coslet et al., 1984; Jones and Dinolt, 1952). In either instance, the functional result of the lesion would be to impair the processing of auditorily presented linguistic stimuli.

Theoretical analyses have focused upon whether or not pure word deafness is caused by a general low-level auditory processing impairment or by a more specific impairment in linguistic processing. These impairments have been labelled Type 1 and Type 2, respectively (Auerbach et al., 1982). Type 1 pure word deafness is observed following bilateral lesions and is attributed to a pre-phonemic, apperceptive disorder that is likely to impair the processing of other types of auditory stimuli as well (e.g., Albert and Bear, 1974; Auerbach et al., 1982; Motomura, Yamadori, Mori et al., 1986; Tanaka et al., 1987; Yaqub et al., 1988, Lambert et al., 1989). By contrast, Type 2 pure word deafness is observed following a unilateral left hemisphere lesion and is attributed to an impairment in phonemic discrimination (e.g., Saffran, Marin and Yeni-Komshian, 1976; Metz-Lutz and Dahl, 1984). Most recently, it has been suggested that word deafness may be attributable to both a general auditory deficit and a specific phonetic impairment (Praamstra, Hagoort, Maassen et al., 1991).

One curious aspect of these theoretical analyses is that they have paid little attention to whether pure word deafness is apperceptive or associative in origin. Recently, however, Corballis (1994) suggested that pure word deafness may represent an apperceptive auditory deficit and that its associative counterpart is pure word meaning deafness – a very rare disorder in which patients can hear and repeat words, but do not know their meaning (see Ellis and Young, 1988; Franklin, Turner, Ralph et al., 1996).

PHONAGNOSIA

The final auditory processing disorder to be reviewed, phonagnosia, is also the most recently discovered, and thus least well examined. Van Lancker and Canter (1982) coined the term to refer to an impairment in the ability to recognise familiar voices, just as prosopagnosia reflects an impairment in the ability to recognise familiar faces (Bodamer, 1947). They observed an impairment in recognising famous voices on a test in which patients were asked to identify which of four names or faces matched a particular famous voice. Subsequent research replicated this finding, and produced evidence for a double dissociation between memory for familiar voices and the ability to discriminate between unfamiliar voices: One group of patients performed normally on the discrimination task but was impaired on the memory task, whereas another group of patients performed normally on the memory task, but was impaired on the discrimination task (Van Lancker et al., 1988). Despite being unable to identify voices, patients suffering from phonagnosia appear to be able to understand speech and identify nonverbal sounds (Van Lancker and Kreiman, 1987).

The functional double dissociation between the memory and discrimination tasks was strengthened by analysing the lesion location in a group study (Van Lancker and Kreiman, 1987) and six case studies (Van Lancker et al., 1988). Performance on a discrimination task was impaired by a lesion to either temporal lobe, whereas performance on the famous voices task was impaired by lesions to the right parietal lobe. Based on these findings, Van Lancker and colleagues have argued that voice discrimination and voice recognition were not only independent, but also unordered abilities. Furthermore, based on the observation that four patients who were impaired on the recognition or discrimination tasks but had no difficulty with environmental sounds (Van Lancker and Kreiman, 1987), they suggested that voices may be processed by an independent system analogous to the independent system that has been implicated in face processing (e.g., Bruce and Young, 1986). Whether or not phonagnosia dissociates from impairments in understanding affectively intoned speech (e.g., Heilman et al., 1975) in the same way that identifying affective facial expressions dissociates from prosopagnosia (e.g., Bowers, Bauer, Coslett et al., 1985; Parry, Young, Saul et al., 1991) remains to be determined.

IMPLICATIONS FOR THEORIES OF AUDITORY PROCESSING

These four brief reviews have served to illustrate that auditory processing disorders have usually been considered in isolation and theorising has tended to remain specific to the individual disorders. Tacit acknowledgement of the other disorders has occurred in some instances, but issues regarding the general architecture of the auditory system have been overlooked. The remainder of this paper attempts to rectify this situation by outlining some of the problems with the single system theory that has been prominent in the auditory processing literature, and proposing that a modular conceptualisation of auditory processing

analogous to the dominant theory in visual processing overcomes many of these problems.

In the limited literature on the relationship between disorders of auditory processing, most researchers have favoured a model that construes the disorders in terms of a continuum that reflects the output of a single serial processing system (e.g., Goldstein, 1974; Graham et al.,1980; Buchman et al., 1986; Mendez and Geehan, 1988). The main evidence marshalled in favour of this position appears to be the preponderance of co-occurring deficits (suggesting that selective deficits do not exist), and the observation that cortical deafness often resolves into auditory agnosia or pure word deafness. Based on this evidence, Buchman et al. (1986) concluded "the common features that can be delineated in reported cases of pure word deafness, auditory agnosia and cortical deafness suggest that the variation in auditory comprehension is a difference in degree rather than a manifestation of separate syndromes" (p. 498). However, theorising about normal function based on clusters of impairments has been criticised as "psychologically weak" (Caramazza, 1984), and Shallice (1988) concluded that "clusters of impairments found in common across patients have proved a treacherous foundation on which to build a bridge to normal function" (p. 34). These criticisms have been levied because: (a) neurological impairments rarely respect cognitive boundaries, making it difficult to observe damage to a specific module in isolation; and (b) it would not be surprising if similar stimuli are processed by nearby cortical regions, making them susceptible to impairment by a single lesion even if functionally independent modules are operating. These criticisms become more forceful when consideration is given to the neuroanatomy of the auditory system discussed in the introduction. Therefore, although identifying co-occurring deficits may be clinically useful and important, it does not provide strong grounds for theorising about the normal auditory system.

Although less influential in auditory research, evidence for a modular system exists in the auditory literature in the form of double dissociations across patients; that is, some patients are unable to identify non-linguistic sounds, but can understand speech (e.g., Spreen et al., 1965), whereas other patients cannot comprehend speech, but can identify non-linguistic sounds (e.g., Metz-Lutz and Dahl, 1984; Saffran et al., 1976). If one adopts the strong position that a double dissociation is enough to postulate independent processing modules (Shallice, 1988), then the performance of these patients indicates that linguistic and non-linguistic auditory sounds are processed by independent modules (cf. Albert, Sparks, Von Stockert et al., 1972).

In addition to this evidence, the variety of patterns in which auditory disorders develop and resolve supports a modular theory. It has been argued that because cortical deafness resolves into auditory agnosia or pure word deafness, that the disorders represent a processing continuum (Buchman et al., 1986; Mendez and Geehan, 1988). But, if a continuum existed, resolution across patients would be expected to occur based on an identifiable variable such as task difficulty or some dimension of the stimuli. For example, if auditory processing relied on a single system and identifying speech was inherently more difficult than identifying non-linguistic sounds, then cases in which cortical

deafness resolved would all be expected to be impaired at identifying speech for longer than they were impaired at identifying nonlinguistic sounds. However, no such orderly progression in the resolution of auditory processing disorder has been observed, nor has any variable that could account for the patterns of deficit (e.g., frequency range) been identified. Therefore, a modular theory that allows for several patterns of deficits to be observed based on the severity of damage to the individual modules seems to provide a better account of the existing data.

A modular theory also provides a more coherent account of cases in which a selective impairment in one type of stimulus (e.g., sounds) is attributed to a perceptual deficit, but the patient shows normal performance with another type of stimulus (e.g., words; see Fujii et al., 1990). In a monolithic, serial system, a low-level perceptual impairment would be expected to affect the processing of all types of stimuli. By contrast, if different modules process different types of information, then a low-level impairment in one module would not necessarily affect the manner in which stimuli are processed in the other module(s). In short, it is difficult to understand how a perceptual impairment can impair the processing of one class of stimulus (i.e., non-linguistic sounds) but not another class (i.e., linguistic sounds) unless independent processing modules exist.

Further support for this type of model can be garnered by drawing an analogy with disorders of visual processing.[2] Specifically, each of the four auditory disorders can be deemed to be analogous to an established disorder in the visual domain: cortical deafness and blindness represent sensory impairments; the agnosias represent perceptual and/or semantic impairments; the dyslexias and pure word deafness represent receptive language impairments and phonagnosia and prosopagnosia represent person information impairments. Given the widespread support for modularity in visual processing and the strength of these analogies, it is surprising that modular theory has had so little impact on theorising about auditory processing.

Although the accumulated evidence supports a modular auditory system that operates in parallel more than a monolithic system that operates serially, the modular proposal is open to the same criticisms levied against the modular visual system. First and foremost, is the so-called "binding problem"; that is, if constituent parts of a stimulus are processed by independent modules, then the information must be re-combined at some stage, but that stage remains unspecified. This problem remains unanswered in visual processing, so it would premature to expect an answer in the auditory domain. A related issue concerns the apparently "homuncular" nature of the system; that is, sounds appear to be transported to the appropriate cortical region for analysis, but the mechanism that "decides" to which region a stimulus is sent, and the process by which information reaches those regions, remain unspecified. Although only speculative, one possible resolution to this problem is to eliminate the need for such a selective mechanism by hypothesising that the auditory signal is not segmented for processing by modules, but rather, shared by the entire system (e.g., Fujii et al., 1990). According to this hypothesis, all modules receive the

[2]Boles and Pasquarette (1996) suggest that differences exist in the modular structure of the visual and auditory systems, and therefore urge caution when drawing analogy. We agree that caution may be warranted when considering specific processes within the system, but we believe that analogies are appropriate at this more macro level of analysis.

same input, but they differ either in the nature of the information they process, or in the efficiency with which they process different aspects of the auditory signal. In addition to eliminating the need for a decision mechanism, this type of model can also account for the observation that when auditory processing impairments resolve, they often do so relatively quickly, by hypothesising that when one module is destroyed another module compensates without requiring extensive neural re-organisation. This type of model is similar to the suggestion that the visual system has separate processors to analyse words and faces, but that both processors contribute to the analysis of objects (Farah, 1991, 1994). To the extent that pure word deafness and phonagnosia are auditory analogies to visual disorders in processing words and faces, two types of auditory processors can be postulated – one for voices and the other for words. This possibility is a modification of the theory that verbal and nonverbal sounds are processed independently (Albert et al., 1972).

A modular theory of auditory processing is also likely to confront questions relating to the specificity of deficits and the number of modules that exist, which have been raised in the visual domain in conjunction with the vexing relationship between prosopagnosia and visual associative agnosia. Whereas, some researchers prefer to construe prosopagnosia as an independent visual processing impairment associated with damage to neural hardware dedicated to processing faces (e.g., Seeck, Mainwaring, Ives et al., 1993; Sergent, Ohtaz and MacDonald, 1992), others suggest that prosopagnosia is associated with an impairment in the complex, within-category processing required to identity faces, and that it could be observed on any task involving within-category discrimination (e.g., Damasio, Damasio and Van Hoesen, 1982). This issue is particularly difficult to resolve because it becomes confounded with possible effects of expertise with certain stimuli (i.e., faces), and differences between novel and previously known stimuli (i.e., anterograde and retrograde amnesia; see Farah, Levinson and Klein, 1995). To the extent that phonagnosia and auditory associative agnosia are auditory analogies for prosopagnosia and visual associated agnosia, then the same questions are likely to be raised in the auditory domain.

Modular conceptualisations of function are generally closely aligned to localisationist theories in which each module is associated with an identifiable region of the brain, usually in the cortex. In the case of auditory processing, however, although disorders appear to reflect impairments in the operation of distinct modules, attempts at localisation have met with limited success, at least partially because of the neuroanatomical proximity of the putative components of the auditory system. Thus, for example, there is relatively little neural space between the regions involved in perception and semantics in the auditory system, making it less likely that these components would be selectively impaired in a given patient. By contrast, the relatively large neural space between these regions in the visual system makes observing selective impairments more common. In addition, the bilateral projections in the auditory system mean that bilateral lesions are often required to observe any deficit. Indeed, with the exception of phonagnosia, most disorders appear most frequently following bilateral lesions.

Although no single region has been implicated in a particular behavioural disorder, and similar behavioural disturbances have been observed following dramatically different lesions, some evidence for hemispheric specialisation has been demonstrated. Thus, pure word deafness is generally attributed to left hemisphere lesions (e.g., Takahashi et al., 1992) and auditory agnosia and phonagnosia are generally attributed to right hemisphere lesions (Schnider et al., 1994; Van Lancker and Canter, 1982).

Furthermore, Ulrich (1978) reviewed a number of cases of auditory impairments following damage to the right and left hemispheres on successive occasions, and found that the side of the initial insult correlated significantly with the subsequent nature of the disorder: An initial insult in the left hemisphere produced 11 cases with predominantly linguistic impairments, but only two patients with predominantly non-linguistic impairments, whereas an initial "injury" in the right hemisphere produced two cases with linguistic impairments but five cases with non-linguistic impairments. This finding suggests that the left hemisphere is more critical to linguistic processing and the right hemisphere is more critical to non-linguistic processing, allowing another parallel to be drawn with the visual domain in which reading is associated with left hemisphere and face recognition is associated with the right hemisphere. However, the dangers associated with ascribing normal function to an impaired brain region make it clear that some form of converging evidence is required before any firm conclusions can be drawn regarding the location of the postulated modules in the auditory domain. Recent evidence that hemispheric differences in auditory perception appear to parallel those found in visual perception (Ivry and Lebby, 1993) provides one form of evidence. In future, functional imaging studies of normal subjects with either PET or fMRI might help localise specific components of auditory processing, thereby providing critical information regarding the architecture of the auditory system and its instantiation in the brain. Success using PET has already been observed in a study of musical processing (Sergent, Zuck, Terriah et al., 1992), and there is no reason why it would not be equally successful when applied to the auditory stimuli that cannot be processed in word deafness, auditory agnosia and phonagnosia.

Given the frequent analogies to visual processing made throughout this review, it seems appropriate to conclude by drawing on that literature to suggest directions for future research in the auditory domain. For example, error analyses have been used to distinguish between apperceptive and associative forms of auditory agnosia, but it may be possible to develop auditory counterparts to the visual tests used to identify apperceptive and associative impairments. One hallmark of visual apperceptive agnosia is impaired processing of perceptually degraded representations (e.g., Warrington and James, 1967), and it may be possible to use degraded auditory stimuli (cf. Schacter and Church, 1992) to identify cases of apperceptive auditory agnosia. Similarly, it may be possible to construct tests for associative auditory agnosia that involve same/different judgements of different exemplars of a category of animal producing the same sound (i.e., two dogs barking) that would be conceptually similar to the tests used to test for visual associative agnosia (e.g., De Renzi,

Scotti and Spinnler, 1969). In addition to being applicable to patient populations, these tests could also be used with normal subjects and with volunteers in functional imaging studies to provide additional empirical evidence that is likely to aid theorists in constructing functional and neuroanatomical models of normal auditory processing, regardless of whether or not the ultimate architecture is modular in nature.

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References

ALBERT, M., and BEAR, D. Time to understand: A case of word deafness with reference to the role of time in auditory comprehension. *Brain, 97:* 373-384, 1974.

ALBERT, M.L., SPARKS, R. VON STOCKERT, T., and SAX, D. A case of auditory agnosia: Linguistic and non-linguistic processing. *Cortex, 8:* 427-443, 1972.

AUERBACH, S.H., ALLARD, T., NAESER, M., ALEXANDER, M.P., and ALBERT, M.L. Pure word deafness: Analysis of a case with bilateral lesions and a defect at the prephonemic level. *Brain, 105:* 271-300, 1982.

BALLAS, J.A. Common factors in the identification of an assortment of brief everyday sounds. *Journal of Experimental Psychology: Human Perception and Performance, 19:* 250-267, 1993.

BALLAS, J.A., and MULLINS, R.T. Effects of context on the identification of everyday sounds. *Human Performance, 4:* 199-219, 1991.

BARRETT, A.M. A case of pure word deafness with autopsy. *Journal of Nervous and Mental Disease, 37:* 73-92, 1910.

BASTIAN, H.C. Some problems in connexion with aphasia and other speech defects. *Lancet, 1*: 933-942, 1005-1017, 1131-1137, 1187-1194, 1897.

BAUER, R. Agnosia. In K. Heilman and Valenstein (Eds.), *Clinical Neuropsychology* (Third Edition). New York: Oxford University Press, 1993, pp. 215-278.

BEATON, A., and MCCARTHY, M. On the nature of auditory neglect: A reply to Hugdahl and Wester. *Brain and Language, 48:* 351-358, 1995.

BEATON, A., and MCCARTHY, M. "Auditory neglect after right frontal lobe and right pulvinar thalamic lesions": Comments on Hugdahl, Wester and Asbjørseon (1991) and some preliminary findings. *Brain and Language, 44:* 121-126, 1993.

BODAMER, J. Die Prosop-Agnosie, *Arch. Psych. Nerv., 179:* 6-53, 1947. [Translated by H.D. Ellis and M. Florence. Bodamer's (1947) Paper on Prosopagnosia, *Cognitive Neuropsychology, 7:* 81, 1990].

BOLES, D.B., and PASQUARETTE, S.J. Correlated auditory asymmetries in lexical and non lexical tasks. *Cortex, 32:* 527-545, 1996.

BOWERS, D., BAUER, R. COSLETT, H.B., and HEILMAN, K.M. Processing of faces by patients with unilateral hemisphere lesions: 1. Dissociation between judgements of facial affect and facial identity. *Brain and Cognition, 4:* 258-272, 1985.

BRAMWELL, E. A case of cortical deafness. *Brain, 50:* 579-580, 1927.

BREGMAN, A.S. *Auditory Scene Analysis: The Perceptual Organization of Sound.* Cambridge: MIT Press, 1990.

BREGMAN, A.S. Auditory scene analysis: hearing in complex environments. In S. McAdams and E. Bigand (Eds.), *Thinking in Sound.* Oxford: Clarendon Press, 1993, pp.10-36.

BRUCE, V., and YOUNG, A. Understanding face recognition. *British Journal of Psychology, 77:* 305-327, 1986.

BUCHMAN, A.S., GARRON, D.C., TROST-CARDMONE, J.E., WICHTER, M.D., and SCHWARTZ, M. Word deafness: One hundred years later. *Journal of Neurology, Neurosurgey and Psychiatry, 49:* 489-499, 1986.

BUCHTEL, H.A., and STEWART, J.D. Auditory agnosia: Apperceptive or associative disorder? *Brain and Language, 37:* 12-25, 1989.

BULL, R., and CLIFFORD, B.R. Earwitness voice recognition accuracy. In G.L. Wells and E.F. Loftus (Eds.), *Eyewitness Testimony: Psychological Perspectives.* Cambridge: Cambridge University Press, 1984, pp. 92-123.

CAPLAN, D. *Neurolinguistics and Linguistic Aphasiology: An Introduction.* Cambridge: Cambridge Univeristy Press, 1987.

CARMON, A., and NACHSON, I. Ear asymmetry in perception of emotional non-verbal stimuli. *Acta Psychologica, 37:* 351-357, 1973.

CARAMAZZA, A. The logic of neuropsychological research and the problem of patient classification in aphasia. *Brain and Language, 21:* 9-20, 1984.

CELESIA, G.G. Organisation of auditory cortical areas in man. *Brain, 99:* 403-414, 1976.

CHIU, C.-Y.P., and SCHACTER, D.L. Auditory priming for nonverbal information: Implicit and explicit memory for environmental sounds. *Consciousness and Cognition, 4:* 440-458, 1995.

CLARK, W.E., and RUSSELL, R. Cortical deafness without aphasia. *Brain, 61:* 375-383, 1938.

COLTHEART, M., PATTERSON, K.E., and MARSHALL, J.C. *Deep Dysxlexia* (2nd ed.). London: Routledge and Kegan Paul, 1987.

CORBALLIS, M.C. Neuropsychology of perceptual functions. In D.W. Zaidel (Ed.), *Neuropsychology*. San Diego: Academic Press, 1994, pp. 83-104.

CORBALLIS, M.C., and OGDEN, J.A. Dichotic listening in commissurotomized and hemispherectomized subjects. *Neuropsychologia, 26:* 565-573, 1988.

COSLETT, H.B., BRASHEAR, H.R., and HEILMAN, K.M. Pure word deafness after bilateral primary auditory cortex infarcts. *Neurology, 34:* 347-352, 1984.

CRITCHLEY, M., and MENSON, R.A. *Music and the Brain: Studies in the Neurology of Music.* London: William Heinemann, 1977.

DAMASIO, A.R., DAMASIO, H., and VAN HOESEN, G.W. Prosopagnosia: Anatomic basis and behavioral mechanisms. *Neurology, 32:* 331-341, 1982.

DENES, G., and SEMENZA, C. Auditory modality-specific anomia: evidence from a case of pure-word deafness. *Cortex, 11:* 401-411, 1975.

DE RENZI, E., SCOTTI, E., and SPINNLER, H. Perceptual and associative disorders of visual recognition: Relationship to the site of lesion. *Neurology, 19:* 634-642, 1969.

EARNEST, M.P., MONROE, P.A., and YARNELL, P.R. Cortical deafness: Demonstration of the pathologic anatomy by CT scan. *Neurology, 27:* 1172-1175, 1977.

ELLIS, A.W., and YOUNG, A.W. *Human Cognitive Neuropsychology*. Hove: Lawrence Erlbaum Associates, 1988.

FAGLIONI, P., SPINNLER, H., and VIGNOLO, L.A. Contrasting behavior of right and left hemisphere damaged patients on a discriminative and semantic task of auditory recognition. *Cortex, 5:* 366-389, 1969.

FARAH, M.J. Patterns of co-occurrence among the associative agnosias: Implications for visual object representation. *Cognitive Neuropsychology, 8:* 1-19, 1991.

FARAH, M.J. Specialization within visual object recognition: Clues from prosopagnosia and alexia. In M.J. Farah and G. Ratcliff (Eds.), The *Neuropsychology of High-level Vision: Collected Tutorial Essays*. Hillsdale: Lawrence Erlbaum Associates, 1994, pp. 133-146.

FARAH, M.J., LEVINSON, K.L., and KLEIN, K.L. Face perception and within-category discrimination in prosopagnosia. *Neuropsychologia, 33:* 661-674, 1995.

FRANKLIN, S., TURNER, J., RALPH, M., MORRIS, J., and BAILEY, P. A distinctive case of word meaning deafness. *Cognitive Neuropsychology, 13:* 1139-1162, 1996.

FRAUENFELDER U.H., and TYLER, L.K. The process of spoken word recognition: An introduction. *Cognition, 25:* 1-20, 1987.

FREUD, S. *Zur Auffassung der Aphasien.* Wien: Deuticke, 1891. [English translation, *On Aphasia*, London: Imago, 1953].

FUJII, T., FUKATSU, R. WATABE, S., OHUMA, A., TERAMURA, K., KIMURA, I., SASO, S., and KOGURE, K. Auditory sound agnosia without aphasia following a right temporal lobe lesion. *Cortex, 26:* 263-268, 1990.

GAZZANIGA, M., VELLETRI GLASS, A., SARNO, M.T., and POSNER, J.B. Pure word deafness and hemispheric dynamics: A case history. *Cortex, 9:* 137-143, 1973.

GESCHWIND, N. Disconnexion syndromes in animals and man. *Brain, 88:* 237-294, 1965.

GLISKY, E.L., POLSTER, M.R., and ROUTHIEAUX, B.C. Double dissociation between item and source memory. *Neuropsychology, 9:* 229-235, 1995.

GOLDSTEIN, M.N. Auditory agnosia for speech ("Pure word deafness"): A historical review with current implications. *Brain and Language, 1:* 195-204, 1974.

GOLDSTEIN, M.N., BROWN, M., and HOLLANDER, J. Auditory agnosia and cortical deafness: Analysis of a case with 3-yr followup. *Brain and Language, 2:* 324-332, 1975.

GOODGLASS, H. *Understanding Aphasia.* San Diego: Academic Press, 1993.

GRAHAM, J., GREENWOOD, R., and LECKY, B. Cortical deafness: A case report and review of the literature. *Journal of the Neurological Sciences, 48:* 35-49, 1980.

HEILMAN, K.M., and VALENSTEIN, E. Auditory neglect in man. *Archives of Neurology, 26:* 32-35, 1972.

HEILMAN, K.M., SCHOLES, R., and WATSON, R.T. Auditory affective agnosia: Disturbed comprehension of affective speech. *Jorunal of Neurology, Neurosurgery and Psychiatry, 38:* 69-72, 1975.

HEMPHILL, R.E., and STENGEL, E. A study of pure word-deafness. *Journal of Neurological Psychiatry, 3:* 251-262, 1940.

HENSON, R.A. The language of music. In M. Critchely and R.A. Henson (Eds.), *Music and the Brain.* Southampton: Camelot Press Ltd., 1978, pp. 233-254.

HUGDAHL, K. *Handbook of Dichotic Listening: Theory, Methods and Research.* Chichester: John Wiley, 1988.

HUGDAHL, K., and WESTER, K. Auditory neglect and the ear extinction effect in dichotic listening: A

reply to Beaton and McCarthy (1933). *Brain and Language, 46:* 166-173, 1994.
HUGDAHL, K., WESTER, K., and ASBJØRNSEN, A. Auditory neglect after right frontal lobe and right pulvinar thalamic lesions. *Brain and Language, 41:* 465-473, 1991.
IVRY, R.B., and LEBBY, P.C. Hemispheric differences in auditory perception are similar to those found in visual perception. *Psychological Science, 3:* 41-45, 1993.
JACKENDOFF, R., and LERDAHL, F. Grammatical parallel between music and language. In M. Clynes (Ed.), *Music, Mind, and Brain: The Neuropsychology of Music.* New York: Plenum Press, 1982, pp. 83-117.
JERGER, J., WEIKERS, N.J., SHARBROUGH, F.W., and JERGER, S. Bilateral lesions of the temporal lobe. *Acta Otolaryngology, Supplement 258:* 7-51, 1969.
JONES, J., and DINOLT, R. Pure word deafness. *Laryngoscope, 62:* 194-203, 1952.
KANSHEPOLSKY, J., KELLEY, J.J., and WAGNER, J.D. A cortical auditory disorder: clinical, audiologic and pathologic aspects; *Neurology, 23:* 699-705, 1973.
KIMURA, D. Functional asymmetry in the brain in dichotic listening. *Cortex, 3:* 163-178, 1967.
KLEIN, R., and HARPER, J. The problem of agnosia in the light of a case of pure word deafness. *Journal of Mental Science, 102:* 112-120, 1956.
KUSSMAUL, A. Disturbances of speech. In H. von Ziemssen (Ed.), *Cyclopedia of the Practice of Medicine, Vol. 14.* New York: William Wood and Co., 1877, pp. 581-875.
LAMBERT, J., EUSTACHE, F., LECHEVALIER, B., ROSSA, Y., and VIADER, F. Auditory agnosia with relative sparing of speech perception. *Cortex, 25:* 71-82, 1989.
LHERMITTE, F., CHAIN, F., ESCOUROLLE, R., DUCARNE, B., PILLON, B. and CHEDRU, F. Études des troubles perceptifs auditifs dans les lésions temporales bilatérales. *Revue Neurologique, 124:* 329-351, 1971.
LICHTEIM, L. On aphasia. *Brain, 7:* 433-484, 1885.
LISSAUER, H. Ein Fall von Seelenblindheit nebst einem Beitrag zur Theorie derselben. *Archiv für Psychiatrie, 21:* 222-270, 1890. [Edited and reprinted in translation by Jackson, M. Lissauer on agnosia. *Cognitive Neuropsychology, 5:* 155-192, 1988].
MARIE, P. What to think about subcortical aphasia (pure aphasia). *Semaine Medicale, 26:* 493-500, 1906. [Translated in M.F. Cole and M. Cole (Eds. and Transl.), *Pierre Marie's Papers on Speech Disorders.* New York: Hafner, 1971, pp. 75-102].
MARR, D. Visual information processing: The structure and creation of visual representations. *Philosophical Transactions of the Royal Society, B290:* 199-218, 1980.
MARR, D. *Vision.* San Francisco: Freeman, 1982.
MARSHALL, R.C., RAPPAPORT, B.Z., and GARCIA-BUNEL, L. Self-monitoring behaviour in a case of severe auditory agnosia with aphasia. *Brain and Language, 24:* 297-313, 1985.
MCADAMS, S., and Bigand, E. *Thinking in Sound*. Oxford: Clarendon Press, 1993.
MCCARTHY, R.A., and WARRINGTON, E.K. *Cognitive Neuropsychology: A Clinical Introduction.* San Diego: Academic Press, 1990.
MENDEZ, M.F., and GEEHAN, G.R. Cortical auditory disorders: Clinical and psychoacoustic features. *Journal of Neurology, Neurosurgery and Psychiatry, 51:* 1-9, 1988.
METZ-LUTZ, M., and DAHL, E. Analysis of word comprehension in a case of pure word deafness. *Brain and Language, 23:* 12-25, 1984.
MICELI. G. The processing of speech sounds in a patient with cortical auditory disorder. *Neuropsychology, 20:* 5-20, 1982.
MICHEL, F. PERONNET, F., and SCHOTT, B. A case of cortical deafness: Clinical and electrophysiological data. *Brain and Language, 10:* 367-377, 1980.
MILLS, C.K. On the localisation of the auditory centre. *Brain, 14:* 465-472, 1891.
MOTOMURA, N., YAMADORI, A., MORI, E., and TAMARU, F. Auditory agnosia: Analysis of a case with bilateral subcortical lesions. *Brain, 109:* 379-391, 1986.
OPPENHEIMER, D.R., and NEWCOMBE, F. Clinical and anatomic findings in a case of auditory agnosia. *Archives of Neurology, 35:* 712-719, 1978.
PARRY, F.M., YOUNG, A.W., SAUL, J.S.M., and MOSS, A. Dissociable face processing impairments after brain injury. *Journal of Clinical and Experimental Neuropsychology, 14:* 534-547, 1991.
PATTERSON, K.E., COLTHEART, M., and MARSHALL, J.C. *Surface Dyslexia: Neuropsychology and Cognitive Studies of Phonological Reading.* Hove: Lawrence Erlbaum Associates Ltd., 1985.
PERETZ, I. Auditory agnosia: A functional analysis. In S. McAdams and E. Bigand, *Thinking in Sound.* Oxford: Clarendon Press, 1993, pp. 199-230.
PERETZ, I., KOLINSKY, R., TRAMO, M., LABRECQUE, R., HUBLET, C., DEMEURISSE, G., and BELLEVILLE, S. Functional dissociations following bilateral lesions of auditory cortex. *Brain, 117:* 1283-1301, 1994.
PISONI, D., and LUCE, P.A. Acoustic-phonetic representations in word recognition. *Cognition, 25:* 21-52, 1987.
POLK, M., and KERTESZ, A. Music and language in degenerative disease of the brain. *Brain and Cognition, 22:* 98-117, 1993.
PRAAMSTRA, P., HAGOORT, P., MAASSEN, B., and CRUL, T. Word deafness and auditory cortical function. *Brain, 114:* 1197-1225, 1991.

RAPIN, I. Cortical deafness, auditory agnosia, and word-deafness: How distinct are they? *Human Communication Canada, 9:* 29-37, 1985.
REINHOLD, M. A case of auditory agnosia. *Brain, 72:* 203-223, 1950.
ROSATI, G., DE BASTIANI, P., PAOLINO, E., PROSSER, S., ARSLAN, E., and ARTIOLI, M. Clinical and audiological findings in a case of auditory agnosia. *Journal of Neurology, 227:* 21-27, 1982.
RUBENS, A.R. Agnosia. In K. Heilman and E. Valenstein (Eds.), *Clinical Neuropsychology.* New York: Oxford University Press, 1979.
SAFFRAN, E.M., MARIN, O.S.M., and YENI-KOMSHIAN, G.H. An analysis of speech perception in word deafness. *Brain and Language, 3:* 209-228, 1976.
SCHACTER, D.L., and CHURCH, B. Implicit memory in amnestic patients: When is auditory priming spared? *Journal of the International Neuropsychology Society, 1:* 434-442, 1995.
SCHACTER, D.L., and CHURCH, B.A. Auditory priming: Implicit and explicit memory for words and voice. *Journal of Experimental Psychology: Learning, Memory and Cognition, 18:* 915-930, 1992.
SCHACTER, D.L., and TULVING, E. *Memory Systems 1994.* Cambridge: MIT Press, 1994.
SCHNIDER, A., BENSON, D.R., ALEXANDER, D.N., and SCHNIDER-KLAUS, A. Non-verbal environmental sound recognition after unilateral hemispheric stroke. *Brain, 117:* 281-287, 1994.
SEECK, M., MAINWARING, N., IVES, J., BLUME, H., DUBUISSON, D., COSGROVE, R., MESULAM, M-M., and SCHOMER, D.L. Differential neural activity in the human temporal lobe evoked by faces of family members and friends. *Annals of Neurology, 34:* 369-372, 1993.
SERGENT, J., OHTA, S., and MACDONALD, B. Functional neuroanatomy of face and object processing: A positron emission tomography study. *Brain, 115:* 15-36, 1992.
SERGENT, J., ZUCK, E., TERRIAH, S., and MACDONALD, B. Distributed neural network underlying musical sight-reading and keyboard performance. *Science, 257:* 106-109, 1992.
SHALLICE T. *From Neuropsychology to Mental Structure.* Cambridge: Cambdridge University Press, 1988.
SHOUMAKER, R.D., AJAX, E.T., and SCHENKENBERG, T. Pure word deafness (auditory verbal agnosia). *Diseases of the Nervous System, 38:* 293-299, 1977.
SPARKS, R., and GESCHWIND, N. Dichotic listening in man after section of neocortical commissures. *Cortex, 4:* 3-16, 1968.
SPINNLER, H., and VIGNOLO, L.A. Impaired recognition of meaningful sounds in aphasia. *Cortex, 2:* 337-348, 1966.
SPREEN, O., BENTON, A.L., and FINCHAM, R.W. Auditory agnosia without aphasia. *Archives of Neurology, 13:* 84-92, 1965.
SQUIRE, L.R., *Memory and Brain.* New York: Oxford University Press, 1987.
TAKAHASHI, N., KAWAMURA, M., SHINOTOU, H., HIRAYAMA, K., KAGA, K., and SHINDO, M. Pure word deafness due to left hemisphere damage. *Cortex, 28:* 295-303, 1992.
TANAKA, Y., KAMO, T., YOSHIDA, M., and YAMADORI, A. So-called cortical deafness: Clinical, neurophysiological and readiological observations. *Brain, 114*: 2385-2401, 1991.
TANAKA, Y., YAMADORI, A., and MORI, E. Pure word deafness following bilateral lesions: A psychophysical analysis. *Brain, 110:* 381-403, 1987.
ULRICH, G. Interhemispheric functional relationships in auditory agnosia: An analysis of the preconditions and a conceptual model. *Brain and Language, 5:* 286-300, 1978.
VAN ESSEN, D.C., ANDERSON, C.H., and FELLEMAN, D.J. Information processing in the primate visual system: An integrated systems perspective. *Science, 255:* 419-423, 1992.
VAN LANCKER, D.R., and CANTER, G.J. Impairment of voice and face recognition in patients with hemispheric damage. *Brain and Cognition, 1:* 185-195, 1982.
VAN LANCKER, D.R., and KREIMAN, J. Voice discrimination and recognition are separate abilities. *Neuropsychologia, 25:* 829-834, 1987.
VAN LANCKER, D.R., CUMMINGS, J.L., KREIMAN, J., DOBKIN, B.H. Phonagnosia: A dissociation between familiar and unfamiliar voices. *Cortex, 24:* 195-209, 1988.
VAN WALLENDAEL, L.R., SURACE, A., HALL PARSONS, D., and BROWN, M. "Earwitness" voice recognition: Factors affecting accuracy and impact on jurors. *Applied Cognitive Psychology, 8:* 641-660, 1994.
VIGNOLO, L.A. Auditory agnosia: A review and report of recent evidence. In A.L. Benton (Ed.), *Contributions to Clinical Neuropsychology*. Chicago: Aldine Publishing Co., 1969, pp. 172-206.
VIGNOLO, L.A. Auditory agnosia. *Philosophical Transactions of the Royal Society (London), B 298:* 49-57, 1982.
VON STOCKERT, T.R. On the structure of word deafness and mechanisms underlying the fluctuation of disturbances of higher cortical functions. *Brain and Language, 16:* 133-146, 1982.
WARRINGTON, E.K., and JAMES, M. Disorders of visual perception in patients with localised cerebral lesions; *Neuropsychologia, 5:* 253-266, 1967.
WERNICKE, C. *Der Aphasische Symptomenkomplex: Eine Psychologische Studie auf Anatomischer Basis.* Breslau: Max Cohn und Weigert, 1874. [Translated in *Boston Studies in Philosophy of Science, 4:* 34-97, 1965]

WERNICKE, C., and FRIEDLANDER, C. Ein Fall von Taubheit in Folge von doppelseitiger Läsionen des Schlafenlappens. *Fortschritte der Medizin, 1:* 177-185, 1883. [Reprinted, "A case of deafness as a result of bilateral lesions of the temporal lobe", in G.H. Eggert (Ed.), *Wernicke's Work on Aphasia.* 1977, pp. 164-172.

WOHLFART, G., LINDGREN, A., and JERNELIUS, B. Clinical picture and morbid anatomy in a case of "pure word deafness". *Journal of Nervous and Mental Disease, 116:* 818-827, 1952.

YAQUB, B.A., GASCON, G.G., ALNOSHA, M., and WHITAKER, H. Pure word deafness (acquired verbal auditory agnosia) in an Arabic speaking patient. *Brain, 111:* 457-466, 1988.

YOUNG, A.W. Functional organisation of visual recognition. In L.Weiskrantz (Ed.), *Thought Without Language*. Oxford: Oxford University Press, 1988.

ZIEGLER, D.K. Word deafness and Wernicke's aphasia. *Archives of Neurology and Psychiatry, 67:* 323-333, 1952.

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