Abstract
The inherent heterogeneity of central auditory processing disorders (CAPDs) has led to difficulty developing a precise definition of what constitutes a CAPD. One method of acknowledging the varied behaviors and assessment findings observed in cases of CAPD has been that of categorization or subprofiling. This paper describes a method of subprofiling of CAPDs that relates each subprofile to its underlying neurophysiologic region of dysfunction in the brain as well as to its higher-level language and learning implications and sequelae. Three primary subprofiles, representing primary auditory (left) cortex, nonprimary auditory (right) cortex, and interhemispheric (corpus callosum) dysfunction, are described. In addition, two secondary subprofiles that describe dysfunction in associative (left) cortex and efferent and/or temporal-to-frontal cortex and which represent the gray area between audition and language/executive function are offered. These subprofiles are intended to aid in the interpretation of central auditory and related assessments so that deficit-specific management plans for individuals with CAPD may be devised.

The problem of agreeing on a precise definition of central auditory processing (CAP) and its disorders (CAPD) is an issue that has plagued audiologists and other interested professionals for decades, with much disagreement among factions and disciplines. Definitions of CAP have ranged from the very general (i.e., “What we do with what we hear,” Katz, 1992) to the very specific (i.e., an auditory modality-specific deficit in bottom-up processing of acoustic features of speech; McFarland & Cacace, 1995). In 1996, ASHA provided a definition of CAP that delineated the auditory behaviors that rely on central auditory mechanisms and processing (i.e., sound localization and lateralization, auditory discrimination, auditory pattern recognition, temporal aspects of audition, and performance with competing and/or degraded acoustic signals). Although this definition succeeded in decomposing audition into some of its constituent auditory behaviors, it failed to uncover underlying mechanisms responsible for such behaviors and was unable to illuminate how deficiencies in such behaviors may lead to difficulties in higher-level language, learning, and communicative tasks.

It is this author’s contention that the difficulty in defining CAPD stems directly from the fact that, like all other learning, language, and communicative disorders, CAPDs are inherently heterogeneous in nature and, thus, elude precise definition. When one speaks of learning disabilities or language disorders, it is accepted that different permutations exist that result in vastly different behavioral manifestations, and that management will be directed toward the specific type of disorder and the individualized behavioral sequelae associated with the deficit. There is no reason to believe that CAPDs are any different. Thus, perhaps Katz’s (1992) definition of CAP as “what we do with what we hear” was not so far off, after all.

One way to deal with the inherent heterogeneity of CAPDs is to examine constellations of behaviors and deficits, and to derive separate categories or subprofiles of CAPDs, each of which would, in theory, lead to different intervention recommendations and would allow for individualization of CAPD management. The concept of subprofiling of CAPDs is not a new one. For example, Katz, Smith, and Kurpita (1992) and Musiek, Gellegly, and Ross (1985) have described profiles of auditory processing deficits that have been instrumental in interpreting results of central auditory assessment and planning management strategies.

This paper will describe an alternative method of subprofiling CAPDs. Although some similarities exist between the Bellis/Ferre model (Bellis, 1996; Bellis & Ferre, 1996; Bellis & Ferre, in press; Ferre, 1997) and those that have gone before, there are subtle differences, as well. First, our model, like that described by Musiek et. al. (1985), is driven fundamentally by the assumption that dysfunction in specific brain regions will lead to deficits in the auditory processes associated with those regions. However, rather than focusing solely on the auditory manifestations of central processing disorders, our model also delineates the cross-modal deficits that can be expected to arise from dysfunction in a given brain region, as well as the higher-level cognitive, psychoeducational, and communicative ramifications. Thus, our model is firmly grounded in neuropsychology, or the study of brain-behavior relationships (Kolb & Whishaw, 1996) and, as such, results of central auditory assessment are viewed in light of the overall neuropsychological profile of the individual.

Second, like the model proposed by Katz et. al. (1992), ours is a descriptive model in which we relate specific findings on central auditory assessment to behavioral characteristics and complaints and additional speech-language results and academic difficulties. However, our model more precisely considers the relationship between the auditory and related presenting deficits and the underlying neuroanatomy and neurophysiology of the system, allowing for more finely delineated categorization. For example, in the Katz et. al. (1992; Stecker, 1998)
Subprofiles continued

Buffalo Model, both phonemic decoding (a primary auditory cortex – usually left hemisphere – task; e.g., Phillips & Farmer, 1990) and prosody errors (a nonprimary auditory – usually right hemisphere – task; e.g., Tomkins, 1995) are identified as characteristics of individuals with the subtype termed “decoding deficit.” In our model, the different neurophysiologic sites for phonemic and prosodic processing, combined with the different associated behavioral and neuropsychological sequelae associated with left hemisphere and right hemisphere dysfunction, respectively, is seen as evidence in favor of the need for differentiation between the two types of deficit and would, therefore, suggest entirely different management strategies.

Thus, the Bellis/Ferre model may be described as both a neurophysiologic and neuropsychological one, in which subprofiles are derived that encompass the whole of audition, from underlying auditory mechanisms to language, learning, and other higher-level, complex behaviors. The differential diagnosis and management of the individual with a CAPD is dependent on the administration of auditory-dedicated tests that have been shown to be sensitive to dysfunction in various brain regions. Findings on these tests are examined along with subtest analysis of speech/language, psychoeducational, cognitive, and neuropsychological measures for specific patterns. In this manner, a cohesive picture of the underlying deficient process(es) and implications for language, learning, and communication is achieved.

The remainder of this paper will provide for the reader a brief overview of the Bellis/Ferre model of central auditory processing disorders. As with any model of information processing, ours is a dynamic one that is continually revised as new information regarding neurophysiologic bases for behavior and treatment efficacy is brought to light. Therefore, although the basic components of this model have been described elsewhere (Bellis, 1996; Bellis & Ferre, 1996; Bellis & Ferre, in press; Ferre, 1997), some alterations in the model have occurred and will continue to occur. It is not within the scope of this paper to provide a detailed discussion of the neurophysiology of the auditory system, the assessment tools used, and the intervention strategies suggested; however, the reader is referred to the above sources for such detailed information.

CAPD Subprofiles: The Bellis/Ferre Model

This model includes three primary profiles of CAPDs and two secondary profiles. As will be seen, the three primary profiles represent auditory and related dysfunction in the primary auditory cortex (usually left hemisphere), nonprimary auditory cortex (usually right hemisphere), and corpus callosum (interhemispheric). The secondary profiles, on the other hand, represent dysfunction and associated sequelae that may rightly be considered to represent higher-level language, attention, and/or executive function and, therefore, some may argue against their inclusion under the umbrella of CAPD. At the present time, it is not possible to say precisely where audition ends and language/attention/output begins, and the two secondary profiles may be considered to reside in that gray area. They are included in our model of CAPD because they yield definitive findings on central auditory assessment and because auditory complaints often are the primary presenting factor for these individuals; however, it should be emphasized that their inclusion is not intended to suggest that CAPD cannot be differentiated from primary language and attentional deficits, or that all language and associated deficits arise from dysfunction in the auditory system. Finally, it should be noted that these profiles may occur singularly or in combination in a given individual with CAPD, and that not all individuals with auditory-related complaints will evidence a CAPD when central auditory assessment is undertaken.

Primary CAPD Subprofiles

Auditory Decoding Deficit. Individuals with Auditory Decoding Deficit exhibit difficulty hearing in situations in which noise or reverberation is excessive and frequently appear to “mishear” what is said to them, often substituting similar-sounding words for the actual auditory target. Spelling and reading decoding difficulties are common; however, this difficulty typically is confined to word attack abilities, or the ability to phonetically sound out/spell regular and nonsense words. Cognitive and related testing often reveal verbal skills poorer than visuospatial abilities, and receptive and expressive vocabulary often is weak. Poor sound blending abilities are noted frequently, as is difficulty learning foreign languages.

On central auditory assessment, the individual with Auditory Decoding Deficit exhibits bilateral deficit on dichotic speech tasks, combined with significant deficit on monaural low-redundancy speech tasks, a pattern that is suggestive of left-hemisphere dysfunction. It is likely that the word attack difficulties exhibited by individuals with Auditory Decoding Deficit are secondary to poor neural representation of acoustic/phonetic features of speech in the primary auditory cortex (Kraus, McGee, Carrell, Zecker, & Koch, 1996), leading to poor phonemic representation and speech-to-print skills. Likewise, other left-hemisphere-based tasks may be affected, such as the ability to analyze visual, auditory, and written information into its constituent parts. In essence, Auditory Decoding Deficit has characteristics similar to that of high-frequency hearing loss, in which portions of the auditory message are missing or poorly heard, thus leading to similar behavioral and educational complaints as those seen with hearing impairment.
Subprofiles continued
leading to similar behavioral and educational complaints as those seen with hearing impairment.

Management of individuals with Auditory Decoding Deficit includes components of traditional aural rehabilitative strategies used with hearing impairment. Environmental modifications to improve signal clarity are recommended, including preferential seating, visual augmentation, provision of a notetaker, and use of assistive listening technology. Drill-type speech sound training often is indicated, particularly focusing on stop consonants and other “hard-to-hear” contrasts. Activities to enhance auditory closure abilities via the use of contextual cues often are useful. Remedial reading activities focusing on the association of the speech sound with the orthographic symbol on the page (i.e., speech-to-print skills) frequently are an integral part of intervention for the individual with Auditory Decoding Deficit. Finally, the individual with Auditory Decoding Deficit should be counseled with regard to self-advocacy for listening, including recognition of adverse listening conditions and methods of dealing with them.

Prosodic Deficit. Prosodic Deficit, in essence, may be seen as the “flip side” of Auditory Decoding Deficit. Individuals with Prosodic Deficit often exhibit good word attack skills; however, sight word difficulties (i.e., the ability to spell and recognize irregularly spelled words in the language) are poor, due to inefficient gestalt patterning abilities. Comprehension of oral messages often is impacted, particularly if the message is linguistically complex; however, unlike with Auditory Decoding Deficit, individuals with Prosodic Deficit often will state that they “hear, but don’t understand.” In particular, the individual with Prosodic Deficit frequently comprehends the general content of the message, but misinterprets the intent, with particular difficulty understanding sarcasm, humor, and irony. Thus, social communication skills and pragmatics are often areas of weakness. Additional difficulty with comprehending the main idea of a spoken or written narrative and with taking notes during lecture-based classes are common. The individual with Prosodic Deficit may speak with little or no affect, and may be a monotonic, or flat, reader. Poor musical abilities are typical.

On central auditory assessment, individuals with Prosodic Deficit exhibit a pattern consisting of left-ear deficits on dichotic speech tasks combined with difficulty with both verbally labeling and humming tonal patterns (e.g., frequency and/or duration patterns testing; Musiek, 1994). This pattern is suggestive of right hemisphere dysfunction, as an intact right hemisphere is required both for processing of left-ear dichotic stimuli (Kimura, 1961) and for frequency/duration discrimination and tonal pattern perception (Zatorre, Evans, & Meyer, 1994). Other neuropsychologic and academic characteristics of the individual with Prosodic Deficit that are consistent with right hemisphere dysfunction include performance abilities often lower than verbal, difficulty with visual-spatial abilities, and difficulty with mathematics calculation and gestalt (part-to-whole) patterning (White, Moffitt, & Silva, 1992). Social-emotional concerns may be present due to deficient social judgement

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<td>Auditory Decoding Deficit</td>
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<td>Prosodic Deficit</td>
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<td>Integration Deficit</td>
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and social interaction skills, and individuals with Prosodic Deficit are prone to depressive disorders and, in extreme cases, may be at risk for suicide (Rourke, Byron, Young, & Leenaars, 1989). In short, the individual with Prosodic Deficit exhibits many of the typical complaints of right-hemisphere-based communication disorders and nonverbal learning disability (Badian, 1992; Rourke, 1989; Gross-Tsur, Shalev, Manor, & Amir, 1995), including symptoms common with attention deficit disorder (ADD).

Management techniques appropriate for the individual with Prosodic Deficit include placement with an animated teacher and specific therapy focusing on perception and production of suprasegmental aspects of speech (i.e., rhythm, stress, and intonation) and gestalt patterning skills. In addition, it often is necessary to include training activities that focus on searching for and extracting key words from oral or written narratives of increasing linguistic complexity. Psychological counseling for social/emotional concerns may be an integral component of the overall management plan, and speech-language intervention for pragmatics may be indicated. The individual with Prosodic Deficit likely will also require special educational services to improve math calculation and sight word reading abilities. Environmental modifications may include placement with an animated teacher and visual augmentation; however, use of assistive technology is seldom indicated as the individual’s primary difficulty is not related to clarity of the acoustic signal.

**Integration Deficit.** Whereas Auditory Decoding Deficit represents left-hemisphere dysfunction and Prosodic Deficit represents right-hemisphere dysfunction, Integration Deficit is a deficit in interhemispheric integration via the corpus callosum, or the large fiber tract that connects the left and right hemispheres. As such, auditory symptoms of Integration Deficit vary widely, and may include difficulty linking prosodic elements with linguistic content of a spoken message (Klouda, Robin, Graff-Radford, & Cooper, 1988); deficits in auditory verbal learning and memory; and syntactic, pragmatic, and semantic receptive language deficits (Dennis, 1981). Difficulty hearing in noise is common, not because of decreased intrinsic redundancy in the auditory system, as is the case with Auditory Decoding Deficit, but because a critical component of speech-in-noise skills, auditory localization and the concept of auditory space, is affected in cases of corpus callosum involvement (Lepore, Pito, & Guillemot, 1986). Phonological processing and decoding difficulties have been reported to be associated with inefficient interhemispheric integration, likely due to the difficulty combining component phonetic features into a gestalt pattern to achieve a cohesive whole (Temple, Jeeves, & Vilarroya, 1989).

The pattern of central auditory assessment results consisting of left-ear deficit on dichotic speech tasks and difficulty on temporal patterning tests requiring verbal labeling of the tonal pattern, with normal performance in the humming condition is characteristic of individuals with Integration Deficit (Musiek, Pinheiro, & Wilson, 1980). One will note the similarity of this central assessment profile when compared to that of Prosodic Deficit. The reason for this similarity is that a left-ear deficit on dichotic speech tasks may result from either right hemisphere or corpus callosum involvement. The use of temporal patterning tests provides the ability to differentiate between the two sites of dysfunction, as right hemisphere dysfunction will result in deficit in both the linguistic labeling and humming condition, whereas interhemispheric dysfunction affects only the linguistic labeling component. Therefore, it is critical that all test results be examined for patterns across findings, rather than merely interpreting one test in isolation.

Other neuropsychological findings consistent with interhemispheric dysfunction include difficulty combining visual and auditory input, bimanual coordination deficits, and difficulty with any other task that requires integration between right- and left-hemisphere-based activities (Lepore, Pito, & Jasper, 1986). As a result, individuals with Integration Deficit may do more poorly when visual or tactile augmentation is added. Instead, it is recommended that information be provided via one modality at a time, and can be reinforced via another modality provided the two do not occur simultaneously. Similarly, a notetaker should be provided in classroom situations so that the individual is not required to listen and write—a task that requires efficient interhemispheric dynamics.

Direct intervention techniques for the individual with Integration Deficit include many aspects of sensory integration therapy, and often the occupational therapist is an integral part of the management team. Specific activities designed to improve interhemispheric transfer (Bellis, 1996; Musiek & Chermak, 1995) are useful additions to the intervention plan. Finally, educational services directed at the specific academic difficulties exhibited by the individual are indicated.

**Secondary CAPD Subprofiles**

**Auditory Associative Deficit.** It could be suggested that this specific profile may be more properly considered an “auditory language” deficit rather than a CAPD, and such a suggestion would have merit. Auditory Associative Deficit is characterized by an inability to apply the rules of language to incoming auditory input. Receptive language skills are poor, including syntactic difficulties, especially with linguistically complex messages such as passive voice and irregular verb tenses. Semantic skills also are affected, with poor use and understanding of antonyms, categorizations, synonyms, or homonyms. Understanding words that have multiple meanings or negative “wh-” questions may be difficult for the individual with Associative Deficit. Written
Subprofiles continued

language may be impacted, with errors of punctuation, grammar, verb tense, and capitalization and use of run-on sentences, indicating difficulty with the use of the rules of the language. Frequently, learning a foreign language is particularly difficult for these individuals.

Individuals with Associative Deficit often will request clarification, rather than repetition, of information, and will state that he or she “didn’t understand.” Although Auditory Associative Deficit has many characteristics in common with receptive language disorders or, at its most severe, receptive childhood aphasia, it is included as a secondary CAPD profile because the difficulty is most pronounced with auditory/oral input and because a clear pattern of findings emerges on central auditory assessment. Specifically, individuals with Auditory Associative Deficit exhibit bilateral deficit on dichotic speech tasks, suggesting a left-hemisphere site of dysfunction. However, auditory closure and phonemic decoding abilities are intact, which indicates intact functioning of the primary auditory cortex. Therefore, the associative auditory cortex (usually left hemisphere), where acoustics and meaning come together and syntactic analysis occurs, is hypothesized to be the region of dysfunction in Auditory Associative Deficit.

Other associated difficulties characteristic of Associative Deficit include good reading decoding skills with poor passage comprehension abilities and poor understanding of math word problems, despite good calculation ability. It should be noted that early academic performance may be grade appropriate for the child with Associative Deficit; however, the deficit manifests itself at about the third grade level and becomes more obvious as the linguistic demands within the academic program increase.

Management for individuals with Auditory Associative Deficit includes rephrasing of information, using smaller linguistic units. Whole language environments rarely are appropriate for children with Auditory Associative Deficit, as such environments assume that the child will intuit the rules of the language via experience and exposure, without explicit training in such. Instead, the child with Associative Deficit will do far better in an environment that includes the use of a systematic, multisensory, rule-based approach to language and learning. Finally, speech-language therapy focusing on receptive language deficits and training in metalinguistic/metacognitive strategies to enhance auditory comprehension and memory are indicated.

Output-Organization Deficit. If Auditory Associative Deficit may also be considered a receptive language disorder, Output-Organization Deficit might be argued to represent an expressive language/executive function disorder. Individuals with Output/Organization Deficit exhibit difficulty in acting on incoming auditory information, including inefficient sequencing abilities, poor notetaking and assignment completion, and weak expressive language, articulation, and syntactic skills. The individual often complains of significant difficulty hearing in backgrounds of noise; however, even in quiet environments, he or she will indicate that he or she “heard it, understood it, but couldn’t remember it.” Like the organization deficit described by Katz (1992), Output-Organization Deficit is characterized by disorganization and impulsive or perseverative behavior both at home and at school or in the workplace.

This deficit is included in this discussion of CAPD for two primary reasons. First, difficulties often are more apparent when the auditory/verbal mode of information presentation is involved, and individuals may do better with written instructions. Second, specific auditory findings are apparent on central and peripheral auditory testing. Specifically, the individual with Output-Organization Deficit will perform poorly on any auditory task that requires report of more than three critical elements. In addition, and perhaps most revealing, contralateral acoustic reflexes frequently are elevated or absent in the individual with this deficit. On the other hand, performance on tasks requiring phonemic decoding and report of only one element, as with monaural low-redundancy tasks, is spared. Because of this pattern, along with associated behavioral difficulties described above, it is hypothesized that Output-Organization Deficit represents dysfunction in the efferent system, and likely also involves inefficient temporal-to-frontal intrahemispheric communication.

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<td>Auditory Associative Deficit</td>
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<td>Output/Organization Deficit</td>
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Subprofiles continued

Management of individuals with Output-Organization Deficit is similar to that of those with Associative Deficit, including placement in a systematic, rules-based learning or work environment, and provision of directions and information one step at a time. Training in the use of organizational aids often is useful, including making lists, using planning books and calendars, and reauditorizing to strengthen the memory trace. Speech-language therapy focusing on expressive language and word retrieval deficits typically is indicated, as is occupational therapy to address planning and execution skills. Because some of these individuals also exhibit motor planning difficulties, the physical therapist may be included in the management team, as well. Finally, because of hearing-in-noise difficulty presumably secondary to inefficient efferent function, the individual with Output-Organization Deficit may benefit from the use of environmental modifications designed to enhance signal-to-noise ratio, including use of assistive listening technology.

An overview of the two secondary CAPD subprofiles – Auditory Associative Deficit and Output-Organization Deficit – is provided in Table 2.

Conclusions

This paper has described a model of categorizing CAPDs in which subprofiles of auditory processing deficits are related to underlying auditory mechanisms as well as to higher-level cognitive, language, and learning difficulties. Although this model shares some characteristics with other subprofiling models that have been suggested in the literature, subtle differences are evident. Thus, the model described in this paper may be considered to be firmly grounded in both underlying auditory neurophysiology as well as to be representative of current neuropsychological tenets. Three primary profiles were described that represented primary auditory cortex, nonprimary auditory cortex, and interhemispheric dysfunction. In addition, two secondary profiles that probed the gray area between audition and language/executive function were offered. It is felt that the use of this model may greatly aid the practitioner in understanding the underlying processing deficits exhibited by individuals with CAPD, as well as in interpreting results of central auditory assessment and relating the findings to observed and reported academic, language, and behavioral complaints so that a management program may be developed that is both individualized and deficit-specific.

References


