We appreciate the opportunity to respond to Cacace and McFarland’s (2005) provocative article on the use of modality specificity as a framework to conceptualize and diagnose (central) auditory processing disorder [(C)APD]. We hope that their article and our response will serve to advance the authors’ stated intent to further the critical discussion of issues surrounding the diagnosis of (C)APD. We concur with Cacace and McFarland that “improving specificity of diagnosis is an imperative core issue to the area of CAPD” (p. 112). In fact, there are a number of points of agreement among ourselves and Cacace and McFarland. In this response, we...
will identify these points of agreement, as well as points upon which we disagree with Cacace and McFarland. We also will provide readers with our recommendations regarding current, best clinical practices for conceptualizing and approaching (C)APD, and we will identify areas for further research to advance clinical practice.

Cacace and McFarland (2005) characterize accurately two primary caveats regarding diagnosis of (C)APD: (a) diagnosis of (C)APD should not occur “in a vacuum” without regard to performance across other modalities, as such an approach might lead to the inappropriate diagnosis of (C)APD based on any performance deficits on tests of auditory processing; and (b) the efficiency of diagnostic tests of (C)APD should not be evaluated by imprecise criteria [e.g., “presumed” or “suspected” (C)APD], which do not provide accurate measures of the true sensitivity and specificity of these tests for diagnosis of central auditory dysfunction. We have advocated consistently and adamantly for a multidisciplinary approach to differential diagnosis of (C)APD, and we have emphasized the need to use tests that have demonstrated sensitivity and specificity for disorders of the central auditory nervous system (CANS; e.g., Bellis, 2003; Bellis & Ferre, 1999; Chermak & Musiek, 1997). These basic premises have been embraced and expounded upon in the recent technical report and position statement set forth by the American-Speech-Language-Hearing Association (ASHA) Working Group on Auditory Processing Disorders (ASHA, 2005a, 2005b).

**Conceptual Overview**

In their conceptual overview, Cacace and McFarland (2005) offer their opinion that (C)APD “has not reached the knowledge stage of development” (p. 113), that “this area of inquiry has stalled somewhere between the experimentation and consensus stages of development” (p. 113), that “validated models do not yet exist” (p. 113) for the treatment and management of (C)APD, and that the area of CAPD has become stagnant “such that it has not progressed in any meaningful way” (p. 113). However, the authors did not provide any references to the auditory processing literature in their conceptual overview to support their assertions or conclusions. Many audiologists and hearing scientists, as well as researchers in related fields of auditory and cognitive neuroscience, would disagree with their assertions on several counts. There have been considerable advances in our knowledge of (C)APD in recent years, and the accumulating body of literature supporting the existence of (C)APD and the methods of diagnosing and treating the disorder should not be ignored.

Although Cacace and McFarland (2005) assert that there is a paucity of research supporting the existence of (C)APD, an impressive body of controlled studies has been published in peer-reviewed scientific and professional journals that demonstrates the presence of abnormal neurophysiological representation of both speech and nonspeech acoustic stimuli in children and adults with listening and associated learning difficulties, as well as in animals (e.g., Clark, Rosen, Tallal, & Fitch, 2000; Cunningham, Nicol, Zecker, Bradlow, & Kraus, 2001; Fitch, Tallal, Brown, Galaburda, & Rosen, 1994; Jerger et al., 2002; Jirska, 1992; Jirska & Clontz, 1990; King, Warrier, Hayes, & Kraus, 2002; Kraus, 2001; Kraus et al., 1996; Musiek, Charette, Kelly, Lee, & Musiek, 1999; Purdy, Kelly, & Davies, 2002; Warrier, Johnson, Hayes, Nicol, & Kraus, 2004; Wible, Nicol, & Kraus, 2002). Studies also abound documenting that these neurophysiological abnormalities often are accompanied by patterns of deficits on psychophysical tests of central auditory function (e.g., Hendler, Squires, & Emmerich, 1990; Jerger, Chmiel, Tonini, Murphy, & Kent, 1999; Jerger et al., 1991; Jerger, Martin, & McColl, 2004; Jerger, Moncrieff, Addis, & Wambacq, & Seipel, 2000; Musiek et al., 1999; Rappaport et al., 1994). There also is increasing neuropsychological and psychophysical evidence that atypical interhemispheric transfer of auditory information may be a factor contributing to the listening difficulties seen in some children and in aging adults (Bellis, Nicol, & Kraus, 2000; Bellis & Wilber, 2001; Chmiel & Jerger, 1996; Chmiel, Jerger, Murphy, Pirozzolo, & Tooley-Young, 1997; Jerger, 1997; Jerger et al., 2002; Musiek, Gollegly, & Baran, 1984; Musiek, Pinheiro, & Wilson, 1980). In each of these studies, a global cognitive or a related disorder cannot account for the listening difficulties, nor can decreased peripheral hearing sensitivity. These studies provide compelling evidence of the existence of (C)APD as currently defined (ASHA, 2005a, 2005b). They also underscore the robustness and utility of both electrophysiological and psychophysical tests of central auditory function. Many of these tests (e.g., temporal pattern perception, dichotic listening) evolved from the classic work of Neff (1961) in animals and Kimura (1961) in humans, and an extensive body of research has accumulated documenting the relevance of the concepts they introduced to the clinical assessment of the CANS (e.g., Cranford, Igarashi, & Stramler, 1976; Cranford, Stream, Rye, & Slade, 1982; Hugdahl et al., 1999; Kileny, Paccioiretti, & Wilson, 1987; Musiek, 2004; Musiek, Baran, & Pinheiro, 1990, 1992; Musiek et al., 1980; see Bellis, 2003, and Chermak & Musiek, 1997, for a review). Despite the fact that some of these tests have been in use for several decades, they have not “outrived their usefulness” (p. 113), as Cacace and McFarland (2005) allege. In fact, one might argue that their longevity is an indicator of their robustness in diagnosing disorders of the CANS.

Moreover, Cacace and McFarland’s (2005) assertion that “validated models [of treatment] do not yet exist” (p. 113) for (C)APD is not fully representative of the state of knowledge regarding intervention. Although studies with high levels of evidence (e.g., randomized controlled trials and meta-analysis of such trials) are lacking, a solid base of evidence documents improved psychophysical performance, neurophysiological representation of acoustic stimuli, and listening and related function in children and adults following targeted auditory training (as well as in animal models; e.g., Hayes, Warrier, Nicol, Zecker, & Kraus, 2003; Jirska, 1992; Kraus & Disterhoff, 1982; Kraus et al., 1995; Merzenich, Grajski, Jenkins, Recanzone, &
Modality Specificity and (C)APD

In the body of their article, Cacace and McFarland characterize the ASHA (2005a, 2005b) position as a “unimodal inclusive framework.” In fact, these documents clearly state that, whereas tests with documented sensitivity and specificity for CANS dysfunction are necessary for a diagnosis of (C)APD, multidisciplinary (i.e., multimodality) input is critical for differential diagnosis. Demonstration of distinctive patterns across these multidisciplinary tests helps distinguish (C)APD from supramodal cognitive, language-based, and/or supramodal attention deficits. These patterns are derived from comparison of performance on behavioral tests of central auditory function and neurophysiological results from auditory evoked potentials with behavioral and neurophysiological measures of other sensory, language, and cognitive systems (Bellis, 2003; Bellis & Ferre, 1999; Chermak, 2004; Chermak, Hall, & Musiek, 1999; Chermak & Musiek, 1997). Further, the ASHA technical report also states that “it is recognized that individuals with (C)APD exhibit sensory processing deficits that are more pronounced in the auditory modality and, in some individuals, auditory-modality-specific effects may be demonstrated” (ASHA, 2005a, p. 2). This is entirely congruent with Cacace and McFarland’s assertion that “the primary deficit in CAPD should be linked directly to the processing of acoustic information; deficits should not be apparent (or at least should be manifest to a lesser degree) when similar types of information are presented to other sensory modalities” (p. 113).

The ASHA (2005a, 2005b) position also makes clear that (C)APD is “best viewed as a deficit in the neural processing of auditory stimuli that … is not the result of … dysfunction in other modalities” (ASHA, 2005a, p. 3) and emphasizes that it would be inappropriate to apply the label of (C)APD to listening difficulties exhibited by individuals with higher order, global, multimodal, or pansensory disorders (i.e., referring to higher level mechanisms that are common to and that support processing across all modalities as in attention-deficit/hyperactivity disorder [ADHD], autism, and cognitive delay) unless a comorbid deficit in the CANS can be demonstrated. This position is consistent with Cacace and McFarland’s (2005) assertion that “CAPD should be distinguishable from cognitive, language-based, and/or supramodal attentional problems in which modality-specific perceptual dysfunctions are not expected” (p. 113). Thus, both positions concur that “a key conceptual element for differentiating CAPD from other conditions is derived from the premise that CAPD represents an auditory perceptual dysfunction” (p. 113).

We do not agree, however, that it follows from this logic that (C)APD must therefore be defined as an exclusively modality-specific perceptual dysfunction that is not due to peripheral hearing loss. Rather, we define (C)APD as a primarily modality-specific perceptual dysfunction that cannot be attributed to peripheral hearing loss or higher order, global cognitive, attention, or related disorders. Therefore, although the ASHA (2005a, 2005b) documents reinforce the need for demonstration of CANS dysfunction for a diagnosis of (C)APD, they reject the concept of complete modality specificity as a diagnostic criterion for (C)APD. Further, it would appear from the arguments set forth by Cacace and McFarland (2005) that they, too, acknowledge the interrelatedness of processing and, by inference, also have rejected the notion of complete modality specificity in their conceptualization of (C)APD. This is evidenced by (a) their repeated use of terms such as primarily when discussing modality specificity of (C)APD; (b) their assertion, as noted above, that deficits in (C)APD “should be manifest to a lesser degree … when similar types of information are presented to other sensory modalities”; (c) their recognition that “sensory input can be ‘modulated’ by concurrent stimulation from other sensory modalities and/or by top-down influences” (p. 118); and (d) their discussion of polysensory processing areas within the cortex. Thus, Cacace and McFarland seem to agree with us that the expectation of absolute modality specificity of this (or any) processing disorder is unreasonable. It is this perspective that is expressed in the ASHA (2005a, 2005b) documents.

Where we diverge, however, regards the manner in which testing must be conducted to determine that an auditory deficit exists. Certainly, as Cacace and McFarland (2005) point out, any psychophysical measure can be affected by multiple factors due to the influence of higher order, non-modality-specific factors such as attention, memory, motivation, and decision processes, and the underlying multimodal, cross-modal, and supramodal neural interfaces supporting performance of these behavioral tasks and practically any other test that involves a behavioral response. Indeed, we have argued consistently for a number of years for testing methods and careful...
interpretation of test outcomes that reduce the potential confounding effect of factors not under direct examination in behavioral testing for (C)APD. These methods and strategies have included the following: (a) use of nonverbal stimuli or, alternatively, stimuli that carry a light linguistic load; (b) employment of intrasubject comparisons, including ear differences, intertest and cross-discipline (multimodal) analysis to rule out supramodal effects; (c) use of binural separation/integration tasks during dichotic listening (e.g., a consistent left-ear deficit, given symmetrical hearing sensitivity, is unlikely to result from a supramodal deficit); and (d) use of simple response mode (ASHA, 2005a; Bellis, 2002, 2003; Bellis & Ferre, 1999; Chermak et al., 1999; Chermak & Musiek, 1997; Jerger & Musiek, 2000).

We agree with Cacace and McFarland (2005) that “one way to evaluate the impact of … supramodal processes is to systematically vary the nature of the stimulus while holding all other factors constant” (p. 113), and methods that incorporate this approach have been suggested in numerous publications (e.g., Bellis, 2003; Chermak, 2003; Jerger & Musiek, 2000). Chermak (2003) noted that, although use of analogous modality testing may identify sensory system deficits in other modalities, it still may be insufficient to assess fully cognitive and pansensory issues. Instead, she argued that testing of executive function also is essential to evaluate more clearly supramodal/pansensory status versus multimodality function.

Keeping in mind the real and practical challenges of the clinic, we would argue (and have recommended) that there are other ways to examine multimodality issues beyond varying the stimulus, which is the fulcrum of Cacace and McFarland’s (2005) suggested approach. The equivalence of multimodal tests that differ only in sensory stimuli has not yet been demonstrated sufficiently. Cacace and McFarland cite no literature demonstrating that these purported analog tests are indeed analogous. For example, how does one define a true visual analog of frequency discrimination when central integration is organized differently in the visual and auditory systems? Further, what would be the functional relevance of such a visual analog? In the absence of documented comparability, it is not clear that so-called analogous tests offer meaningful comparisons. Further, we cite literature below to support our position that the neural substrates underlying these tests are not modular. Of further concern is the lack of data supporting the sensitivity and specificity of these multimodal analogs to differentially identify known central auditory versus pansensory dysfunction. In fact, it is not entirely clear how such sensitivity and specificity could be demonstrated for analogous tests. Therefore, although some studies recently have begun to investigate the use of multimodal analogs in the differential diagnosis of (C)APD and pansensory deficits such as ADHD (e.g., Ross & Bellis, 2005), the clinical utility of these analogs remains questionable at this time. Instead, we contend that multidisciplinary testing as well as testing analogous processes within other sensory systems, interpreted within the context of an interactive brain composed of interfacing sensory, cognitive, and linguistic networks, will in combination with the sensitized test battery of the CANS lead to accurate diagnoses that will guide treatment and management of (C)APD.

Another concern is the clinical feasibility of administering a multimodal test battery such as that suggested by Cacace and McFarland (2005). First, how many modalities must be assessed before one concludes that a disorder is restricted to a single modality? Moreover, is it within the audiologist’s scope of practice, clinical education, or clinical training to administer tests that assess visual, somatosensory, and perhaps other modalities? Do we, as audiologists, have sufficient training in these related areas to do this? Should we be trained in these areas, considering our unique role as specialists in auditory and vestibular system function and the access to other professionals whose scopes of practice and areas of expertise encompass these areas? We think not. We assert that it is more reasonable to adopt a multidisciplinary approach to differential diagnosis of (C)APD, as elaborated in the ASHA (2005a, 2005b) documents and in our own publications cited previously. Such an approach encourages professionals with the relevant expertise and scope of practice to obtain validated measures of other modalities and of higher order cognitive, language, attention, and related function (e.g., neuropsychologists, educational diagnosticians, and speech-language pathologists).

In addition, although specific paradigms to evaluate analogous tasks in multiple modalities have been described by Cacace and McFarland and others (e.g., Chermak, 2003; Ross & Bellis, 2005; Voyer & Boudreau, 2003), it is important to note that many of these tests are not currently available to clinicians engaged in diagnosing and treating auditory disorders. Similarly, although we agree that testing “under computer control and tasks … embedded within forced-choice psychophysical paradigms” (p. 115) is preferable, this approach also is not yet clinically feasible in most audiology clinics. For a test to translate successfully from laboratory to clinic, it must be relatively straightforward and easy to administer, score, and interpret, and it must use equipment that is commonly available in clinical practice. It is for these reasons that there have been many research paradigms described for evaluating central auditory function that have not yet made their way into general clinical use (e.g., click lateralization, tests of localization, cross-channel gap detection, backward masking, and interaural timing procedures). Although we strongly support ideas and concepts that will spark research that leads to better diagnosis and treatment of the patient, we emphasize that these tests and procedures must be clinically feasible so that they can be incorporated within the constraints and conditions of typical clinical settings. It must be kept in mind that the step from the lab to the clinic is, indeed, a giant one, and one that can only proceed incrementally. Therefore, at present, we recommend that a comprehensive, multidisciplinary evaluation of patients suspected of (C)APD is the most appropriate and clinically feasible method of assessing central auditory function while, at the same time, ruling out higher order, global, or supramodal disorders that may masquerade as (C)APD.
We see another flaw in Cacace and McFarland’s (2005) argument for multimodal comparisons. The concept of cross-modality comparisons is based on the assumption of independent function of each modality. Although this independence may be demonstrated some of the time, there are several instances in which it cannot be demonstrated. For example, the well-recognized disorder multiple sclerosis may affect the auditory system as well as other systems (e.g., Levine et al., 1993). A diagnosis of the CANS deficit (and, thus, appropriate rehabilitation of that disorder) should not be withheld because other systems are involved. Likewise, individuals who have suffered head trauma may exhibit a distinct and verifiable central auditory deficit along with dysfunction in other sensory systems subserved by the same injured brain regions (e.g., Musiek et al., 2004). To not diagnose and treat the auditory component of the brain injury would do a grave disservice to the patient.

Medical conditions in which unrelated impairments occur in more than one sensory modality pose another problem for Cacace and McFarland’s (2005) view that a unimodal framework is “unable to delineate modality-specific processes from more generalized dysfunction” (p. 112). For example, despite the presence of dysfunction in two modalities, Usher’s syndrome would not accurately be characterized as a supramodal or pansensory deficit. Nor would such a label be appropriate for a patient who exhibits neuropathies across sensory or other systems, as is often the case with auditory neuropathy/dysynchrony (see Rapin & Gravel, 2003, for review). In these examples, the presence of a documented auditory deficit leads to a diagnosis of auditory dysfunction that is then amenable to auditory rehabilitation. At no time is it suggested that the presence of dysfunction in other modalities negates the diagnosis of the auditory deficit. As argued previously, multidisciplinary assessment is essential to fully evaluate the sensory, cognitive, and language systems that may be contributing to the patient’s disability and to begin the appropriate course of intervention.

The “label” of (C)APD is not an exclusive label. An individual can have central auditory dysfunction that presents comorbidly with other valid diagnoses such as dyslexia, specific language impairment, attention deficit, and learning disability (e.g., Cunningham et al., 2001; Gomez & Condon, 1999; Kraus et al., 1996; Moncrieff & Musiek, 2002; Pillsbury, Grose, Coleman, Conners, & Hall, 1995; Purdy et al., 2002; Riccio, Hynd, Cohen, Hall, & Molt, 1994; Tillery, Katz, & Keller, 2000; Warrier et al., 2004; Wible et al., 2002; Wible, Nicol, & Kraus, 2005; Wright et al., 1997). In cases of comorbidity, the (C)APD must be diagnosed fully and accurately and a treatment program must be developed and implemented by a team of professionals to address all significant functional deficits.

Finally, we agree that, as Cacace and McFarland (2005) assert, “the most effective interventions will be based on accurate knowledge of the problem” (p. 117). We are convinced, based on the literature and on our clinical experience, that the use of central auditory tests, when administered and interpreted appropriately by well-educated, trained, and experienced clinicians, will provide information that (a) is sensitive and specific to disorders of the CANS, (b) can be used to determine whether central auditory dysfunction is present in a given individual, and (c) offers, when combined with multimodal (multidisciplinary) evaluations by other professionals, the best basis for obtaining “accurate knowledge of the problem” in clinical practice. Further, such an approach also satisfies the criterion that higher order global, supramodal or pansensory cognitive, language, attention, or related disorders be ruled out when diagnosing (C)APD.

The Gold Standard

We agree with Cacace and McFarland (2005) that too many studies attempting to validate diagnostic tests of central auditory function have been imprecise and based on an inadequate standard of “presumed” or “suspected” (C)APD (e.g., Keith, 1986; Singer, Hurley, & Preece, 1998). However, a considerable number of lesion studies, which were not cited by the authors, demonstrate the effects of various types and levels of lesions on the currently used central auditory test battery. As such, although an absolute gold standard may never exist due to the heterogeneity of disorders affecting the CANS, it is clear that test efficiency measured on subjects with well-defined lesions of the CANS provides an important guide for establishing the validity of central auditory diagnostic tests. Such studies demonstrate the sensitivity and specificity of our current central auditory test battery. Thus, we advocate for the use of measures that have been shown to be sensitive and specific to documented dysfunction in the CANS, as reflected in our previous writings and in the ASHA (2005a, 2005b) documents.

As previously discussed, the foundation of central auditory assessment and diagnosis of (C)APD dates back to the classic work of Neff (1961) in animals and to Kimura (1961) in humans. Since that time, a vast corpus of work has been completed documenting both the behavioral and electrophysiological results in animals and humans with confirmed lesions of the CANS who exhibit evidence of auditory difficulties despite normal peripheral hearing (e.g., Cranford et al., 1976, 1982; Hugdahl et al., 1999; Kileny et al., 1987; Musiek, 2004; Musiek et al., 1980, 1990, 1992; see Bellis, 2003, and Chermak & Musiek, 1997, for a review). These human and animal subjects have true central auditory deficits. If certain test patterns have been demonstrated to have good sensitivity and specificity in these laboratory cases of confirmed CANS lesions, then one may presume a high degree of likelihood that the same pattern of test results, when observed in an individual undergoing testing for central auditory dysfunction, confirms a (C)APD in that individual. This use of pattern analysis of performance on tests validated through lesion studies to infer brain–behavior relationships in clinical populations is not new to the field of (C)APD. Indeed, it is a well-accepted and time-honored approach that has been used for decades in neuropsychology and related fields.

We agree with Cacace and McFarland (2005) that if lesion studies are used to validate central auditory tests,
then the lesions, ideally, should be restricted to areas exclusively involved in auditory processing. This ideal is difficult to attain, however, given the organization of the brain (as discussed below) and the varied causes of some cases of central auditory dysfunction (e.g., accidents, injuries). Nonetheless, this strict standard has been met in some studies, and in others the lesions are reasonably circumscribed (e.g., Baran, Bothfeld, & Musiek, 2004; Baran, Musiek, & Reeves, 1986; Musiek et al., 1980). However, central auditory lesions often extend beyond artificial boundaries that are increasingly recognized as inaccurate reflections of true brain organization. Because lesions, including central auditory lesions, may be less than circumscribed, involving multiple areas and levels of the brain, it is to be expected that brain–behavior relationships will be less than perfect in individuals with CANS dysfunction. More importantly, because of the lack of modality specificity of most brain regions, as discussed below, even relatively circumscribed lesions affecting areas involved in auditory processing likely will also result in comorbid dysfunction in other systems due to shared neurophysiological substrates (e.g., corpus callosum, striatum, insula).

We disagree with Cacace and McFarland’s (2005) argument that demonstration of modality specificity via multimodal measurements provides an objective criterion for validating tests of (C)APD. While it is true that lesions are sometimes poorly defined and not completely restricted to auditory regions, human lesion studies remain the most objective criterion for validating tests of (C)APD. As we have stated previously, multimodal testing is important, especially for accurate differential diagnosis; however, as elaborated above, multimodal analogs of central auditory tests cannot be used to validate specific measures of central auditory function. In fact, given the paucity of evidence supporting modularity in the CNS (as discussed below), one cannot ascertain the physiological status of the neural substrate upon which each modality being tested is dependent. Moreover, each of the so-called modality-specific tests would need to have demonstrated high sensitivity and specificity to make the claim that one modality is normal whereas another is not. It is unclear as to how evaluation of the sensitivity and specificity of each of the modality-specific tests would be accomplished; that is, what would be the gold standard for validation of these multimodal analogs? Until the foregoing issues are resolved, it is our opinion that it would be inappropriate to recommend use of multimodal analogs for validation of diagnosis of CANS dysfunction. Therefore, despite their limitations, lesion studies clearly still offer the best way to validate tests of central auditory function and to provide some guide as to which tests may be most efficient to use (ASHA, 2005a, 2005b).

**Brain Organization: Nonmodularity and the Nature of (C)APD**

Cacace and McFarland (2005) state that their “view” of the neuroscience literature diverges from that expressed in the ASHA (2005a) technical report. We trust that we have demonstrated, however, that Cacace and McFarland have incorrectly classified that framework as a unimodal, inclusive framework when in fact the true framework underlying the conceptualization of (C)APD as described in that report incorporates a multidisciplinary and multimodal perspective. Cacace and McFarland sum it up themselves in stating, “Although we cannot say for certain, it is probable that there is not a sharp demarcation, so that some border areas will be found to be mainly, but not entirely, sensitive to auditory stimuli (i.e., predominantly specialized for auditory processing and driven mainly by auditory stimuli), while others will not” (p. 120). This is precisely our point. Moreover, Cacace and McFarland state:

Multimodal processing may occur relatively early, or it may occur later in time. Likewise, multimodal processing may involve primary sensory cortex, or it may be largely restricted to higher order sites. This issue of early versus late fusion of input from different sensory modalities is currently the subject of much experimentation and debate. The eventual outcome of this issue will determine whether modality-specific processes are best seen as being restricted to early, low-level processes or whether they might also apply to later, higher level processes. It is our contention that this issue will determine the extent of the domain of CAPD, an issue that remains to be resolved. (p. 120)

We agree that the resolution of this issue will require additional research and will affect not only our conceptualization of (C)APD but that of all complex, clinical entities involving deficits in reading, memory, attention, and language representation and processing.

Although there may be some brain regions that are auditory-specific, we agree with Cacace and McFarland (2005) that neurons in these areas respond “primarily” (not exclusively) to auditory stimuli. We argue, however, that the complexity as well as the geographic proximity of these areas to polymodal association areas render it unlikely that only the tiny auditory-specific area would be affected by dysfunction or insult to these primarily auditory regions. Cacace and McFarland cite some literature to support their argument for absolute modality-specific divisions of the cortex but omit several more recent references that argue against complete modality specificity. For example, Sams et al. (1991) demonstrated that neuronal activity in even the primary auditory cortex (an area that Cacace and McFarland would suggest is modular) is modified by visual input. Similarly, Calvert et al. (1997) more recently demonstrated that these areas formerly thought to be sensitive only to auditory stimuli are activated during solely visual tasks.

Cacace and McFarland (2005) note that the evidence for modularity is not definitive and, in their discussion of this topic, cite findings that actually support a non-modality-exclusive viewpoint. For example, they present the finding of polysensory regions in the superior temporal sulcus recruited to support McGurk-like effects. They cite Booth et al.’s (2002) findings that both modality-specific and polysensory activations were observed during judgments of semantic relatedness of words presented in the visual.
and auditory modalities. Moreover, they admit that “multisensory” neurons are rare, but not nonexistent, in rat cortical neurons. Further, they cite Wallace, Ramachandran, and Stein (2004), who found that “multisensory” neurons were rare, but again not nonexistent, within the major modality-specific domains. We would add that auditory neurons in the cerebrum exhibit interconnectedness with a variety of neurons in other nonauditory areas of the brain. In Streifeld’s (1980) excellent review of this topic, she points out the interconnections between specific brain regions and other areas with totally different functions. For example, the auditory cortex in primates has direct and indirect connections to the limbic system, cingulate gyrus, hippocampus, and frontal lobe. Bamiou, Musiek, and Luxon (2003) reported the presence of considerable auditory activity in the insula—a structure not usually considered an auditory region. Additional areas of the brain that have been identified as auditory responsive include the amygdala, striatum, and frontal lobe, among others (e.g., Poremba et al., 2003; Salvi et al., 2002). Thus, this literature as well as the research cited by Cacace and McFarland support nonmodularity and nonexclusively segregated brain organization or boundaries.

Our position derives from more recent studies (e.g., Poremba et al., 2003; Salvi et al., 2002) that demonstrate a great deal of interaction among even those areas that were considered previously to be sensory specific. These studies show that auditory tasks (e.g., listening in noise) activate auditory and nonauditory areas of the brain, including areas involved in attention, executive control, working memory, language processing, and motor planning. Consider the growing evidence of the involvement of cognitive processes in basic perceptual events. For example, working memory has been shown to be integral to numerous auditory processes, including localization, temporal resolution, and pattern recognition (Marler, Champlin, & Gillam, 2002; Martinkauppi, Rama, Aronen, Korvenoja, & Carolson, 2002; Zattore, 2001). Taken together, these studies demonstrate clearly that polysensory regions are located within modality-specific regions and raise serious questions regarding the likelihood that a disorder will affect one small, possibly modular area, leaving a polysensory area a micron away unaffected. It seems clear that complete modality specificity is neurophysiologically untenable, as stated by ASHA (2005a, 2005b).

Following their argument for a modality specificity requirement for diagnosis of (C)APD, Cacace and McFarland (2005) state that their “position does not exclude the possibility of modality-specific linguistic or nonlinguistic processes, attention, memory, and so on” (p. 113). They reference Polster and Rose (1998), who demonstrated dissociation in cases of cortical deafness, pure word deafness, and auditory agnosia. It should be noted, however, that in many cases of central (cortical) deafness, the dissociations are not as clear as Cacace and McFarland suggest. For example, it has been demonstrated that individuals with pure word deafness not only exhibit word deafness (i.e., a linguistic deficit) but also other auditory (i.e., nonlinguistic) deficits, such as extremely poor frequency and/or intensity discrimination that often are not reported because in many cases they were not tested (Buchman, Garron, Trost-Cardamone, Wichter, & Schwartz, 1986; Musiek, Baran, & Pinheiro, 1994). In addition, individuals with central deafness often exhibit compromised cognitive abilities of varying degrees, accompanied by other deficits including language, music perception, and speaking (Habib et al., 1995; Musiek et al., 1994; Tramo, Bharucha, & Musiek, 1990). In these cases, the degree, types of deficits, and dissociations are related to the locus and extent of the neural damage. It is interesting to note Cacace and McFarland’s statement that “it is not certain to what extent similar dissociations can be shown in other populations suspected of having CAPD, such as children with learning disabilities or the elderly” (p. 116). Until and unless such evidence can be found in the literature, we conclude that it is premature to ask the professional community to adopt a strict, absolute modality specificity requirement for diagnosis of (C)APD.

**Relationship Between (C)APD and “Meaningful Disability”**

Finally, we take issue with Cacace and McFarland’s (2005) characterization of (C)APD as a disorder that is not “actually associated with a meaningful disability” (p. 114). It should be remembered that these clients come to our clinics for evaluation because of listening problems that typically are affecting communication, learning, language processing, attention, and related areas. The finding that performance on selected measures of fine-grained auditory processing does not predict future academic skills (e.g., Watson et al., 2003) is irrelevant. The component processes and skills that ultimately influence academic success are vast and certainly many levels of operation removed from central auditory processing. While links between inefficient auditory processing and language or learning problems have been documented both behaviorally and electrophysiologically (e.g., Bellis & Ferre, 1999; Kraus et al., 1996; Moncrieff & Musiek, 2002; Wible et al., 2005), (C)APD is not posited as a direct cause of all or even most cases of academic failure, learning disability, reading disability, and so on; however, as is true of many disorders, (C)APD certainly can exacerbate academic challenges (e.g., listening in noisy classroom environments). As stated by ASHA (2005a, 2005b) and previously (e.g., Bellis, 2003), the complexity and heterogeneity of both learning disorders and (C)APD preclude a simple one-to-one correlation in large groups. Instead, the relationship between (C)APD and learning depends on the nature of both the (C)APD and the learning issue. Further, auditory processing is just one small, albeit important, aspect of listening, learning, and communicating, and many other factors combine ultimately to determine learning and communication success.

From a clinical perspective, the audiologist’s job is not to predict future academic skills, though certainly this is of interest. Our responsibility is to assess and evaluate auditory (and vestibular) function. Posttherapy improvements on central auditory tests and other psychoacoustic measures document the effectiveness and efficacy of (C)APD intervention directed toward improving central
Conclusions

McFarland and Cacace (1995) discussed three categories of individuals who perform poorly on tests of auditory function: (a) those who represent (C)APD in its “purest” form and perform poorly solely on auditory tests, (b) those who exhibit auditory perceptual problems that coexist with other specific processing problems and who thus present with a mixed pattern of deficits, and (c) those who perform poorly on auditory tests because of a global, supramodal problem involving, cognition, attention, language, memory, or related skills, and who perform poorly on both auditory and visual tasks. In this categorization scheme, the authors recognize the possibility of comorbidity of disorders. We maintain that we must be able both to identify (and, thus, rehabilitate) the auditory deficits present in individuals falling into the first two categories while, at the same time, “ruling out” those individuals who fall into the third category. Through our recommended approach that combines multimodality (multidisciplinary) evaluation along with specific tests of central auditory function that have been demonstrated to be both sensitive and specific for disorders of the CANS, we are able to achieve this goal at the present time.

We acknowledge that further research will render us even better able to make such distinctions. We encourage Cacace and McFarland to continue their work to help us develop tools that are both efficient and practical for the examination and differential diagnosis of (C)APD. We also encourage our multidisciplinary colleagues to continue to assist us in disentangling clinical profiles and diagnoses by working with audiologists and speech-language pathologists to build the multimodal context needed to differentiate diagnoses, especially in those who appear to present with related, comorbid disorders.

Finally, we should point out that the ASHA (1996) technical report on which the majority of Cacace and McFarland’s arguments are based has been superseded by the ASHA (2005a, 2005b) technical report and position statement, which forms the basis for our present conceptualization of (C)APD, as set forth in this critique. We note that there appears to be a great deal of congruence between the views espoused by Cacace and McFarland and the approach advocated in the ASHA (2005a, 2005b) documents and in our own previous writings. Thus, we contend that, at present, there is sufficient evidence both to support the existence of (C)APD as a meaningful clinical diagnostic entity and to provide guidance for valid and reliable diagnosis and treatment of the disorder. At the same time, we acknowledge the need for and encourage further research to explore the range of issues and questions raised in Cacace and McFarland’s article and in this response.

References


Musiek et al.: Implications of Nonmodularity for (C)APD 135


