Central Auditory Processing and Disorders

Sanford E. Gerber, and George T. Mencher

To deal with central auditory disorders, it is essential for treatment that we differentiate them from peripheral auditory disorders. This is not to say that a given patient can't have both. But where is the line, if there is one, separating peripheral from central? Young and Protti-Patterson (1984) reviewed the "top-down" and "bottom-up" notions of central auditory processing problems. The "bottom-up" theorists claim that language processing is a function of basic auditory processes, while the "top-down" theorists advocate that higher order auditory cognitive processes cannot be distinguished from basic auditory processes. As is typical of work in this field, Young and Protti-Patterson concluded that both groups of theorists are correct. Both are represented in these pages *What is central?*

Where does the bottom end and the top begin? The contributors to this issue of Human Communication Canada met, and took as a charge an attempt to define central function. Such a definition is necessary as a basis for the further discussion of how central function becomes disordered and how such a disorder may be diagnosed and treated.

We first approached this definition anatomically: central auditory function is that which occurs after the reponse of the VIII nerve. An imaginary line separates bottom from top, or peripheral from central, at the point where the VIII cranial nerve finds the cochlear nucleus. Hence, disorders reflected behaviourally and/or physiologically in the sound conducting mechanism of the external and middle ears, the cochlea, its afferent projections, and the VIII nerve itself, are defined as peripheral. Central auditory processing and central auditory disorders begin anatomically at the level of the cochlear nuclei (Dublin, 1978).

Those who are more educationally and less physiologically oriented may prefer the definition of the American Council on Learning Disabilities that the term central auditory dysfunction is a generic one referring "to a heterogeneous group of disorders manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning, or mathematical abilities." The ACLD also tells us that these disorders are "presumed due to central nervous system dysfunction." It is this presumption which makes the difference, because that menu of symptoms could describe peripheral auditory disorders just as well. Children who are born with severe sensory-neural hearing impairments indeed have difficulties in the acquisition and use of listening, speaking, etc. The Canadian Council on Learning Disabilities goes beyond the mere presumption of central nervous system dysfunction, and claims that these disorders are "due to identifiable or inferred central nervous system damage." There is probably no debate over the identifiability; it is the inference or presumption which usually gives us problems.

Of course, modern technology — both behavioral and electro-physiological — may improve our ability to presume or infer central nervous system dysfunction. At least, some of these techniques permit us to identify peripheral dysfunction as defined. Hence, the answer to the question "What is central?", at least for the moment, is that central auditory dysfunction will have an identifiable or presumed or inferred pathology of the central nervous system at the level of the cochlear nuclei or higher. Again, this does not preclude the possibility or probability of peripheral dysfunction, especially since peripheral and central dysfunction could very well have a common etiology.

Over 30 years ago, Myklebust (1954) made the point that "early life aphasia" can be confused with other things affecting the growth and development of language. This led him to use the term "auditory disorders due to aphasia". That is probably what we are discussing. He described the disorder as one of symbolic function, or an inability to comprehend the spoken language of others, or an inability to speak. He referred to "an inability to use language internally for purposes of thinking of oneself." He told us that such a child does not "initially acquire normal symbolic behavior." He even used the same anatomical distinction we have used, specifying that central deafness is present when lesions occur between the cochlear nucleus in the medulla to the temporal lobe. He even endorsed what we have called the "bottom-up" theory, sayng that such a disorder represents "a deficiency in transmitting auditory impulses to the higher brain centers..." One role of the papers in this journal is, at least in part, to test the veracity of Myklebust's suppostions. Have we learned anything in the last 30 years?

Sanford E. Gerber, Ph.D.
Professor of Audiology
University of California
Santa Barbara, CA 93106
and
George T. Mencher, Ph.D.
Nova Scotia Hearing & Speech Clinic
5599 Fenwick Street
Halifax, Nova Scotia.

What is central auditory dysfunction?

Clinically, central dysfunction can be identified when there is evidence of an auditory disorder in the absence of only peripheral dysfunction which would explain that disorder. This dysfunction can take many different forms and be exhibited in varying degrees of severity. The most prominent revealing symptom is difficulty in the perception of speech and the auditory comprehension of language. The central auditory pathway may be disordered as a result of (documented) neurological deficit or lesion or as a result of disordered brain function not associated with demonstrable neuroanatomical lesions. Patients manifesting the first type of disorder include the following: genetic, traumatic, inflammatory, neoplastic, autoimmune, vascular, toxic, epileptic (as in epileptic aphasia), malformation, transneural degeneration, and unknown causes. Patients manifesting a second type of disorder may have functional, regional blood flow, or neurochemical differences which are not detectable by currently employed techniques. Still others may be socially or culturally different such that they may be identified as language impaired, learning disabled, verbal agnosic, or by means of other diagnostic labels. For children diagnosed as having attentional deficit disorders of hyperactivity, the possibility of a contributing or consequent central auditory disorder must also be considered. We recommend that children manifesting or suspected of having central auditory disorders be studied by means of the following techniques and procedures:

a) Electrophysiological techniques and procedures.

The auditory brainstem evoked responses which provide information about the integrity of both the lower and upper relays of the brainstem should be supplemented, as appropriate, with frequency following response measures. In some cases this may be important to deliniate cochlear versus retrocochlear pathology, or it may be useful when there is reason to doubt brainstem findings which conflict with those obtained from cochleograms. Middle potentials, thought to reflect activity in the geniculate bodies and primary auditory cortex, and late potentials to verbal and non-verbal stimuli are both essential. These must also include related potentials measured during discrimination tasks if one is to differentiate cochlear deafness from auditory agnosia and/or verbal auditory agnosia. Preference should be given to techniques providing a mapping of responses over the entire scalp.

Behavioral and electrophysiological assessments should be used to complement and validate one another. EEG is essential for all children with severe comprehension deficits. The use of spectral analysis is desirable. Other techniques, such as nuclear magnetic resonance imaging, hold great hope. Unfortunately, techniques such as measurement of cerebral blood flow and positron emission tomography may be very informative, but will probably have limited application except in children with severe illnesses.

Although written with the peripherally hearing impaired child in mind, recommendations of the Elks Conference held in Saskatoon (Gerber & Mencher, 1978), which focused on the early diagnosis of hearing loss, are applicable here. Specifically, recommendations 5 and 6 are germane (See Table 1).

Table 1

Whereas, physical examination in the diagnosis of hearig loss in infants adds greatly to the total information about the child and to the understanding of the etiology of the hearing loss;

Resolved: A comprehensive assessment of any child suspect for hearing loss should include these procedures:

A. *Essential* to the Assessment
   1. Standard pediatric examination
   2. Pneumatic otoscopy and/or oto-microscopy
   3. Fundoscopic examination
   4. Appropriate observations for specific physical abnormalities (See Appendix on pages 21-29, Gerber, S.E. and Mencher, G.T. (eds.) *Early Diagnosis of Hearing Loss).*

B. *Strongly Recommended in the Assessment*
   1. General laboratory examinations
   2. Appropriate serology examination for toxoplasmosis, rubella, cytomegalovirus and Herpes
   3. Urinalysis
   4. Family audiograms

C. *Include When Indicated*
   1. Thyroid function
   2. Polytomography of middle and inner ear (Except in established cases of antenatal infections)
   3. Electrocardiogram
   4. Chromosomal study
   5. Flourescent trepanemal antibody (FTA) absorption tests for syphilis
   6. Appropriate testing for Mucupolysaccharidosis

Whereas, correct diagnosis and appropriate treatment require as much information as possible about the auditory function of a suspect infant;

Resolved: A comprehensive auditory assessment of a suspect infant should include:
   A. An extensive behavioral history by parental report
   B. Observations of behavioral responses to appropriate auditory stimuli
   C. Visual Reinforcement Audiometry (where age appropriate)
   D. Acoustic immitance measurements (includes tympanometry, acoustic reflex, and static compliance)
   E. Electric response audiometry as indicated

b) Behavioral Assessment.

These assessments should include behavioral audiometry, visual reinforcement audiometry (VRA), tangible reinforcement operant conditioned audiometry (TROCA), play audiometry, conventional and sensitized audiologic procedures as appropriate, and otoimmitance

techniques. Assessment should include measures of auditory function as well as visual and tactile perceptual function. This can be accomplished by tests of discrimination, identification, sequencing, and serial memory for speech and non-speech stimuli, and supplemented with verbal and non-verbal stimuli in the visual and tactile domains. There should be emphasis on the rate of auditory and visual processing. Detailed speech, oral motor, and linguistic examinations — phonologic, syntactic, semantic, and pragmatic — should be included.

c) Pathology assessment

Post mortem studies in patients with documented congenital profound sensory-neural hearing loss, including those with absence of the cochlea and those with a severe bilateral hearing loss, are strongly recommended. Detailed post mortem examination is also recommended in children with documented involvement of the central auditory pathway particularly those with word deafness, dyslexia, congenital dysphasia, etc. It is also very important to document the presence of transneural degeneration in the central auditory pathway. Vestibular testing is also considered essential in children with serious auditory disorders as deficits may influence motor development. This problem may also lead to an unnecessarily detailed examination of the central auditory system, when in reality the children have peripheral problems.

Treatment

What are the indicators for treatment of central auditory processing disorders whether in children or adults, congenital or acquired? Perhaps, as Sloan (1985) indicated there are three important questions to be answered first:

1) What is the nature of the disorder? Is it fixed or can performance be modified?
2) If performance can be modified, what conditions improve it and can that improvement be maintained when those conditions are removed?
3) If the deficit is fixed, what compensatory skills can improve function?

We still lack a data base to definitively identify infants and young children who may be at high risk for subsequent language and learning delay or disability. Young, pre-linguistic, high risk children should be followed longitudinally to determine if they do develop problems. If they do, we need to describe in detail the different profiles and subgroups that emerge. There is a need to determine whether the same or different intervention approaches apply equally to all subgroups. Intervention approaches (both goals which are in current use and those which may be developed) should include the broad range of services and treatments which must be implemented early. Empirical investigation of each stage of this process is urgently needed to improve our understanding of central auditory disorders and possible approaches to intervention.

Thus far, intervention has been considered from the point of view of aiding the severely involved child, one with an obvious disorder. However, there are milder, less obvious involvements. Hyperactivity, emotional lability, and perhaps even some memory dysfunction can be related to less traumatic lesions. Older children — who use compensatory strategies but who are having problems in school and problems in their families — begin to be called by other disorders. Adults have similar difficulties at work and at home. Somehow, our diagnostic and intervention programs haven't extracted the subtler or milder forms of central auditory disorders. For some patients, even a mild disorder may be a serious problem; if not in speech perception, then in reading or learning or behavior. It then becomes difficult to discriminate between central auditory dysfunction and learning disability, and this too may have therapeutic implications.

Keith (1985) touched on this issue too, indicating how confusing it is. He pointed out that the name for the problem changes from central auditory disorder to auditory perceptual problem to auditory deficit, depending on the patient's age, the work setting, the examiner's training, etc. He also noted that many of our diagnostic tests are not well normed; some with small N's, questionable assumptions, and inaccurate generalizations. There is an important reason for including this observation. If we don't take a specific look at this problem — and in order to do that use tests that are going to have a ceiling sufficiently high to allow the difficulties to show — we are likely to miss a central auditory processing difficulty. In other words, some patients do beautifully in pure tone sensitivity or standard monosyllabic word tests. It is necessary to stress the system. The revealing symptom is difficulty in perception of speech or other auditory stimuli under a variety of listening conditions which might be artificially developed for diagnostic purposes, but a variety of listening conditions and variety of stimuli must be included. We have the same problems with adults where we find some subtle dysfunction relating it to everyday life. We would have failed to find such dysfunction by any of the tools we have been using clinically. However, there are materials which could be used, such as interrupted speech, binaurally alternated speech, time compressed speech, etc.

Summary

There is historical merit to discovering normal by studying the abnormal. Central auditory dysfunction appears in various ways and with all degrees of severity. These may be manifested behaviorally by the patient who evidences an auditory disorder in the presence of normal peripheral hearing. Can we then alter our evaluative procedures or develop new ones so that we may uncover such difficulties in the patient whose disorder is less than disabling? Some patients are disabled; some may be mute, others may complain that they cannot hear themselves. Some potential patients, however, cannot complain because they are new born. What are the signs to cause us to study them clinically? Certainly a patient —adult, child, or infant — who is very ill requires diagnostic study. From childhood onward, a battery of behavioral and electrophysiologic tests can aid in

differential diagnosis. But do behaviors, pathologies, and tests permit us to adequately plan treatment? Is it possible to have such a mild deficit that one could function in a normal school setting? What are the implications, if any, of lack of cerebral organizational patterns even in the deaf? Do normal hearing children with mild attentional and memory deficits have the same organizational patterns as so called "normals"? Or as the deaf?

We still lack a data base to identify infants and young children who may be at high risk for subsequent language and/or learning delay or disability. Young, pre-linguistic, high-risk children should be followed longitudinally to determine if they do develop problem(s). If they do, we need to describe in detail the different profiles and subgroups which emerge. There is need to determine whether the same or different intervention approaches (both those in current use and those to be developed) may include a broad range of services and treatments and should be implemented early.

Furthermore, what are the appropriate interventions? Auditory comprehension problems are multifaceted, and whether a patient comprehends depends on many variables in addition to the linguistic message itself. Difficulties in processing phonologic, semantic, and syntactic aspects of messages obviously influence auditory comprehension. In addition, the psychological dimension may be as important as how the material is presented. For example, whether the messge is delivered in a familiar situational context or how the patient must indicate whether he or she understands are all contributory. In other words, cognitive utilization of contextual information contributes to success in auditory comprehension.

Is signing, preferably by a system which incorporates conventional grammatical markers and equivalent of word order, an appropriate initial approach for establishing language in developmentally aphasic children? In children with central auditory disorders? Is early acquired aphasia different in its expression and progress from developmental aphasia? From adult aphasia?

In fact, from this confusing and confused array, one finding emerges virtually universally. Hearing or deaf, young or old, patients with central auditory processing dysfunctions demonstrate an inability to produce and perceive information in rapid succession. Whatever other problems they have — deafness, epilepsy, neuromotor disability, reading difficulties — they cannot respond correctly or promptly on tasks that require sequencing of rapidly presented information. The most familiar and important example of such information is speech.

References

Dublin, W.B. (1978). The auditory pathology of anoxia. *Annals of Otology, Rhinology, and Laryngology, 86,* 27-39.

Gerber, S.E. & Mencher, G.T. (Eds.) (1978). *Early diagnosis of hearing loss.* New York: Grune & Stratton, Inc.

Keith, R.W. (1985). Central auditory testing: Some ongoing questons. This volume.

Myklebust, H.R. (1954). Auditory disorders in children. New York: Grune & Stratton, Inc.

Sekula, J. (1976). Two studies of languge disorders of central origin in children. In S.E.D. El Hommossani (Ed.), *Multi-national conference on rehabilitative research in disorders of central language processing.* Cairo: Ministry of Social Affairs.

Sloan, C. (1985). Auditory processing disorders: What are the implications regarding treatment? This volume.

Young, M.L. & Protti-Patterson, E. (1984). Management perspectives of central auditory problems in children: top-down and bottom-up considerations. In F.W. Musiek (Ed.), *Selected topics in central auditory dysfunction,* Vol. 5, No. 3 of *Seminars in Hearing.* New York: Thieme-Stratton.