Hearing Screening Programs and Identification of Central Auditory Disorders

George T. Mencher

The causal role played by prenatal or birth trauma in delayed speech, language, and/or cognitive development is generally acknowledged. Papers concerned with physical anomalies and neonatal behavior; perinatal drugs and neonatal behavior (Brazelton, 1971); preschool intelligence and newborn size (Babson et al., 1969), maternal age at birth and intellectual function of the child (Lobl et al., 1971), fetal outcome as related to gestational age at time of birth (Cushner and Mellits, 1971), and of course, fetal alcohol syndrome studies, all agree, those factors have some potentially deleterious effect on the development of the child.

While most of this research offers considerable information, there has been a notable separation of variables. In terms of pure research, this approach is probably the best. However, clinical experience suggests that most children are the products of several interacting prenatal or birth events, and thus, isolation of single factors often falls short of providing predictive information regarding developmental delays or minimal brain damage.

Obviously, this is a significant problem. As we know, during the child's first year, auditory stimulation becomes increasingly important for mental development, strongly influencing the formation of associations represented by symbols in a language system. Even the mildest form of cortical damage, at this stage in development, tends to cause variations in threshold excitability for different psychological and psychomotor functions. The result is sometimes disorganized perceptions and difficulty engaging in symbolic behavior (Strauss & Kephart, 1955).

Predictive Patterns

If, however, unusual behavioral or electrophysiological response patterns which may be predictive of speech, language, or cognitive delays can be identified and coupled with pre-and perinatal data to target this group at birth, then therapeutic programs may be instituted at the earliest possible time, professional personnel can be used more effectively, and safeguards can be employed to prevent further language deficits.

The primary purpose of neonatal hearing screening programs is the earliest possible identification of hearing loss. Both behavioral and electrophysiological procedures demonstrate regular, sequential, and predictable changes associated with maturation of the infant and have recognizable and standardized response patterns. Further, both procedures have been successfully utilized in one form or another with high risk infants in neonatal intensive care units. There are those who argue, however, that neonatal hearing screening procedures, both behavioral and electrophysiological, not only examine for hearing loss, but may also tell us if certain aspects of auditory neurosensory function are intact. This argument is supported electrophysiologically by variance in tracings and interpeak relationships, and behaviorally by the many differing response patterns seen in the nursery (Mencher, Mencher, & Rohland, 1985; Gerber, Wile, & Hamai, 1985). To paraphrase Gerber, those events or conditions which may or do cause the peripheral auditory processing system to dysfuntion can (and often do) cause the central system to dysfunction. Hence, it is important that we examine infants for those high risk conditions (endogenous and exogenous; congenital and adventitious) which produce deafness, as they may also cause learning disabilities, aphasia, central deficits, etc.

If neonatal hearing screening tests, in fact, view some aspect, no matter how small, of the newborn's auditory neurosensory function, then it is conceivable that specific response patterns may reflect specific deficits in that function. Specific deficit at birth does not mean so throughout life, of course. However, evaluation of several unusual patterns, and a longitudinal-prospective examination of the children demonstrating them, could pinpoint any long term significance of those deficits, and consider their importance as part of a procedure to screen infants for potential delay in speech, language, and/or cognition.

Three distinct unusual response patterns are most often seen in reaction to auditory stimulation, and may have implications for identification of children at risk for delay (Downs, 1971; Rosenberg et al., 1969; Field et al., 1967; Gerber, 1971; Taylor & Mencher, 1972; Kileny, Connelly, & Robertson, 1980). These include:

1) No response to either electrophysiological or behavioral screening, when normal hearing is present;
2) Paroxysmal or extremely hyperactive response to routine signals; and
3) Absence of response decrement during behavioral screening procedures.

Background

This paper considers one pattern, absence of response when normal hearing is present.

George T. Mencher, Ph.D.
Director, Nova Scotia Hearing and Speech Clinic
Professor and Acting Director, Dalhousie University
School of Human Communication Disorders
5599 Fenwick Street
Halifax, Nova Scotia, Canada, B3H 1R2

Concern with the false positive rate — that is, those failing to respond to auditory stimuli even though they have normal hearing — has consistently been reported for behavioral screening tests. For example, Stewart (1971) tested 2973 children and found 23 (.7%) who did not respond in the nursery, and who were later found to have normal hearing. Downs & Hemenway (1969) failed 500 (3%) of the 17,000 they tested, the majority of whom were judged to have normal hearing. Hardy and his colleagues (1970) reported a 2% failure rate in the presence of normal hearing. Mencher (1973) suggested "No Response" will occur in approximately 3.3% of normal children and 5.4% of those classed as high risk for hearing loss. To this date, we have considered this false positive rate a weakness or disadvantage of the behavioral programs, and now that similar patterns have emerged with ABR screening, we consider it a disadvantage there.

Perhaps we have been viewing that information incorrectly. Consider the false-positives from another perspective. For example, in the Stewart study (1971), it was found that 23 babies of the nearly 3000 tested were false-positives for hearing loss, but it was also found that 18 of the 23 did have some other abnormality.

Hardy, Hardy & Hardy (1970), on the other hand, in a follow-up of children failed in their study, indicated that lack of satisfactory response in the nursery did not necessarily relate in any way to later abnormality. They did report a "slight," but not clearly significant, relationship between failure to respond and presence of neurological abnormality. Perhaps their inconclusive results were due to the insensitivity of their testing instruments to subtle language and cognitive delays, and the length of time involved in the follow-up.

Most recently, of course, electrophysiological screening tests have been employed in addition to or instead of behavioral procedures. These, notably the Auditory Brainstem Response or ABR, appear to be much finer measures of nervous system integrity. There is some question concerning the long term implications of failure on an ABR screening, but there is little question that the procedure ultimately tells us about the status of at least one portion of the auditory neurosensory system.

There are many ABR studies which could be described here which support the false-positive notion before us. The recent report by Stein et al. (1983), which focused on neurological disorders in children who failed ABR hearing screening, is typical and should serve as a clear example. First, they isolated all obvious neurologically impaired children in the neonatal ICU and removed them from the study. Then they followed 9 neonates who failed an ABR screening. They found that, of the 9 infants studied, 2 were normal, 4 had some type of hearing loss, 1 died, and 2 were neurologically abnormal. In other words, aside from the 2 normals, 3 of the 9 ABR failures did not have a peripheral hearing loss; but, for 30% of the false positive group, something else was definitely wrong.

Another of the popular newborn hearing screening tools is the Crib-O-Gram as developed by Simmons & Russ (1974). To say that that instrument has recently been the subject of some criticism, would be something of an understatement. Nevertheless, in Japan, Kawakami et al. (1984) have applied the instrument in a new fashion and have produced some interesting results. They followed 292 children, or 16.9% of those originally tested with the device. All those followed had failed the Crib-O-Gram screening at least once and were on the high risk register for hearing loss. After 9 months, 28 (10%) neurologically impaired children were detected, compared to only 12 (4%) in a control group, the difference between groups was significant ($<.01$). When Kawakami refined his procedures, and only children who had failed the Crib-O-Gram twice but who had normal hearing were considered, the results indicated a striking 34% of the babies with neurological impairment. Kawakami et al. concluded that all children who fail the Crib-O-Gram twice should be referred for neurological evaluation immediately. Note the comparison between 34% here and 30% in the Stein et al. study.

There was an interesting side issue raised in Kawakami's paper. Of the original 292 infants who failed on the Crib-O-Gram, 8 died in infancy, two of SIDS. Infant death, often by SIDS, seems to be a frequent finding in infant hearing screening studies. Stein et al. reported that 1 of the 9 normal hearing children they failed died; and Mencher et al. (1978) reported 5 deaths among the 111 children in their follow-up of children behaviorally screened in a Jerusalem study, and of course, now Kawakami reports 8 of 292. Orlowski et al. (1979) suggested that there may be a relationship between abnormal ABR and SIDS. Gupta et al. (1981) and Kileny et al. (1982) have seriously challenged that idea, but their work has been primarily with siblings of SIDS children or with a group of near miss babies; and, while the results are clear for their subject groups, it is difficult to draw any definitive conclusion about those who have actually died. Further, perhaps the question may not be limited to abnormal ABR tracings, but rather maybe one of a more generalized auditory system dysfunction which we haven't yet learned to identify, or which is really only a transitory feature associated with some yet totally undefined CNS anomaly. The only conclusion possible, at this time, is that behavioral and electrophysiological screening failures who have "normal hearing" appear to be at risk for more than auditory deficit.

Thus far, we have considered risk for neurological deficit and possible risk for SIDS or early death. But the major issue at discussion is risk for speech, language, and cognitive deficit. Are normal hearing screening failures in this category too? If, as a prerequisite to normal symbol usage and meaningful speech, the infant must first pass through a developmental stage involving selective signal function, then it seems likely that they are at risk. The basis for the argument is really quite simple. Response to a screening test utilizing unusual or nonenvironmental auditory stimuli, shortly after birth, when such stimuli are new, is a demonstration of selective signal function, and is normal. In turn, failure to respond is not normal.

Response, it should be stressed, involves many trials on different occasions where behavioral tests are concerned, and standard click generated tracings when electrophysiological procedures are utilized. So, failure to respond refers to a fairly comprehensive assessment, and not just to a single stimulus-no response incident. With that concept as our base, we have studied several groups of hearing screening failures, and, among other things, examined them for speech, language, and/or cognitive deficit.

Current Investigations

At the 1st Elks conference, McCulloch, Stick, & Mencher (1976) reported a follow-up of 10 such failures along with a group of matched controls. The entire test battery consisted of 49 tasks which were grouped according to major test headings including: verbal, perceptual-performance, quantitative, general cognitive, memory, and motor. The children were also examined by some standardized tests including the Peabody Picture Vocabulary Test, the Denver Articulation Screening Examination, and the Vineland Social Maturity Scale. We found that on every comparison the mean performance of the control children was better than that of the experimental group. Although there was some overlap in performance between the highest level subjects in the experimental group and the lowest level in the control group, statistical analysis clearly demonstrated group differences and a need to explore this topic in more detail.

Subsequently, in a retrospective study of 106 hearing screening failures from the Jerusalem study of Feinmesser and Tell (1976), Mencher et al. (1978) examined birth, general health, and educational records for signs of abnormal educational, psychological, behavioral, or neurological patterns. A matched control group was also studied. At the outset, it was discovered that 16.2% of the subject group had diagnoses which included such items as retardation, cerebral palsy, cleft palate, SIDS, and hearing loss. Only 3% of the normal control group demonstrated any abnormality.

Next, 32 normal hearing children who had failed the newborn screening were selected and matched with 32 controls born at the same time, in the same hospitals, and who had passed the screening. Comparison included such general areas as physical coordination, reading and mathematical skills, memory, and teachers' overall ratings of performance of the children in school. In every single category, the controls outperformed the newborn screening failures. Remember, both of these groups of children were the same from any point of view, and both were apparently normal. The only known difference was that one group consisted of children who had failed a hearing screening test as newborns nearly 8 years earlier, and the other group had all passed. The hypothesis under study was quite simple. Based on results from previous studies, it was predicted that the hearing screening failure group would exhibit delays in speech, language, and/or cognition; or would, in the general sense of the term, demonstrate a central auditory disorder. After all, if central auditory processing involves the way the cerebral mechanism receives, perceives, manipulates, decodes, and utilizes signals (Eisenson, 1985), then there is a high probability that a normal hearing child who consistently fails to deal with auditory stimuli at birth is a child with a central auditory disorder.

The results of the study did not completely meet expectations. Hyperactivity and lack of inhibition were the most common things noted among the subject group. We also found that 9 of the 32 children in that group had been referred to pediatric neurology by the school system at some time in the first 3 grades. None of the control group had ever been referred. We had predicted that deficits in reading and mathematics and language delay would be the most common factors we would find. The results were almost exactly opposite. The performance of the two groups was closest in those three areas. They were furthest apart in behavior, mental adjustment, and memory.

Sloan (1985), in the treatment program she outlined, was able to improve virtually all aspects of a child's language function, except memory. She was not able to improve it even with training. In the Mencher et al. study, memory was also a major deficit area, suggesting that spontaneous improvement with maturation is not going to eliminate the problem. Further research is clearly required into this phenomenon, particularly if prolongation of auditory and speech stimuli is necessary to help the child with a central auditory processing disorder (Tallal, 1985). If a major deficit area is memory, and prolongation of stimuli is necessary for treatment, a delicate therapeutic balance must be achieved to be successful. Clearly, this is an area for extensive research.

Conclusion

What is the conclusion to draw from all this, particularly about predicting those who will require educational, clinical or medical support? Do these results have any implications for prevention?

It seems evident that the nature of the problem described in this paper, if not a specific disorder, is clear and well defined. That is, failure on a properly controlled neonatal hearing screening (behavioral or electrophysiological) definitely suggests the child should be followed, and perhaps even referred immediately to pediatric neurology. In apparent contradiction to this, however, it also seems that one of the reasons so few children are seen with prolonged signs of deficit is that the effects of their early problems are not maintained or fixed at the initial level. In other words, there is a natural "catching up," something Byrne and Miller (1985) discussed. Their results suggested some abnormality in an at-risk group early in their study and a normal response, suggesting a "catch-up," by six months of age. They concluded that early deviant patterns of attention may reflect a deficit or delay in efficient processing of auditory stimuli which, in turn, may be related to delay in early language acquisition. They suggested repeat assessments at key periods in

development to help identify truly at risk children. In other words, those who won't "catch up" enough.

Perhaps the "catch up" process really does exist; or perhaps any deficits are masked with age and by the addition of new skills and cognitive utilization of contextual cues; or perhaps our tools are not sophisticated enough to accurately measure them. Perhaps it is all three.

Elliott (1985) alluded to the masking of deficits, indicating ways in which cognitive utilization of contextual information and experience contribute to word intelligibility. Subjects with prior experience to draw upon or larger vocabularies perform much better than those without such benefits. With good intellectual function and a lesion limited to the auditory pathway, it may be difficult to identify deficits in word intelligibility. Elliott may be correct; however, other factors may also be at work here. System redundancy could mask less quantifiable deficits, and further, increased age and new skills may allow for the development of alternative channels for processing.

It is interesting to note that Shewan (1985) in writing of adult aphasic patients offers similar comments. She has suggested that the auditory comprehension problems demonstrated by aphasics are multifaceted, and whether a patient comprehends depends on many variables in addition to the linguistic message itself. Difficulties in processing phonological, semantic, and syntactic aspects of messages obviously influence auditory comprehension. In addition, however, the psychological dimension may be as important as how the material is presented. For example, whether the message is delivered in a familiar situational context or how the patient must indicate whether he or she understands are all contributory. In other words, the adult aphasic's cognitive utilization of contextual information contributes to success in auditory comprehension. In essence, with good contextual information and motivation, deficits can be masked.

Finally, Keith (1985) referred indirectly to this issue when he noted how the identifying terminology changes from central auditory disorder to auditory perceptual problem to auditory deficit, depending on the patient's age, and work setting, the examiner's training, etc. He also noted that many of our diagnostic tests are not well normalized; some with small N's, questionable assumptions, and inaccurate generalizations. It may be that normal maturation — linked with less efficient testing and an ever increasing development of new skills — simply masks earlier deficits and thus, the child "catches up".

Summary

Newborn screening programs may be touching and perhaps even specifying some of the children likely to demonstrate central impairment. The majority of children properly denoted as having "central impairment" suffer from such mild and restricted lesions that they most likely "catch up". This latter term refers to individuals who function relatively normally, but often at the lower edge of that scale, often flirting with academic difficulty. Memory deficits are the most easily identified. Perhaps the "catch up" process can be even further accelerated by a strong, supportive growth environment utilizing stimulation, education, and nurturing caregiving. Perhaps we need to consider automatically enrolling our screening failures in parent/therapy, language stimulation, and behavior management programs to ensure that there is every opportunity for normal development to occur.

References

Babson, S.G., Henderson, N.B., & Clark, W.M. Jr. (1969). Preschool intelligence of oversized newborns. *Proceedings of the Annual Convention of the American Psychological Association, 77,* 267—268.

Brazelton, T.B. (1971). Influence of perinatal drugs on the behavior of the neonate. In J. Hellmuth (Ed.), *Exceptional Infant: Studies in abnormalities:* Vol. 2. New York: Brunner/-Mazel.

Byrne, J.M. & Miller, C.L. (1985). Cardiac responsivity to speech in normal and at-risk infants: implications for clinical assessment. This volume.

Cushner, I.M. & Mellits, E.D. (1971). The relationship between fetal outcome and the gestational age and birth weight of the fetus. *The John Hopkins Medical Journal, 128,* 252-260.

Downs, M.P. (1971). Audiological evaluation of the congenital deaf infant. *Otolaryngologic Clinics of North America, 4,* 347-358.

Downs, M.P. & Hemenway, W.G. (1969). Report of the hearing screening of 17,000 neonates. *International Audiology, 8,* 72-76.

Eisenson, J. (1985). Central auditory disorders and developmental aphasia: is there a difference? This volume.

Elliott, L.L. (1985). Cognitive contributions to word intelligibility. This volume.

Feinmesser, M. & L. Tell. (1976). Neonatal screening for detection of deafness. *Archives of Otolaryngology, 102,* 297-299.

Field, H., Copack, P., Derbyshire, A.J., Driessen, G.J., & Marcus, R.E. (1967). Responses of newborns to auditory stimulation. *Journal of Auditory Research, 7,* 271-285.

Gerber, S.E. (1971). *Should we and can we test the hearing of the newborn?* Paper presented at the 19th Annual Conference of the California Speech and Hearing Association, Los Angeles.

Gerber, S.E., Wile, F., & Hamai, N.T. (1985). Central auditory dysfunction in deaf children. This volume.

Gupta, P.R., Guilleminault, C., & Dorfman, L.J. (1981). Brainstem auditory-evoked potentials in "near-miss" sudden infant death syndrome. *Journal of Pediatrics, 98,* 79.

Hardy, J.B., Hardy, W.G., & Hardy, M.P. (1970). Some problems in neonatal screening. *Transactions of the American Academy of Ophthalmology & Otolaryngology, 74,* 1229-1235.

Kawakami, T., Asano, K., Akamatsu, H., Yamamoto, K., & Okamoto, M. (1984). *The neurological prognosis of the babies who failed the audiological screening by method of Crib-O-Gram.* Paper given at the 17th International Congress of Audiology, Santa Barbara.

Keith. R.W. (1985). Central auditory testing: some ongoing questions. This volume.

Kileny, P., Connelly, C., & Robertson, C. (1980). Auditory brainstem responses in perinatal asphyxia. *International Journal of Pediatric Otorhinolaryngology, 2,* 147-159.

Kileny, P., Finer, N., Sussman, P., & Schopflocher, D. (1982). Auditory brainstem responses in sudden infant death syndrome: Comparison of siblings, "near-miss", and normal infants. *Journal of Pediatrics, 101,* 225-227.

Lobl, M., Welcher, D.W., & Mellits, E.D. (1971). Maternal age and intellectual functioning of offspring. *Johns Hopkins Medical Journal, 128,* 347-361.

McCulloch, B.J., Stick, S.L., & Mencher, G.T. (1976). The University of Nebraska neonatal hearing project — one year later. In G.T. Mencher (Ed.), *Early identification of hearing loss.* Basel: Karger.

Mencher, G.T. (1973). Unpublished report to the United States Joint Committee on Infant Hearing, Estes Park, CO.

Mencher, G.T., Mencher, L.S., & Rohland, S.L. (1985). Maturation of behavioral response. *Ear & Hearing, 6,* 10-14.

Mencher, G.T., Tell, L., Levi, C., & Baldursson, G. (1978). *Mass behavioral screening and follow-up.* Paper presented to the National Research Council, National Academy of Sciences, Committee on Hearing, Bioacoustics & Biomechanisms, Omaha.

Orlowski, J.P., Nodar, R.H., & Lonsdale, D. (1979). Abnormal brainstem auditory-evoked potentials in infants with threatened sudden infant death syndrome. *Cleveland Clinic Quarterly, 46,* 77.

Rosenberg, P., Lovrinic, J., Katinsky, S., & Pinkus, A. (1969), *Some observations on neonatal hearing screening.* Paper presented at the American Speech and Hearing Association Convention.

Shewan, C.M. (1985). Auditory comprehension problems in adult aphasic individuals. This volume.

Simmons, F.B. & Russ, F.R. (1974). Automated newborn hearing screening, the Crib-O-Gram. *Archives of Otolaryngology, 100,* 1-7.

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

Stein, L., Özdamar, O., Kraus, N., & Paton, J. (1983). Follow-up of infants screened by auditory brainstem response (ABR) in the neonatal intensive care unit. *Journal of Pediatrics, 103,* 447-453.

Stewart, I.F. (1971). Personal Communication to M.P. Downs, Forwarded to all members of the Joint Committee on Newborn Hearing Screening.

Strauss, A. and Kephart, N. (1955). *Psychopathology and education of the brain injured child, Volume II, Progress in theory and clinic.* New York: Grune and Stratton, Inc.

Tallal, P. (1985). Neuropsychological research approaches to the study of central auditory processing. This volume.

Taylor, D.J. & Mencher, G.T. (1972). Neonatal response: the effect of infant state and auditory screening of newborn infants. *Archives of Otolaryngology, 95,* 120-124.