AGING AND ADULT DEVELOPMENT CONSIDERATION FOR PHYSICIANS, SPEECH PATHOLOGISTS, AND AUDIOLOGISTS REGARDING COMMUNICATION AND COMMUNICATION DISORDERS

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In the United States of America in recent years, gerontology and geriatrics have received increased attention relative to the social and medical implications related to aging. The comments and data presented in the paper will be limited to information which is available regarding the area of geriatrics and gerontology in the United States, primarily because this type of information was not available to the author as it pertains to the country of India. It is the author's belief, however, that the information to be presented below is basic to any population. Although the specific numbers may vary significantly and perhaps in some cases not so significantly from country to country, nevertheless the basic similarities between India and the U.S.A. in this area are probably much greater than their differences. Further, much of the details to be presented can be found in greater detail in the book, *Aging: Communication Processes and Disorders,* edited by D.S. Beasley and G.A. Davis (1980) (see references).

Part I: Adult Development

Personal-Social Changes

In the United States, a recent survey indicated that only 3 out of 87 medical schools had specialities in geriatric medicine, and this in spite of the fact that individuals who are older than 65 years of age occupy 33 per cent of the hospital beds and 90 per cent of long-term care beds in the country. In addition, only 25 out of 84 schools of social work offered geriatric specialities in social work, and less than 10 per cent of the programs in speech and hearing offered courses related to geriatrics or gerontology. However, in the United States, change is imminent in that people are beginning to have a much greater interest in the study of aging and particularly as it relates to communication disorders. There are probably a number of reasons for this, not the least of which is that the mean age of the population in the United States has increased significantly in recent years. For example, in the study conducted by the United States Bureau of the Census in 1977, 10 per cent of the population was 65 years or older, but it is predicted that by the year 2040 nearly 20 per cent of the population will be 65 years of age and older. Investigators and clinicians in the speech and hearing

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sciences have become increasingly interested in the problem of communication disorders as a function of *adult development* and *aging,* and particularly as these problems or changes may relate to the social and personal characteristics of individuals and their interpersonal relationships with their families and other members of society.

Knox (1980) has provided a nice overview of the concept of adult development. He has indicated that education, equality, and pluralism all have contributed to a greater awareness of adult development, including awareness of those changes that are gradual such as increased confidence or decreased speed of reaction time, as well as awareness of changes that are abrupt, such as retirement, sudden change in living conditions, and of course, death. He has indicated a number of so-called tasks that the clinical practitioner must begin to consider when working with aging populations, including the task of making sure that those individuals who are involved in these kinds of clinical settings assist in making the awareness of adult development accurate and based upon facts and not upon speculation and stereotype. In addition, the clinician must assist adults in adapting to change and help them to understand and cope with the seeming paradox of stability and change—'both of which interact as part of the total life cycle. Of course, the clinician also is charged with the task of helping the client prepare for such changes. Knox goes on to provide a number of suggestions regarding how we might go about accomplishing these tasks, but this forum does not permit an indepth analysis of this activity. Rather, the reader is encouraged to pursue this area through the various references that have been provided by Knox (1980).

One particular area of the social sciences which may have a direct bearing on activities in medical practice will be the study of adult cognitive development (Denny, 1980), based upon investigative data to the extent that is possible. Denny has described two basic types of research designs which have been used with this population and the various problems associated with each. The *cross-sectional* type design includes individuals of all ages in a single study and has the advantage of being completed within a relatively short period of time. Unfortunately this type of study confounds *age changes* and *change events* within the study itself. That is, change events, which may have significant impact upon the individual's life at any given point in time, are assumed to be ' equivalent ' for the 45 year old and the 75 year old. Of course we know this is not true, but in the cross-sectional design this is an assumption that must be made, and therefore it becomes necessary to qualify our conclusions based upon this assumption. Another confounding variable in the cross-sectional design is that of dealing with cohort differences (that is, generation or peer group members) whereby we make the assumption that the 45 year old individual has the same number and type of friends as the 75 year old individual. An example of how this could confound the results would be through the measurement of the area of intellience, whereby we sometimes assume that intelligence decreases as a

function of age, but this decrease may simply be an artifact based on an individual's current peer group.

The other type of research design which Denny discusses is the *longitudinal* design, which tends to neutralize the confounding factor of peer group relationships. In the longitudinal design, however, it is necessary to study different groups as homogeneously as possible, thereby limiting our generalizations. Of course, longitudinal designs also confound age and time of measurement, for example, a new social program may increase the activity level for * particular older group thereby distorting the appearance of the actual function of aging. Schaie (1978) also has discussed these problems of research design in the study of adult development, and both he and Denny have indicated that in some way these research designs must account for consideration of factors such as age, cohort, and time of measurement. It *is* ideal that at least two of the three factors are controlled at any single point in time for a given study.

In the area of cognitive development, a matter that seems to be of major interest is that of *intelligence* as a function of aging. Horn and Cattell (1967) have indicated that *fluid* intelligence seems to decrease as a function of aging, and this would include such abilities as inductive reasoning and associative memory. On the other hand, *crystallized* intelligence, such as vocabulary use, seems to increase as a function of aging. There tends to be a greater decrease in performance I.Q. than in verbal I.Q., although studies of I.Q. often have failed to take into consideration such things as educational level of the subjects being studied, general health at time of testing, and the cross-sectional nature of the research designs (Denny, 1980). In addition several of the studies in this area have made use of the so-called *problem solving* method of measurement, whereby the subject had to find the ' right answer.' Older adults may simply not be interested in performing this type of task. The problems with intelligence testing are many and have been discussed in detail by Denny (1980), Botwinick and Storandt (1974) and others, and the interested reader is directed to this research.

Denny also has provided some suggestions regarding intervention research and practice. Some of her suggestions include use of the so-called *modelling style,* whereby adults ' model' strategies which are normally used by young adults. In addition, she suggests that on occasion the *direct instruction* approach may be adequate, that is, the clinician directly tells the individuals how to do something. (The research to date suggests that this is not as effective as the modelling technique). In addition, adults need a significant amount of *feedback* and, of course, *practice,* although each of these items are most effective when used with other instructional methods. Further, it seems that brief, short-term intervention procedures are the most effective for adults, and that simply helping them try to increase their speed of response is not necessarily particularly effective.

Another aspect of cognition has been discussed by Smith and Fullerton (1980) and involve matters related to memory. Memory as a function of cogni-

tion is extremely important in that it is closely related to the various aspects of language behavior in adult development. Cognition, as it relates to memory, normally requires the ability to *encode* information in memory, to *store* this information, and subsequently to *retrieve* it by decoding the information for speech and language production. Normally there are considered to be a minimum of three types of memory, namely, *sensory or pre-perceptual storage, short-term memory,* and *long-term memory.* Long-term memory is sometimes subdivided into 2 categories, namely, *episodic memory* whereby we deal with contextually related matters and attempt to remember things that occurred in context, and *semantic memory,* whereby we attempt to remember concepts or facts, but not necessarily the context in which they were learned. Traditionally, it has been assumed that the sensory or pre-perceptual storage memory tends to decay rapidly with time, particularly for older individuals, but this idea has been disputed by Smith and Fullerton (1980). In short-term memory, we process information immediately and continuously, so it is sometimes known as the 'working' memory. It generally is considered to have a smaller capacity than long-term memory and appears to be less affected by age than long-term memory. Older individuals seem to take a longer time to search short-term memory for retrieval purposes, thereby leading to the conclusion that errors by older adults on memory tasks may be more related to 'caution' in decision-making rather than mistakes of memory.

Thus Denny (1930), Smith and Fullerton (1980), and others have discussed various aspects of the cognitive activities of adult development in detail, and the interested reader is encouraged to pursue more indepth study of these areas. Suffice it to say at this time that for the physician and allied health practitioner, it is necessary to consider these various social-personal factors in both diagnostic and therapeutic activities in order to obtain the most accurate data and to provide the most effective programs for these individuals.

Neurophysiological Changes

It is imperative that we understand the basic sociological and psychological implications of aging in order to provide the most adequate clinical and medical services to these individuals. Associated with each of these factors, however, are the very real neurological and physiological changes associated with the effect of aging. Valenstein (1980) has provided two classes of brain structure changes associated with aging. First is that of the *developmental changes* which normally occur within the first three years of life, as evidenced by brain weight tripling, growth of the neuronal processes including myelinization, and synaptic development. Of course, it needs to be understood that these functions will continue growing and developing up through the 4th decade of life. But this early rapid development contributes to the maturing of perceptual, motor and cognitive abilities.

The second class of brain structure changes associated with aging are the so-called *involutional changes* which are normally associated with " old age " but which may occur anytime in life. These include an increase in lipofuscin as early as the first decade of life and the loss of nerve cells by middle age. There also tends to be a diminution of plasticity throughout life and a decrease in axonal sprouting with age. Morphologically, the brain weight tends to increase up until about the age of 18, at which time it tends to plateau. We begin to lose brain weight around the age of 40 or so, and this seems to hold true for both males and females, although the female brains do tend to be statistically lighter in weight throughout life.

In recent years there has been increased use of *computerized tomogrophy* as a tool for studying neurological changes in the higher central nervous systems. This particular technique has certain advantages over *pneumoencephalography* and *angiography* in that we can use normal subjects with less risk to the subjects of these studies. Valenstein has reported that the *ventricles* tend to increase in size slightly between the ages of 30 and 70, and then more rapidly after the age of 70. Roberts and Caird (1976) found that this increase in ventricle size tended to correlate with scores on memory tests. It also has been found that cortical atrophy tends to occur with increasing age as evidenced by a thinning of the gyri and widening of the sulci, particularly in the frontal lobe. *Cortical atrophy,* however, does not seem to correlate well with increasing ventricle size, thereby raising questions as to whether senile dementia, for example, is more related to cortical atrophy or increase in ventricle size (Willanger *et al,* 1968 and Roberts and Caird, 1976, respectively). Brody (1955), in his classical work with 20 patients ranging from newborns to 95 years of age, showed that there were significant losses of neurons by the age of 70 in the superior temporal gyrus, the pre-central gyrus, and the stria-cortex (in that order), but not in the post-central gyrus. He also showed that there tended to be a greater loss for small granular neurons than the large pyramidal neurons. According to Valenstein, there seems to be little or no loss in the brainstem region such as the ventrocochlear neucli and the inferior olive, but there does seem to be some loss in the locus cerulus (rapid eye movement) and the cerebellum, particularly the purkinje cells. There seems to be little evidence for cell loss in the temporal lobe, but there does seem to be a significant loss of brain cells in the frontal lobes and the prefrontal cortex, particularly for granular neurons. It should be cautioned that these studies normally provided no I.Q. data, and therefore it is difficult to determine whether this lack of neuronal cells, at least within age categories, may have been related at least in part to intelligence levels.

Cragge (1975) suggested that there seemed to be a negative correlation between the number of synapses and age. There is a definite increase in *lipofuscin,* which is a yellow-brown pigment of lipid and protein that accumulates in the cytoplasm of neurons. It has been hypothesized to originate in a number of sources, including the lyosomes, mitochondria, or the Golgi apparatus,

as well as within subcellular organelles. The degree of accumulation of lipofuscin varies among neuronal populations, and the extent of its detrimental effects has yet to be determined. *Granulovacolar degeneration* appears to increase with age as shown, for example, by the loss of cortical neurons in Alzheimer's disease and the basal ganglion neurons in supra-neuclear palsy. There is a significant increase in *neurofibrillary tangles,* which were first described by Alzheimer in 1906 as associated with *pre-senile dementia.* These appear as thick, fibrous bands in the cytoplasm of neurons, but more accurately are double helical filaments or ' little tubules ' which are not found in normal individuals. They may be found in the hippocampus with increasing age as well as associated with a number of central nervous system diseases such as lead poisoning and Downs Syndrome. They have been associated with increases of concentration of aluminum in the central nervous system.

Neuritic plaques appear in neuronal processes and supporting cells, but not within the neural cell bodies *per se.* They appear in granular filaments associated with degenerating neuronal processes and are found mostly in the amygdala, but also can be found in the cortex, the hippocampus, and the basal gangalia, and do not seem to be correlated to the occurrence of neurofibrillary tangles.

Alzheimer's disease, or senile dementia, has come to be associated with the aging process. It has been defined as acquired deficits in intellectual and emotional behavior, but has more broadly been characterized as any dementia with the characteristic findings of age, whether pre-senile (less than 65 years of age) or not. The clinical features suggest that more men than women suffer from Alzheimer's disease and that it tends to be associated with recent memory problems as well as verbal and non-verbal cognitive deficits. In the early stages, certain types of frontal lobe problems can be found to occur, such as loss of motivation and failure to plan, and in the later stages other types of characteristics seem to appear, such as akinesia, gait apraxia, and urinary incontenance. It is characterized by seizures and mild clonic jerks and a reduced life expectancy, and in the later stages may show severe cortical atrophy. It is normally a slowly progressive disease which is characterized by increased protein in the cerebral spinal fluid. The EEG is normal in the early stages, but tends to show a slowing in the later stages.

Alzheimer's disease pathologically may show essentially normal adult aging changes, but to a greater degree than what would be expected. Senile plaques also tend to be highly correlated to dementia and the neurofibrillary tangles appear to go beyond the hippocampus to the cerebral neocortex. There seems to be an extreme involution of neurons correlated with neurofibrillary degeneration of the cell bodies.

Some individuals have indicated that Alzheimer's disease is simply the extreme version of the normal aging process of the central nervous system. The question, however, that arises when we view it this way is simply, why do certain

younger people exhibit it before they are older, that is, why do some people 'age' at a very young age ? More specifically, individuals showing typically normal aging activities generally exhibit at least 50 per cent less neuronal dropout than those exhibiting Alzheimer's disease, although this data at present is speculative. The most dramatic difference between normal aging individuals and those exhibiting Alzheimer's disease is chemical in nature. Dementias are associated with the loss of axondendritic tree and not simply the neurons *per se,* as might occur in normal aging. Normal aging also shows a severe loss of neural transmitting chemicals. But Alzheimer's disease definitely shows diminished choline acetyltransferase, which is required for the synthesis of acetylcholinesterase. This has been associated with the loss of memory function.

Certain physiological measures of aging have come of age in recent years. For example, studies of cerebral blood flow have shown that certain progressive changes as a function of aging seem to occur, namely, a decrease in regional cerebral blood flow in the temporal and parietal regions and the frontal lobes of the brain. Arteriosclerosis has been related to hypertension which may yield focal death of brain tissue (an infarction). Vascular problems, however, are not prevalent in Alzheimer's disease.

The *electroencephalogram* has been used to study the aging process. For example, *alpha rhythms,* which are associated with the posterior regions of the head, seem to decrease with age, and this tends to be correlated with decreasing intelligence test scores. The faster *beta rhythms,* which tend to occur in the anterior regions of the head, seem to be more prevalent during middle age, but tend to decrease after the age of 70. Diffuse, slow activity, which in fact is slower than the alpha rhythms, may be seen in 20 per cent of normal individuals who are younger than 75 years of age and tend to be associated with lower intelligence in younger populations. Thirty to 50 per cent of normal older people show episodic, high-voltage slow activity in the temporal regions of the left hemisphere, and this has been suggested as a predictor of verbal deterioration.

Average event related potentials (AER) also have been used to study aging in an attempt to average out the random EEG activity and to time-lock specific stimulus responses. Two components of the AER include *(a) early component,* which is normally a low amplitude 108 msec stimulus related to the spinal cord and brainstem, and *(b)* the late component, which goes beyond the 150 msec time limit and reflects cortical sensory processing, particularly over the parietal and post-frontal regions, suggesting that higher order of processing is being studied with the late AER components. The latency of the early components tend to increase for audition and vision as a function of aging, although the data to date is controversial. The contingent negative variation wave, which is the slow negative potential shift which occurs prior to the expected signal, increases in magnitude as a direct function of attention. Older individuals, however, tend to show a progressively smaller contingent negative variation than younger individuals. Finally, *sleep studies* have been used to study the aging process,

and it has been found that older people seem to show a larger number of awakenings and increased percentage of awake time while in bed. In addition, *rapid eye movement* tends to decline as a function of age which, as mentioned earlier, may be related to changes in the neural patterns of the locus cerulus as suggested by Brody (1955).

Valenstein (1980), as indicated earlier, has provided an excellent overview of the many neurophysiological changes associated with the general aging process. It is necessary that clinicians understand these changes in order to be able to better understand and subsequently deal effectively with the consequences of disorders of speech, language and hearing as these relate to aging, and particularly older adults.

Part II: Communication Disorders

Hearing

To otolaryngologists and professionals in the area of the allied health sciences, the information relative to general aging which has just been presented has significant impact upon the diagnostic evaluations and treatment programs provided to individuals with communication disorders. At this time, let us turn our attention to problems of audition as related to adult development.

The general problem of peripheral auditory problems and aging has been discussed by Powers and Powers (1978), who indicated that advanced aging appears to be the most common cause of sensory neural hearing loss. Orchik (1980) has described this change as being a bilaterally symmetrical progressive loss which begins around the age of 30, initially observed in the high frequencies subsequently working downward into the low frequencies. It seems to occur earlier in males than females, but progresses more rapidly in females, and the right ear seems more affected than the left. It has been said to encompass 20 per cent to 25 per cent of the older population over the age of 50, and 40 per cent to 50 per cent of those over the age of 75. Ramsdell (1970) has suggested that there are 3 functional, or psychological, levels (in order of severity) of hearing loss in older populations, namely, those at the *social* or *symbolic* level, those at the *warning* level (which includes characteristics of cautiousness, hesitancy, and insecurity), and those which he calls *primitive* level, that is, the warning system itself is hampered sensorally. More specifically, according to Orchik (1980) 80 per cent of the binaural hearing losses in older people occur in those persons over the age of 45, and 55 per cent of the binaural hearing losses occur in persons over the age of 65. But, it must be kept in mind that only roughly 20 per cent to 25 per cent of the older population, in fact, have hearing losses. Therefore, the stereotype of 'old people being hird of hearing ' does not seem valid for 81 per cent of the population, at least in the United States. Given this thought, then, it becomes imperative that we relate audiological data to communication data, that is, we must ask the individuals if they feel like they have a handicap. In addition, Orchik (1980) has suggested that a hearing loss does not automatically

Worsen as one ages. More specifically, if an individual had normal hearing at the age of 60, then a perceived communication problem would be unlikely to develop, although the loss itself might increase somewhat. In addition, he indicated that people with hearing losses do not seem to be much worse off socially than those without hearing losses, contrary to the assumptions often made by society.

Presbycusis has been a term commonly associated with deterioration of audition with age, but the characteristics of presbycusis can occur at any age. Classically, presbycusis has been attributed to changes in the cochlea, although it is hard to sort out sensorineural changes from the conductive changes that are also occurring as a function of aging. Other variables which interact with the findings of presbycusis are noise exposure history, Meniere's disease, labrynthitis, arteriosclerosis, and a history of the use of ototoxic drugs, usch as the myocines and aspirin prescribed for arthritis. Thus, as pointed out by Nadol (1980), presbycusis may actually be an accumulated hearing loss over years due to several disorders or insults contributing to degeneration of the auditory mechanism. Causes, therefore, which may be associated with presbycusis are genetic difficulties (Nance and McConnel, 1973), acoustic trauma (Glorig and Davis, 1961), ototoxicity (Worthington *et al.,* 1973) and circulation (Saxen, 1937), and, of course, true cellular aging. Thus, it is important, that we correlate pertinent neuropathology that my be associated with normal aging in the central nervous system to any findings of presbycusis.

Orchik (1980) indicated that the air-bone gap in audiological testing tends to increase with an increase in degree of loss, and this has been related to cochlear partition changes and to mechanical middle ear changes. He also noted that there was a greater reduction in speech discrimination scores than would be expected from the pure tone data. Pestalozza and Shore (1955) suggested that this was due to degenerative changes in the spiral ganglion of the 8th cranial nerve, and Gaetzinger *et al.* (1961) added that it was likely due to a composite of central nervous system changes and not restricted to any single level. Perhaps the major point here is that older persons with or without hearing losses need to be treated as individuals and not simply as stereotypes, that is, the problems of hearing cannot be separated from the other problems of aging. Given this thought, then, let us turn to some considerations of the structural and physiological changes in the aging ear.

Peripheral Hearing Mechanism and Audiologic Considerations, For discussion purposes, we may assume that the outer ear is not of significant impartance to general hearing acuity, but it may be important in hearing testing and hearing aid fitting. Thus we should be alerted to the fact that the auricle seems to become unusually large with age and seems to become more stiff and rigid. The external auditory canal tends to show a diminished elasticity and may narrow or may widen as a function of aging, and a drying out of the external auditory canal may be observed. The middle ear also may show a thinning and

a less rigid eardrum, and the ossicles seem to become more rigid, due in part to joint calcification as well as disappearance. Deterioration of muscles of the middle ear for protection seems to occur, resulting in less tension and therefore a somewhat flaccid eardrum, at least as this pertains to the tensor tympani. There appears to be atrophy of the muscles and they seem to be less efficient- in performance. We also tend to observe increased eustachian tube difficulties, due perhaps to decreased patency of the tubes themselves.

Within the inner ear, there is a general atrophy of the Organ of Corti and evidence of a loss of hair cells, including a thinning of the basilar membrane and the stria vascularis (perhaps due to blood flow problems) and associated changes in the endolymphatic system. In addition, there seems to be an increased stiffness of the basilar membrane with calcification and an atrophy of the spiral ligaments.

According to Valenstein (1980) and Nadol (1980), there may be an arrest of cell mitosis and a general lack of replacement cells with age in the auditory sensory neural structures of the central nervous system. These cells seem to ' age' by accumulating errors in nucleic acids, including portions of *DNA* which are used to encode *ribosomal RNA* (Birren, 1959). This results in a progressive inefficiency of synthesis of essential proteins due to attrition of ribosomal RNA (Nadol, 1980). In addition, this may result in a progressive decrement of proficiency in speech discrimination tasks (Jerger, Shedd, and Harford, 1959; and Konkle, Beasley, and Bess, 1976). Histologically, there is some evidence suggesting that there is a decrease in the volume of the number of nerve cells in the aging brain and development of subcellular *neurofibrillary tangles* and *senile plaques.* There appears to be a loss of the *dendritic spines* and hence intercellular connections, and an accumulation of *lipofuscin.* Of course, this has been suggested by Valenstein (1980) as a general characteristic of aging in the central nervous system, and Nadol (1980) points out that these general characteristics can be observed specific to the auditory system.

There are a number of audiologic considerations involved in diagnosis of hearing problems as a function of aging. Of course, it is necessary to initially observe and study for the possibility of specific disease processes and to check for impacted cerumen, particularly in dealing with individuals who may be located in institutions or nursing homes. We also need to consider the possibility of otosclerotic involvement. Pure-tone air and bone conduction testing, which may suggest a conductive component, may reveal a *Penero effect* (Carhart, 1958), whereby there is a greater loss by bone conduction than air conduction at 500 Hz. Carhart suggested that this was likely due to a central auditory problem whereby the bone pulses were more affected than the air pulses. It could also be due to impedance changes in the bony tissue of the skull.

Presbycusis may be characterized by *phonemic regression,* which has been defined as the observance of good pure-tone acuity but poor speech discrimination. The more difficult the test material, the poorer the speech discrimination. This

effect is usually observed with sensorineural hearing loss patients, although we need to remember that poor speech discrimination may be more related to the *degree* of sensorineural hearing loss than *age* per se. Kasden (1970) also cautioned that failure to control for peripheral probelms may confound the results of the audiologic speech tests. Specific types of speech measures also may suggest higher central auditory disorders, which is discussed below.

Other measures used to study aging and presbycusis include the short increment sensitivity index (SISI), which to date has not been shown to be very effective as a measure of presbycusis, and recruitment which also has been shown to be only mildly related to presbycusis. Tympanometric data have been correlated to advancing age, including a decrease in compliance scores and a shift in acoustic reflex threshold. It should be noted, however, that when studying auditory acuity and using special tests with the aging population, there tends to be a great dial of variability associated with the obtained results.

The Inner Ear and Audiologic Considerations. Classically, Schuknecht (1964) has presented several categories of presbycusis which are based on extensive study of changes in the cochlea as a function of aging, and examples of the several types have been presented by Nadol (1980). Schuknecht (1955) originally defined two types of presbycusis: *(a) epthelial atrophy,* which was defined as a degeneration of all the structures in the cochlea and tended to be correlated to other bodily changes, and *(b) neural atrophy,* which was suggested to be atrophy of the higher auditory pathways. Later, Schuknecht (1964) suggested a more specific four-type classification for presbycusis :

1. *Sensory presbycusis,* which is a degeneration and loss of hair cells, showing bilaterally symmetrical high frequency hearing losses at frequencies less than 2000 to 2000 Hz, as well as some recruitment. This type of presbycusis tends to begin in childhood and shows a physiological loss of hair cells in the basal turn of the cochlea and possibly some loss of the supporting pillar cells within the cochlea. Accumulation of lipofuscin granules is obvious and there tends to be a minor loss of neural dendrites near the basal end.
2. *Neural presbycusis,* which has been defined as a degeneration and a loss of neural fibers, again showing bilaterally symmetrical pure-tone hearing losses associated with phonemic regression. It has been suggested that this type of presbycusis may be genetically based and may begin at any age, and the neuronal damage tends to be in the areas other than those damaged areas in the Organ of Corti.
3. *Mechanical presbycusis,* which is considered an inner ear loss, also shows bilaterally symmetrical descending audiograms with good speech discrimination. This type of presbycusis has been associated with changes in the structure of the basilar membrane as well as problems of the spiral ligament and thickening of the basilar membrane.

4. *Metabolic presbycusis,* which is characterized as an atrophy of the stria vascularis showing a flat pure-tone audiogram at all frequencies with excellent speech discrimination. There tends to be a slow progression from about age 30 onward and the stria vascularis in the basal turn is seen to atrophy. Endolymph appears to be produced intermittently, thereby changing the endolymphatic potential.
5. (A fifth type has been proposed by Johnson and Hawkins (1972) and has been termed *vascular presbycusis,* but this is similar to Schuknecht's metabolic presbycusis.)

In the evaluation of individuals with hearing loss as a function of aging, the physiological changes associated with the aging process must be kept in mind. For example, there tends to be a degeneration of the ganglion cells in the spiral ganglia, near the basal turn of the cochlea, and this tends to be a life-long process. Also, a narrowing of the internal auditory meatus is common as is nerve degeneration, thereby resulting in reduced speech discrimination scores. There also tends to be additional losses of neuronal fibers at increasingly higher levels of the central nervous system. This, of course, leads us into a consideration of auditory problems associated with the central auditory pathways.

Central Auditory Disorders and Audiologic Considerations. Audiological manifestations of central auditory disorders has been discussed in depth by Hayes (1980) who indicated that such disorders tend to result in an overall decrease in communicative ability. Several factors need to be considered in the study of central auditory processing problems, including the need for an adequate test battery, an understanding that the neurosensory effects are usually diffuse and rarely cite specific (that is, it is difficult to separate ' auditory' problems from non-auditory problems such as memory, disengagement, and change events), and of course, the problem of cross-sectional vs. longitudinal research data.

Hayes has provided a histopathological *time table* of central auditory aging which reflects neurological changes. As noted by Valenstein, there seems to be a decrease in overall brain weight by roughly 11 per cent between the ages of 25 and 65 years of age, which is associated with narrowing of the gyri and widening of the sulci, as well as thickening of the meninges of the brain. Atrophy of the corpus callosum and increased production of cerebral spinal fluid is not uncommon, and there appears also to be a decline in cell density, particularly in the area of the temporal gyrus. Accumulation of lipofuscin in the brainstem auditory nuclei has been observed in middle age. Data to date suggest that those mechanisms for hearing which are most recent on the evolutionary scale tend to be affected first and most as a function of age. Thus it seems safe to say that the affects of central auditory aging can be observed as early as 35 years of age and become progressively worse with increasing age.

The effects of the central auditory aging manifestations include a loss of understanding of speech for both words (Gaeth, 1948) and sentences of various

types (Konkle, Beasley, and Bess, 1977; Beasley and Clasquin, 1980). Amplification may not provide much help in such instances, and in fact, may make the perception of speech more difficult under certain circumstances. Hayes found that as a central affect became more prominent, amplification became less helpful, suggesting that the factor of central nervous system changes had a greater affect on central auditory processing than simply the degree of hearing loss as traditionally measured. This is not due simply to loss of the ability to process high frequency information, but also the result of problems with the temporal processing abilities of individuals. Further, elderly individuals with poor low frequency pure-tone audiograms tend to show more central auditory problems than those whose loss is primarily in the high frequencies (Hayes, 1980). Central auditory problems are likely to occur in the elderly due to cardiovascular accidents and strokes, which are common, as well as arteriosclerosis and other disease processes. Thus there is a definite need for special tests in the area of audiology to evaluate such disorders as well as improvement upon the specific methods of investigation.

Hayes has discussed the four basic methods of investigation that have been used to date, including studies of *histopathologic changes* of a post-mortem nature compared to pre-mortem audiograms. *Cross-sectional analyses* have been carried out comparing various age group differences upon auditory test performance related to age. On the other hand, *longitudinal* studies have been performed whereby the same subjects have been studied over time, most notably in recent years by Bergman (1971) who showed an accelerated rate of change in central auditory processing through the 7th decade. The problems associated with the cross-sectional and longitudinal studies, of course, are those discussed earlier. Finally, Hayes has suggested using a *peripheral/central ratio* classification system in order to minimize the variability due to differences in the rate of aging by individuals when performing audiologic evaluations.

Bergman (1971) showed that normal speech discrimination deteriorated significantly at around the age of 70. Measures using distorted speech stimuli, however, have shown deterioration of scores as early as the age of 40. Time altered speech, for example, has been used to study central auditory disorders by a number of investigators, including Calearo and Lazaroni (1957), who found that older subjects simply had more trouble in processing speech which had been shortened in duration. Bergman (1971) suggested that as age increased, ' time sampling ' by the central auditory processing system essentially decreased in efficiency. Konkle *et al.* (1977), found that intelligibility systematically decreased as a function of increase in amount of time compression, age and decrease in sensation level of presentation, regardless of control for peripheral sensitivity. Konkle *et al.,* also found that a 20 per cent decrease in scores seemed to consistently occur between the ages of 20 to 57 years of age, but between 57 years of age and 70 years of age, a second significant 20 per cent change seemed to occur. They concluded, as had earlier researchers, that the rate of progression

of central auditory disorders does indeed tend to increase with age, and further, that ' Changes in speech intelligibility associated with aging appear to be closely aligned to the temporal resolving power of the central auditory processing system.'

Jerger and Hayes (1977) compared the phonetically balanced word list scores to sentences in the presence of speech competition noise and found a decrease in scores with increasing age for both stimuli, although this effect was greater for sentences than for words. In addition, the sentences tended to show an effect at an earlier age. It also appeared that, regardless of the degree of peripheral hearing loss, subjects continued to show a disproportionate loss in speech intelligibility for sentences when compared to words, and this loss did not seem to be alleviated to any significant extent through the use of simple amplification. Jerger and Hayes (1977) also have found that a low frequency hearing loss in older individuals may be indicative of central auditory pathology. Hayes (1980) has provided a detailed discussion of the research to date in the area of central auditory processing and aging.

Auditory Rehabilitation: After the diagnostic process has been completed, it is not uncommon for a program of auditory rehabilitation to be recommended, and here the effects of central auditory disorders seem to play a prominent role. Hayes and Jerger (1979) studied hearing aid listening performance for sentences-in-noise for subjects over the age of 60, and found that individuals who exhibited central auditory disorders tended to perform significantly poorer than individuals who simply had diagnosed conductive losses under the various aided conditions. They concluded, quite validly, that we can only expect very little in results with the use of hearing aids and amplification with elderly individuals, particularly those who have significant central auditory disorder problems. Indeed the rehabilitation problems of the aged are unique in and of themselves.

Freeman and Sinclair (1980) have gone on to discuss problems associated with hearing aids and amplification for older individuals. They point out that there are a number of considerations for candidacy for amplification, including medical diagnosis, motivation, attitude, and peripheral vs. central auditory pathology, to name a few. They suggest that amplification for individuals who exhibit central neural hearing losses may be limited due to the narrow dynamic range of hearing for these individuals, which may result in an intolerance for amplified sound by such listeners. In addition, they agree with Hayes that speech discrimination with older individuals who exhibit central auditory pathology may not improve much with amplification, and they emphasize the fact that amplification will not help improve any difficulties associated with auditory memory or other cognitive aspects of language processing. Other variables that need to be considered, according to Rupp *et al.* (1977), who developed the *Feasibility Sc,ale for Predicting Hearing Aid Use,* include self-assessment, manual dexterity, and financial resources, and family support. In general, it seems safe to say that older a person, the sooner they should be evaluated for amplification.

Freeman and Sinclair go on to suggest that we need to consider whether an ear level aid is most appropriate for a given individual given the fact that the controls of such aids may be very difficult to use. It may be better to use eye-glasses for these individuals, keeping in mind, however, that such eye-glass aids may in fact be too heavy for certain older individuals, and there is considerable expense when they are lost and must be replaced. In recent years, the in-therear hearing aid has come into vogue and may be useful to certain individuals, although these are very small aids and very difficult to handle. Other considerations include whether or not to use binaural aids, which may prove to be very expensive, and whether to fit the better or poorer ear with the aid, which in fact is a controversial issue. Generally it is suggested that we emphasize the high frequencies while maintaining comfortable gain for conversational speech. Studebaker and his colleagues (1980) at Memphis State University are in the process of carrying out a long-term study to arrive at some rather specific recommendations regarding the placement of amplification on older individuals, and are using listener quality judgments of aided speech intelligibility in conjunction with various forms of distortion.

Approximately 40 per cent of older individuals who are fitted with amplification are not particularly satisfied with it, and only 30 per cent of individuals in nursing homes who are fitted with amplification, in fact, use their aids. In regard to this, Hull and Traynor (1977) have provided some recommendations for improving the evaluation procedures for determining the need for amplification for older individuals. Regardless of what procedures are used, follow-up counselling and training is extremely important, yet this has a very poor record in the United States. There is a need for more clinics to be committed to this activity as well as committed to providing in-service training for the staff and the family. The most important predictors of success for hearing amplification seem to be the health, mental status, and current language abilities of the individual client, and we have failed to develop adequate ways of measuring each of these. We must avoid trying to patronize older individuals, but rather understand that they have life experiences which have made them quite knowledgeable. We need to understand that hearing problems can cause severe social emotional problems for the individual, and may, in fact, cause the family to isolate an older individual, when, in fact, this would not be necessary if adequate rehabilitation procedures had been initiated earlier. Smith and Fay (1977) have provided a number of suggestions which could be used in nearly any setting, based upon actual data. For example, advanced age does not appear to be a major factor in the successfulness of an aural rehabilitation program, but rather the success is more related to the time patience of the individual clinicians. They found a need to educate the family and others who interact with the patient relative to the hearing loss so that they would have a better understanding of the rehabilitation process. They also suggested determining if the patient is an individual who would be apt to lose or misplaced the hearing aid, and they put great emphasis

on the need to select hearing aids based upon the manipulability of the aid by the patient. They further suggested several indicators of success with the aid, including the consistency of its use and the amount of independence in the use of the aid, as well as increased participation in environmental activities. Interestingly, they found that as the patient improved his or her attitude in the aural rehabilitation process, individuals, including the family and staff involved with the patient, also tended to improve their attitudes toward the client.

In summary, it would be naive to assume that all the many facets of hearing loss and adult development could be covered in adequate depth in this single forum. Nevertheless, while there is much information available, a great deal is yet to be discovered in this area. It is hoped that this overview will provide encouragement to the interested reader to pursue more in-depth study. In the meantime, we as clinicians must be cognizant of the significant roles played by each professional who. deals with the auditory problems of the older members of our society, and understand that the medical and allied health diagnosticians must communicate effectively with those who provide rehabilitation programs, if indeed such programs are to be most effective with the individual for whom they are designed: namely, the older adult client.

Speech and Language Pathology

Of course it is necessary, when we are talking about communication disorders, to learn all we can about the aging process as it is related to normal speech and language behaviour. To this end, we must understand the basic anatomical and physiological system upon which speech and language depends, and the changes associated therewith. Kahane (1980) has provided several insights into this area.

Anatomical and Physiological Manifestations: Turning first to the *supralaryngeal* system, the cranial facial skeleton tends to become endentulous and shows slight changes in the mandibular structure, including enlargement of certain aspects of the structure. The *temporomandibular* joint degenerates exclusively and changes position due to a reduction in size of the condyle and added growth in the joint surfaces. There tends to be a reduced *biting force* as a function of aging due to changes in the muscular structure resulting from atrophy and decreased biochemical efficiency. Changes in the mucousa are evidenced as well as possible changes in pressure, touch, and vibratory sensitivity. There tends to be a thinning of the *epithelium of the tongue* and degeneration of the sensory *pappillae.* There also is evidence for degeneration of the *hypoglossal nerve.* The larynx and velum also show changes as a function of aging, including a weakening of muscles, resulting in a *velopharyngeal incompetency* as evidenced in the form of *nasality.* Increased difficulty in swallowing is not uncommon, due to muscular weakness in older persons.

Laryngeal changes also are evidenced through *calcification* and *ossification* of the various cartilages. Which of these factors actually contribute to these

laryngeal changes is currently under investigation by Kahane and his colleagues at Memphis State University, using photomicrographs. Generally the laryngeal muscles seem to atrophy, but which ones and to what degree is still to be empirically determined. There appears to be a decrease in muscle fibre and increased amounts of connective tissue between the fibers, and the vocal folds tend to become 'fragile' and show an accumulation of lipofuscine. The cartilage cells themselves seem to atrophy and undergo some form of degeneration. Ossification seems to be more significant in males than in females and shows a later onset in females.

The respiratory system also shows certain changes as a function of aging, including the so-called 'sunken chest,' which results from a narrowing of the anterior/posterior dimensions due to *senile kyphosis,* which is predominately a curvature of the spine due to a thinning of the vertebral discs. There tends to be increased stiffness of the thorax as well as ossification of the costal cartilages. The lungs and larynx appear to descend, resulting in *laryngeal ptosis.* The pleura of the lungs appears to thin and dry up, causing diminished lung recoil, and this decreased elasticity results in decreased vital capacity and increased residual volume, which in turn causes an increase in respiratory rate.

The results of all these anatomical and physiological changes include problems of inadequacy in the driving force for speech as well as muscular weakening, resulting in *pulsatile variations* and associated prosodic changes in speech. Tnere tends to be a progressive decline on all measures of speech respiration and increased susceptibility to respiratory diseases. *Laryngeal air loss* increases and there tends to be a decrease in the *intra-oral air pressure,* which is necessary for adequate vowel duration during speech. The vocal folds tend to atrophy and the cartilages tend to stiffen with age, thereby causing difficulties with accuracy of articulation.

Of course, associated with these anatomical and physiological changes are resultant acoustic changes in speech production. The *fundamental frequencies* for both men and women show a general increase between the ages of 50 to 85, and a greater pitch variability with increase in age. With age, there tends to be a decreased *speaking rate* and range of fundamental frequency, and an increase in *intensity* as a function of *pitch* (Ryan, 1972). The decreased rate of speech appears related to an increased pause time/speaking time ratio. Frequent dysfluencies of a non-stuttering nature seem to appear and reaction time tends to increase for unfamiliar material and decrease for familiar material. Ryan and Burk (1974) have indicated that certain *perceptual cues* may be associated with the aging voice, including voice tremor, laryngeal tension, air loss, consonantal imprecision, and slowing of articulation. Darley *et ah,* (1969) have suggested that the aging speaker should not be categorized as such, but rather should be considered as a continuum of variable changes.

Speech and Language Considerations, Vocabulary word scores tend to remain stable until the 8th or 9th decades, although verbal association tasks tend

to slow somewhat with age. Word meaning of visually presented stimuli remains stable through adulthood, although increased durations of response times have been observed as a function of age. Symbolic functioning has shown certain kinds of changes (Gordon *et al.,* 1977) including increased hesitant interjections and fillers. However, there does not seem to be a significant increase in pause location or utterance length and the processing of words tends to stay within the normal range. Obler and Albert (1980) found that general comprehension tended to decrease and that older individuals tended to show language behaviour that was more syntactically elaborate but less fluent. There appears to be a decrease in the active use of lexicon, but the passive use tends to the preserved. These changes reflect normal neurological changes cortically and subcortically, as discussed earlier. Of course we must keep in mind that, with each of these characteristics, there tends to be a wide degree of variation.

The problems of speech and language associated with senile dementia have been discussed by Obler and Albert (1980). Accordingly, there are essentially 2 types of senile dementia: (1) *cortical,* which in fact is Alzheimer's disease and may be reflected in Picks disease, and (2) *subcortical,* which is often reflected in the various palsies and Parkinson's disease. The causes of senile dementia are varied and have been related, as indicated above, to Alzheimer's disease if diagnosed before the age of 65, or senile dementia *per se* if diagnosed after the age of 65. The manifestations of senile dementia, according to Obler and Albert, include functional or personality changes, memory problems, inability to manipulate acquired language, apparent slow rate of information processing, perseveration, naming problems, and a general tendency not to talk. There seems to be a loss of memory and forgetting, particularly for recent events, and a loss of orientation as well as a decline in cognitive abilities. Restlessness, paranoia, fantasies, and suicidal tendencies also seem to show an increase at this time. Alzheimer's disease tends to occur more in females than males.

Picks disease, on the other hand, presents fewer memory problems except early in its development; rather its symptoms tend to be similar to those of aphasia, apraxia, and agnosia. *Creutzenfeldt-Jakobs* disease also have been discussed as a function of aging and reflects certain types of bizarre behviour associated with the degeneration of cortical tissue. Arteriosclerosis, of course, occurs with aging and this is often times associated with strokes. Obler and Albert indicate that changes in language with age are not just a simple matter of deterioration, but rather neural behavioural reorganization of the functions of the various language areas *per se.* Thus the more education individuals have in their younger years, the less affected they might be as a function of the normal aging process, thereby emphasizing the need for different strategies for various language rehabilitation programs.

Other disorders which are commonly observed include the problems of *agnosia,* which is an inability to recognize the nature of incoming stimuli, including the body parts, spatial relationships, and so on. Often times we associate disorders

of perception to a type of agnosia. *Auditory agnosia,* for example, is difficult to differentiate from aphasia. Other types include the *verbal agnosias* and the *amusias* (failure to recognize music). Currently, there is no general agreement that agnosia is a reality different from apraxia in speech. The fact that agnosia exists as a function of age is known, but not well-documented.

Apraxia, which is a sort of motor speech programming activity, is evidenced by articulatory and prosodic problems. It should be emphasized that apraxia is not dysarthria, which is a motor speech problem due to neuro-muscular injury, nor is it aphasia, which is basically a neurological insult resulting in language problems. Martin (1974), in fact, dislikes the term 'apraxia' because it implies that speech and language are dichotomous, which, of course, they are not. At any rate, the cause of apraxia has been suggested as being damage to the base of the third frontal convulsoin of Broca's area in the dominant hemisphere. There seems to be little or no relation to aging other than its association with aphasia, and this is purely speculative.

Another area of increasing interest includes that of *dysarthria,* which is a problem of muscular coordination of the speech mechanism due to central nervous system damage, resulting in paralysis, weakness and/or discoordination of the speech musculature. It is suspected that the 600,000 or so aphasics in the United States also suffer some form of dysarthria, and that there are at least 150,000 non-aphasic individuals with speech problems who also have some form of dysarthria. According to Darley *et al.* (1975), there are 6 types of dysarthria including: (1) *flaccid,* which is due to damage of the lower motor neurons in the spinal cord and motor nuclei of the cranial nerves, resulting in bulbar palsy, muscular dystrophy, myasthenia gravis, and polio; (2) *ataxic dysarthria,* which is damage to the cerebellum resulting in problems associated with range, timing, and direction of movement, and is normally caused by such things as CVAs, tumors, multiple sclerosis, toxicity (particularly alcohol) and encephalitis; (3) *spastic dysarthria,* which is considered to be a problem with upper motor neurons resulting in cerebral palsy and pseudo bulbar palsy, resulting in respiratory and laryngeal valving problems; (4) *hypokinetic dysarthria,* which is commonly expressed in Parkinsonism, and presents a slowness of movement with limited range; (5) *hyperkinetic dysarthria,* which shows an abnormal involuntary movement and may be either 'quick' and 'slow'; and (6) of course, *mixed* types of dysarthria.

Finally, the area which has had the most atteation over the years is that of aphasia, which is neurological damage to the language modalities of the brain with the most serious effects normally associated with damage in the left hemisphere. Aphasia has several causes, including CVAs, cardiovascular difficulties, and of course, accidents. There have been many books and other materials written in the area of aphasia and the interested reader is encouraged to pursue these writings. A final area of concern, particularly as it is associated with aging individuals, is that of laryngeal cancer. Approximately 10,000 persons per year

in the United States contract laryngeal cancer and 80 per cent of these tend to be men with a mean age of 62 years of age.

There are a number of different matters to consider in speech and language assessments with aging clients. Measurements of respiration and related physiological factors should be obtained, and information pertaining to problems of confusion, anxiety, and memory loss should be studied. The diagnostician should consider fatigue, the need for positive reinforcement when test items are failed, bizarre responses, and other such test-related behaviors during the diagnosis. Often times there is a lack of co-operation which may require repeat visits, or it may be necessary to encourage trusted friends or family members to participate in testing. Other factors to consider include different types of language difficulties which may require alternate types of tests to use, any severe confusions or intellectual deteriorations which may require certain types of informal testing to be employed, and of course, hearing loss. Remember, the older person will very likely not respond as quickly as the younger person, but this is not to say that their responses will not be accurate. In addition, they tend to be anxious and therefore need more time to become familiar with the examiner and the testing situation. Do not hesitate to repeat instructions or give examples or demonstrations where possible, and alllow adequate response time.

Finally, remember that the adult world centers basically around the family, which has been discussed in detail by Davis and Holland (1980) and Webster and Newhoff (1980), as well as their occupation or past occupations, their community activities, such as their religion, their politics, and their friends, and various societal demands, expectations, and resources. A loss of a sense of communication can present personal and social problems. Societal attitudes can affect performance and self-concept and the stereotypes, of course, can be a problem, know the costs and benefits of various alternative living arrangements for adults, and understand the concept of *disengagement* (that is, older adults tend to become withdrawn and intraverted, which may be an environmental rather than natural phenomenon). Remember that they may feel a tendency to be depressed more often as their friends pass away and that such factors as their health status, their financial security, family attitudes and relationships and relocation (for example, institutionalization) may have an affect on their outlook. Understand the extent to which they have achieved the ' dreams ' that they had as younger people. Help alleviate the fear of the unknown, particularly with sudden communication disorders such as strokes and shortly after change events. It seems that adults experience great stability in the short-run, but experience great changes in the long-run, and the effective clinician must be able to understand and deal with this seeming paradox.

Summary

To be sure, there is a multitude of changes associated with communication as we age. Many of these are neurological and physiological in nature, and are

to be expected. The clinicians' task, whether medical or allied health, is to prepare themselves to deal effectively with these changes when they result in communication disorders. Indeed, to understand the non-normal, we must understand the normal, and to deal effectively with the many personal and social ramifications of communication disorders of aging persons, we must understand the normal changes of adult development. And we must put aside the stereotypes if we are to be effective clinicians. To this end, this paper has been directed, and it is fervently hoped that clinicians from various professional backgrounds will have been stimulated to pursue a more in-depth study and consequent understanding of aging and communication disorders.

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