Austin Journal of Autism & Related Disabilities

Open Access

Austin Publishing Group

Review Article

Auditory Manifestations and Intervention in Children with Autism Spectrum Disorders

Danesh AA[1], Kaf WA[2]*, Abdelhakiem MK[3], Danesh D[4] and Scott J[5]

[1]Departments of Communication Sciences & Disorders and Clinical Biomedical Science, Florida Atlantic University, USA

[2]Communication Sciences and Disorders Department, Missouri State University, USA

[3]Medical College of Wisconsin, USA

[4]Department of Biological Sciences, Florida State University, USA

[5]Departments of Exceptional Student Education and Center for Autism and Related Disabilities (CARD), Florida Atlantic University, USA

***Corresponding author:** Kaf WA, Communication Sciences and Disorders Department, Missouri State University, 901 S National Avenue, Springfield, MO 65897, USA

Received: September 28, 2014; **Accepted:** April 10, 2015; **Published:** April 13, 2015

Abstract

Children with Autism Spectrum Disorder (ASD) often have associated auditory manifestations, such as hyperacusis, difficulty hearing in background noise, and Auditory Processing Disorders (APD). Physiological and neuroanatomical changes in the auditory pathway and the non-classical auditory pathway, including the limbic system and the autonomic nervous system, are known to underlie these auditory deficits. This paper reviews the most common auditory manifestations in children with ASD and discusses the neuroanatomical evidence and the electrophysiological findings that have been used to identify changes in neural processing associated with auditory disorders in ASD. In addition, this paper provides an overview of auditory assessment and its challenges, and discusses intervention strategies for auditory problems in children with ASD.

Keywords: Autism spectrum disorder; Hyperacusis; Limbic system; Neuroanatomical and electrophysiologic findings; Sound desensitization training

Abbreviations

ASD: Autism Spectrum Disorder; APD: Auditory Processing Disorders; ABR: Auditory Brainstem Response; OAEs: Otoacoustic Emissions; MOCB: Medial Olivocochlear Bundle; AIT: Auditory Integration Therapy

Introduction

Hearing is one of the most delicate senses in humans. It is the first sense that we gain after we wake up and the last sense that we lose before falling asleep. From an evolutionary point of view this sense has become an important tool for survival. Abnormalities of the auditory system can result in several auditory manifestations such as hearing loss, tinnitus, hypersensitivity to moderately loud sounds (i.e., hyperacusis), and dislike of certain sounds (i.e., misophonoia). Additional hearing disorders include inability to localize the source of a sound and difficulty understanding speech, particularly in noisy environments.

Just over 70 years ago, Kanner [1] studied 11 children, 2-8 years old, who showed poor or absent affective contact, reduced or nonexistent language abilities and unusual fascination with objects. Kanner identifies to these "inborn disturbances of affective contact" and offers the first clinical description of autism. In addition to this considerable heterogeneity of behavioral deficits, individuals with autism display characteristic behaviors such as "persistent impairment in reciprocal social communication" (DMS-5) often with unusual behaviors, restricted interests and hyper-or-hypo reactivity to a wide range of sensory stimuli. Autism Spectrum Disorder (ASD) now affects 1 in 68 children, is more common in males than females (4:1 ratio) [2], and includes conditions such as Pervasive Developmental Disorders and Asperger Syndrome, currently known as ASD-Level 1 (mild) according to the new Diagnostic Manual (DSM-5) [3]. From a neuroscientific point of view, autism is described as a neurodevelopmental disorder with atypical neural connectivity.

Individuals with ASD share many of the auditory abnormalities that non-ASD individuals have reported. This paper reviews auditory features in the ASD population and provides audiologic recommendations in the alleviation and management of auditory problems for this population.

Neuroanatomical Diversity in Individuals with Autism

There is rich evidence from the scientific literature and imaging studies suggesting differences in brain structures in those with ASD compared to typical brains. These differences are in a variety of areas in the brain, and auditory-related centers are no exception. Here we discuss a variety of conditions associated with ASD. In general it is perceived that children with autism have a larger cranial circumference than their age matched counterparts. Presence of macrocephaly in children with ASD was first reported by Kanner [1] then confirmed by several researchers [4-6]. To be more specific about the central auditory nuclei, differences have been noticed in a major nuclear complex at the rostral medulla referred to as the superior olivary complex. This is the first nucleus in the central auditory pathway where auditory fibers from both ears arrive. The superior olivary complex is known to be involved in the process of sound localization and hearing in background noise [7]. Studies have shown malformation of the superior olivary complex in ASD populations, revealing a significantly smaller and rounder superior olivary complex in children with ASD than in controls [8,9] individuals with ASD was studied [10] in a group of adult participants and their

Citation: Danesh AA, Kaf WA, Abdelhakiem MK, Danesh D and Scott J. Auditory Manifestations and Intervention in Children with Autism Spectrum Disorders. Austin J Autism & Relat Disabil. 2015;1(1): 1005.

performance was compared with a control group. This study revealed no differences in horizontal localization; however, vertical localization was significantly impaired in the ASD group. These findings confirm neuroanatomical changes in the auditory brainstem of the ASD population. Considering that ASD is under diagnosed before two years of age, assessment of the auditory brainstem and the superior olivary complex may be a promising method in order to diagnose or rule out hearing loss and identify ASD early in life. Early identification of both hearing loss and ASD are of critical importance for effective early intervention. Researchers have investigated the timing relationship between embryological development and neuroanatomical changes to identify the onset of ASD. Gillberg [11] reported that the presence of brainstem abnormalities suggests an early prenatal origin of ASD, whereas evidence of temporofrontal abnormalities may suggest later prenatal onset of ASD. Others suggest delayed postnatal onset of ASD due to brain injuries, ischemia, hypoxia, infections and exposure to heavy metals [12].

A variety of neuroimaging studies have revealed differences in the brain structures and neurochemistry of individuals with ASD. For example, evaluation of voxelwise comparisons have shown increased gray matter volume in anterior temporal and dorsolateral prefrontal regions in the ASD population [13]. The advancements in neuroimaging technology and employment of methods beyond MRI and fMRI, such as diffusion tensor imaging and magnetic resonance spectroscopy, have contributed significantly in the analysis and study of neural diversity in the ASD population. Neuroimaging studies have shown differences in a variety of brain structures such as the arcuate fasciculus [14], corpus callosum [15], planum temporale (i.e., the smooth section of the posterior portion of the superior temporal gyrus) [16], and other auditory and language-related brain structures that may be the cause of difficulty processing emotion in others' voices [17]. A neuroimaging study on the size of the planum temporale and Heschl's gyri in children with ASD and typical subjects showed larger left planum temporale compared to the right in the control subjects but not in those with ASD and no significant difference in the size of the Heschl's gyri between the two groups was observed [16]. These differences may contribute to the perceived auditory manifestations such as increased sensitivity to sound in individuals with ASD. The brains structural diversity in syndromic forms of ASD has also been investigated. Increased brain matter volume has been reported in XYY or Jacob's syndrome and this may contribute to the increased frequency of ASD in this population [18]. Additionally, an interesting finding has been reported in reference to the arcuate fasciculus in some of the individuals with ASD. The arcuate fasciculus is the neural fiber tract which connects the anterior language area in the inferior frontal gyrus (i.e., Broca's area) to the posterior language cortex in the superior temporal gyrus (i.e., Wernicke's area). In ASD individuals with tuberous sclerosis complex, a genetic disorder resulting in the growth of benign tumors in the brain, the function of the arcuate fasciculus was diminished compared to those with this genetic disorder without ASD, resulting in poor language skills [19].

Recently, the limbic system, which is a part of the non-classical auditory pathway, was shown to be affected in children with ASD [20]. The limbic system is the area of the brain that is responsible for translating and processing how sensory stimuli emotionally affect us. Normally, the limbic system is hyperactive and highly involved in young children, and then it becomes suppressed and less involved in older children due to maturation [21]. Loud sounds, for example, can frighten a baby while the same level of sound may only annoy an older child. This manifestation of suppressing the hyperactivity of the limbic system in the first years of life is an indication of the normal maturation of the limbic system with age. Also, somatosensory evidence of the maturation of the limbic system has been reported. Moller and Rollins [21] have studied the loudness perception to trains of clicks (20ms duration and 65dB sensation level) with and without electrical stimulation of the median nerve. Their study showed consistent age-related differences in loudness perception in which 70% of the younger children (7-8 years) experienced the largest increase (2.56-1.77 dB) in loudness perception, followed by 50% of older children (13-19 years) and 10 % of the adults (25-43 years) who experienced the least increase in loudness perception (.38-.18 dB). Moller and Collins [21] conclude that the limbic system and the subcortical route to the amygdala are more active in younger children than older children and adults which is an indication of maturation of the limbic system with age.

In contrast, perseverance of the hyperactive reaction of the limbic system to sensory stimuli due to delayed maturation would cause older children to be frightened of sounds yielding reactions similar to those of a baby. Thus, hypersensitivity to sounds and the negative emotional reactions to certain sounds in children with ASD (see the auditory manifestations below) may be explained by the delayed maturation of the limbic system in this population. Several studies have shown alterations in the limbic system in children with ASD. These studies showed increased cell packing density and reduced cell size and dendritic arborization in the hippocampus, subiculum and amygdala [22,23]. According to LeRoy Conel [24] and Jacobson [25], the presence of closely packed, small neurons, with limited dendritic arbors resembles what is typically seen during earlier stages of brain maturation and may therefore reflect features of an immature limbic system. Additional evidence from autopsy research has shown that children with ASD have delayed maturity in the hippocampus of the limbic system [26]. All of these studies suggest alterations in many auditory-related brain structures which can contribute to the atypical auditory manifestations in this population. However, hypersensitivity to sensory stimuli in children with ASD is very complex because additional non-auditory structures can contribute to sensory sensitivity. Woodard et al. [27] found an association between behavioral rating of sensory sensitivity and heart rate in children with autism compared to a control group, suggesting an involvement of the autonomic nervous system. Further research is needed to validate the use of sensory profiles to investigate the association between autonomic and behavioral measures of sensory stimuli in larger numbers of children with autism, including infants and young children.

Auditory Manifestations

As clinicians, when we hear the word Autism we likely imagine a child covering his or her ears in the presence of sound. Actually, this reflexive action of putting the hands over the ears when annoyed by sounds due to hypersensitivity is a result of an emotional response to sounds via stimulation of the limbic system as well as the autonomic nervous system [13,17,20]; this will be discussed in more details

later in this section of the article. In addition to hyperacusis, there are several auditory symptoms observed in the ASD population. These symptoms include hearing loss, tinnitus, difficulty hearing in background noise, inattentiveness to verbal stimuli, selective auditory attention and atypical performance of their central auditory system as well as postural instability [28-35].

There are two major types of hearing loss. A conductive hearing loss is due to the abnormalities of the external or middle ear, in isolation or as a combined pathology. The second major type of hearing loss is referred to as sensorineural hearing loss which is often a result of damage to the inner ear or the hearing nerve. There are conflicting findings regarding sensorineural hearing loss in children with ASD. Beers, McBoyle, Kakande, Dar Santos & Kozak [28] revealed that there is no conclusive evidence of higher prevalence of hearing loss in ASD compared to the general population. However, a recent epidemiological study from Gallaudet Research Institute revealed that one in 59 children with hearing loss also have met the diagnostic criteria for autism [29]. This indicates much higher prevalence than one in 88 normal hearing children reported to be in the spectrum. A study of 199 participants with autism reported that 7.9% had mild to moderate hearing impairment and 3.5% had profound hearing loss [30]. Reports from the literature show that hearing loss and ASD share similar risk factors such as prematurity, low birth weight, hypoxia, viral infections, positive family history and Rh incompatibility [31,12]. This may explain the co-occurrence of hearing loss and ASD. Unfortunately, co-occurrence of hearing loss and ASD often cause a delay in the diagnosis of ASD or hearing loss to about four or more years [32]. Several other studies have also demonstrated some commonalities between individuals with hearing loss and those with ASD, including tinnitus, difficulty hearing in background noise, inattentiveness to verbal stimuli, and selective auditory attention [33-35]. Conductive hearing loss is also a common finding in children with autism mostly due to recurrent otitis media with effusion [36,37]. Routine tympanometric and Otoacoustic Emission (OAE), evaluations should be considered. A recent study [38] revealed that absent OAE responses at lower frequencies and abnormal tympanometric patterns (i.e., flat tympanograms with no defined peak and tympanograms with negative tympanometric peak pressure) were significantly more common in 100 young individuals with autism compared to an age-matched control group.

Hyperacusis is a common manifestation in children with ASD. In general, it is referred to as oversensitivity to moderately loud sounds which are not perceived as annoying by others. Previous studies have shown a prevalence of 18% of hyperacusis in individuals with autism [30]. In our lab we studied subjects with Asperger's syndrome investigating their experience and self-report of hyperacusis and tinnitus. Our findings showed a higher rate of hyperacusis, with 38 out of 55 participants (69%) reporting hyperacusis and 19 (35%) reporting tinnitus [39]. Children who experience hyperacusis to certain sounds such as toilets flushing, fire alarms, and vacuum cleaners often dislike (potentially some form of misophonia) or even experience fear of (phonophobia) these acoustic signals. Hyperacusis could be a sign of auditory pathway problems or a sign of abnormalities in the limbic system [40,41]. While the damage to the peripheral and central auditory system triggers the hyperacusis signal, it is the limbic system that generates a negative emotion to sounds and communicates it to the auditory cortex, which drives the cognitive perception of the negative emotional reaction to sounds, causing hyperacusis [42,43]. Over time, children with ASD would experience hyperacusis to the situation that made the sound, not necessarily to the sound itself? [44]. This indicates some sort of anticipation which can show a similar behavioral response. Because of the connection between the limbic system and the autonomic nervous system via the amygdala, problems in the limbic system and amygdala cause overstimulation of the sympathetic pathway of the autonomic nervous system [45]. Potentially, this interaction is what causes the observed behavioral reactions to certain sounds in children with ASD ranging from running away from sound to completely avoiding sounds and/or rocking or running around in a circle [46]. Fortunately, children who experience hyperacusis due to negative emotions to sounds (68% to 76%) would have significant relief from hyperacusis with appropriate intervention [40,47,48]. Additionally, a recent study evaluated the relationship between hypersensitivity to sound and superior semicircular canal dehiscence [49]. This is a condition where the bony separation between the inner ear and the cranial cavity is very thin or absent. The authors reported that 29% of individuals with ASD, who showed hypersensitivity to sound, had superior semicircular canal dehiscence as revealed by CT studies.

Difficulty hearing in the presence of competing speech or background noise is another manifestation in children with ASD that has been reported [39-40]. It has been suggested that the normal inhibitory (suppressive) function of the Medial Olivocochlear Bundle (MOCB) on the electro motility of the outer hair cells plays a role as an auditory filter; thus improving signal to noise ratio and hence, enhancing hearing in background noise [50,51]. There is evidence from the literature suggesting that the function of the MOCB may be reduced in children with ASD [50]. Both distortion-product and transient OAE measures have been employed in many studies to assess the suppressive function of the MOCB [52,53].

The central auditory pathway plays an important role in the processing of auditory signals in unfavorable acoustic environments such as noisy restaurants. As mentioned earlier, there is evidenced of central auditory abnormalities in ASD population [7-9,11]. These abnormalities can cause children with ASD to struggle hearing in background noise. Additional auditory processing impairments reported in children with ASD includes inattentiveness to verbal stimuli, easily distracted by noise, delayed processing and understanding of speech [33-35]. Lau [54] has reported that 40% of their cohort of 600 individuals with APD had a diagnosis of ASD. The authors also suggest a positive relationship between the severity of ASD and auditory processing deficits. Children with a higher severity of autism will have multiple auditory processing deficits and severe communication deficits due to global involvement of the central auditory pathway and higher centers compared to children with low functioning autism. The presence of impaired auditory processing skills should be a warning sign to parents and clinicians for further testing. Application of auditory electrophysiologic evaluation techniques in this population has been very helpful to identify the site of the lesion along the central auditory pathway to distinguish between auditory processing deficits or a global, higher-order deficit as in ASD.

In addition to auditory manifestations, children may have

disturbance in their control of balance. In this case, children are overwhelmed by heights and may, for example, have extreme fear of stairs. They may also avoid fast moving activities because of fear. Balance abnormalities have been studied in this population. Posturographic evaluation of subjects with ASD has shown a larger sway on the platform compared to control subjects [55]. This finding indicates poor postural control in individuals with ASD. Reduced postural stability has also been reported in this population mostly due to multimodality sensory integration [56]. Regardless of the sensory overloads, systematic desensitization paradigms which involve the use of structured exposure to a feared stimulus would be helpful. Also, vestibular stimulation, such as swinging on a swing, while teaching speech could be effective. These systematic desensitization paradigms help children to habituate to these stimuli through minimizing the fear response.

Auditory Assessments

Comprehensive assessment is required in this special needs population for early identification of auditory deficits. However, evaluation in general is very challenging. Kaf, Akhtar and Barboa [57] have reported a high prevalence of uncompleted otoscopy (18%) when testing children with severe ASD, ages 2 to 9 years. This could be due to lack of cooperation as well as sensory issues with insertion of tips into the ear canal. Cuvo, Reagon, Ackerlund, Huckfeldt, and Kelly [58] referred to otoscopy as an "aversive sensory stimulus" for children with ASD. When evaluating the auditory function in children with ASD, patience and special techniques should be practiced. Kaf et al. [57] have reported that more children protested tympanometry (26%) and OAE (26%) than otoscopy. This lack of cooperation during tympanometry and OAE testing might be explained by several factors such as sensory issues with inserting tympanometry or OAE probes into the ear canal, leading to improper sealing of the ear tip, and the overreaction to the sounds presented into the ear, resulting in an increase of the child's noise level, which can interfere with OAE testing.

Therefore, modification of audiological evaluation is recommended. Best practices in any assessment of children with ASD call for a clinician to take additional time to establish rapport with the child. It is suggested that audiologists should allow the child time to get used to the room before testing. Audiologists, with the help of an assistant, should start behavioral hearing testing in a sound booth before conducting otoscopy, tympanometry or OAE so that the child does not get upset from having their ears touched. To get an overall picture of hearing threshold, Madell [59] suggested that audiologists should test at least one low and one mid-high frequency (500 and 2000 Hz), and to avoid the use of speech stimuli at the beginning of testing since most children with autism usually "tune out" to voices [59]. If results of hearing testing are unreliable in children with ASD because behavioral difficulties may interfere with testing, ABR testing is recommended for objective estimation of thresholds. Gravel, Dunn, Lee & Ellis [52] evaluated the hearing of 22 children with autism and 22 age-matched typically developing children. The researchers showed that the electrophysiologic and physiologic measures such as ABR and distortion-product OAEs had similar results in both groups; however, the behavioral thresholds were worse in the autistic group. These data suggested that the behavioral evaluations are less reliable in the autistic group compared to typically developing children and emphasize the inclusion of electrophysiologic evaluation in the hearing test battery of this vulnerable population to assess the peripheral and the brainstem auditory pathway.

Recording of OAEs with and without noise conditions helps assess the integrity of the MOCB pathway [50]. It is established that suppression of the OAE level reflects the normal inhibitory function of the MOCB on the outer hair cells [60,61]. A study from our laboratory evaluated the distortion-product OAE amplitudes in children with autism and a control group [62]. In the absence of noise, it was shown that distortion-product OAEs were reduced in the children with autism, suggesting cochlear dysfunction. Additionally, when OAEs were recorded in the presence of contralateral noise, the contralateral suppression effect was not as strong as those in the control group, suggesting dysfunction of the MOCB. These findings may explain why children with autism do suffer from hypersensitivity to sounds and difficulty hearing in background noise. A similar study in our laboratory [63] employed DPOAEs and contralateral suppression in children with Asperger's Syndrome (N=18 males) and an age-matched control group. Interestingly, no significant difference could be detected for the distortion-product OAE amplitudes and contralateral suppression between the two groups [63]. Thus, the diminished OAE suppression effect may constitute a neurophysiological evidence for reduced MOCB function mainly in children with low functioning autism, but not in children with high functioning autism.

In order to monitor and assess sound hypersensitivity and hyperacusis in the ASD population, a few questionnaires are available such as the Hyperacusis Questionnaire by Khalfa, Dubal, Veuillet, Perez-Diaz, Jouvent & Collet [64] and the recently validated Auditory Behavior Questionnaire by Egelhoff & Lane [65]. In addition, the use of pure tone measures such as uncomfortable loudness level, discomfort threshold and dynamic range can determine tolerance to loud sounds [66,47]. Evaluation of the tolerance level to sound can indirectly give indications about the site of lesion of hypersensitivity to sounds: the auditory (cochlear) pathway or the non-classical auditory (limbic system) pathway [47]. Children with auditory deficits such as cochlear lesion will have abnormally small dynamic ranges and low discomfort thresholds, which is a manifestation of hypersensitivity to sounds due to the recruitment effect. On the other hand, children with an immature limbic system often have wide dynamic ranges and high discomfort thresholds, indicating that their hypersensitivity to sounds is due to negative emotions to sounds due to involvement of the limbic system, not due to auditory deficits. This differential diagnosis is important to select children who can benefit from sound desensitization training. It has been recommended that children with high tolerance to loud sounds, as measured by the uncomfortable loudness level and discomfort threshold, would be the best candidates for sound desensitization training [40,44].

Although standard behavioral pure tone and speech threshold testing gives some insight about the overall processing of tones and speech, it does not provide specific information about the site of lesion along the central auditory pathway. As mentioned earlier, the use of auditory electrophysiologic measures in individuals with ASD is useful to determine whether the impaired auditory processing skills are due

to APD or due to higher-order deficits. Previous studies have shown some differences in ABR findings. In a study on 153 girls and boys with autism, prolonged III-V interpeak latency was evidenced when compared with data from a control group [67]. Somewhat similar findings were reported in another study which revealed prolonged ABR latencies in autistic individuals [68]. Electrophysiologic auditory event-related potentials and their magnetic counterparts have been employed extensively in the study of autism, as well. A study in 2011 investigated magnetic mismatch negativity field to tonal and vowel stimuli [69]. The data analysis showed significantly delayed responses in ASD participants particularly those with language impairment. Similar results were also recorded through magnetoencephalography which revealed delayed latency for late auditory responses in the ASD population [70]. Additional findings showed that children with autism have significantly smaller P200 and P300 amplitudes than normal controls [71,72]. The authors speculated that these abnormalities in higher auditory processing may be the cause of severe language disorder in children with autism. The above findings are an indication of the abnormal cognitive processing and long-term memory retrieval and can be used as biomarkers for language impairment and autism.

Auditory Management

Management of auditory complications in the ASD population is multi-dimensional and includes management for hearing loss, auditory processing disorder, and sound desensitization training for hyperacusis. Based on the findings of the dynamic range and the discomfort threshold, children with auditory deficits can benefit from auditory-based intervention such as amplification while children whose hyperacusis is due to a problem in the limbic system can benefit from sound desensitization training.

In the presence of hearing loss, use of FM systems has been shown to be very effective particularly for the enhancement of speech perception in noise [73,74]. Studies suggest that hearing aid fitting protocols and verification practices are similar in ASD versus non-ASD populations [75]. Studies on the effects of cochlear implants on profoundly deaf children with autism are promising and show significant improvements in auditory communication following the implantation [76]. The amplification benefit and the prognosis of ASD are much better if children on the spectrum do not have associated APD, which exacerbate the social and communication problems and impede learning through hearing.

In the presence of an abnormality in the limbic system, the management strategies for hyperacusis in the ASD population are comparable to those without ASD. Application of techniques such as sound therapy with the use of custom-made sound tracks, or ear level sound generators combined with habituation therapy and counseling is promising. They are aimed to retrain and reprogram the emotional and non-classical auditory pathway so that a child is not frightened by sounds. One of the most important steps in sound desensitization is avoiding unnecessary use of sound protection devices particularly in environments which are not extraordinarily loud. Unfortunately, many individuals with ASD are equipped with sound protection devices such as ear plugs or are avoiding participation in certain situations. Some parents believe that these kinds of protection are helpful and insist on using them. In most cases, parents should help the child confront the challenges presented by being in the presence of noise and not purposely keep the child from exposure to these settings. According to Lucker and Doman [44] these are inappropriate solutions because they focus on ameliorating the symptoms not treating the underlying cause of the problem.

The sound desensitization training is usually a two-step procedure: listening training for gradual desensitization of the limbic system and then sound desensitization training. The overall goal of the training is to retrain and reprogram the emotional and non-classical auditory pathway so that a child is not frightened by sounds. The first step starts with acoustical enrichment of the environment with pleasant sounds such as music, musical toys and computer generated sounds. Successful listening training should expedite the sound desensitization process. Some clinicians recommend "breaking" the bond between the auditory cortex and the limbic systems, which is potentially causing hypersensitivity to sound [49]. The negative emotional description of loud sounds such as "scary and fearful sounds" should be replaced by words such as "unwanted sounds" [49]. A simple method of sound desensitization is to ask the individuals (or their parents) to provide the clinician with a list of sounds that are more annoying than others. The list of annoying sounds may include fire alarms, toilets flushing, or vacuum cleaners. Most of these sounds can be downloaded as sound files to personal music players. Patients are instructed to listen to these "unwanted sounds" at a low level for 15-20 minutes a day and increase the volume slightly every week. This is a very effective method in reducing negative emotional reactions to sounds and thus reducing hyperacusis in the ASD population. Furthermore, extensive directive counseling and cognitive behavioral therapy by trained practitioners will ease the process of desensitization to sound.

Regarding specific auditory training therapies, three exist for children with ASD. Auditory Integration Training (AIT) is the largest and most studied of these three therapies [77,78]. Supporters of AIT believe that it can improve attention, auditory processing, expressive language, and auditory comprehension, as well as decrease irritability and reduce lethargy [78,79]. However, conflicting results were reported about the efficacy of AIT using systematic review of literature and controlled studies. After review of 28 electronic databases [78,80] and 19 studies [81], these researchers and others reported that AIT is not efficacious or may have small or inconsistent improvement on behavior for school-age children with APD or other related disorders [82]. In contrast, other researchers compared pre- and post- AIT training using controlled studies to determine post training effect on social cognition in children with ASD [83] and on cognitive processing using P300 response in children with ASD and APD [84,85]. These researchers reported improved social cognition and cognitive processing following three months of AIT training. Although the American Academy of Pediatrics [79] allows the use of sensory-based therapies as one of the components of a comprehensive treatment plan, it encourages more research on its effectiveness.

Based on the conflicting literature and inconclusive evidence about AIT benefit, several professional organizations do not support using AIT as a mainstream treatment without compelling evidence based on well-designed, institutionally approved research studies to validate its effectiveness and safety [86-89] as well as assessing its benefit in young children with ASD [90]. According to ASHA

(American Speech-Language-Hearing Association) [91], members may be violating the Code of Ethics if they promote or provide AIT training. In contrast to the inconsistent results of AIT, researchers reported that auditory training programs such as Fast ForWord, Earobics, and neurofeedback have shown improvements in auditory functioning among school-age children with APD [80] and cognitive function of children with ASD [92] as well as reconfiguring the neural network of the auditory pathways [93].

Conclusions

Many children diagnosed with ASD often have auditory manifestations such as hearing loss, tinnitus, difficulty hearing in background noise, inattentiveness to verbal stimuli, selective auditory attention and atypical performance of their central auditory system. Neuroanatomical and electrophysiological changes have been shown to support the underlying auditory deficits in ASD. With continued growth of ASD, the number of individuals needing clinical services and intervention for hearing loss and hyperacusis is expected to follow the same path. Comprehensive audiologic evaluations using behavioral and electrophysiological measures are recommended in this population to assess the peripheral and central auditory system, as well as to monitor intervention progress. Since hyperacusis, a common manifestation in children with ASD, is shown to be related to negative emotional and memory reactions to certain sounds, audiologists should assess the non-classical pathway (limbic system). This is important to determine if hyperacusis is due to an auditory problem or due to a limbic system deficit. This should help in selecting candidates for intervention, amplification and FM systems in auditory deficits, and auditory desensitization training in cases with a limbic system deficit. Although there are different auditory training approaches to retrain the brain, controversies about the benefit of auditory training such as AIT exist, which require further research to meet scientific standards regarding efficacy and safety. The key to success in this process is for audiologists to continue playing an active role by incorporating electrophysiological measures for cross validation of hearing thresholds and determining the site of lesion, selecting the candidates for intervention, and implementing intervention approaches and monitoring their effectiveness.

References

1. Kanner L. Autistic disturbances of affective contact. Nervous Child. 1943; 2: 217-250.
2. Fombonne E. The prevalence of autism. JAMA. 2003; 289: 87-89.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (5th ed). 2013.
4. Fombonne E. Is a large head circumference a sign of autism? J Autism Dev Disord. 2000; 30: 365.
5. Aylward EH, Minshew NJ, Field K, Sparks BF, Singh N. Effects of age on brain volume and head circumference in autism. Neurology. 2002; 59: 175-183.
6. Courchesne E, Carper R, Akshoomoff N. Evidence of brain overgrowth in the first year of life in autism. JAMA. 2003; 290: 337-344.
7. Illing RB, Kraus KS, Michler SA. Plasticity of the superior olivary complex. Microsc Res Tech. 2000; 51: 364-381.
8. Lukose R, Schmidt E, Wolski TP Jr, Murawski NJ, Kulesza RJ Jr. Malformation of the superior olivary complex in an animal model of autism. Brain Res. 2011; 1398: 102-112.
9. Kulesza RJ Jr, Lukose R, Stevens LV. Malformation of the human superior olive in autistic spectrum disorders. Brain Res. 2011; 1367: 360-371.
10. Visser E, Zwiers MP, Kan CC, Hoekstra L, Van Opstal AJ, Buitelaar JK. Atypical vertical sound localization and sound-onset sensitivity in people with autism spectrum disorders. J Psychiatry Neurosci. 2013; 38: 120177.
11. Gillberg C. Neurodevelopmental processes and psychological functioning in autism. Dev Psychopathol. 1999; 11: 567-587.
12. Welsh JP, Yuen G, Placantonakis DG, Vu TQ, Haiss F, O'Hearn E, et al. Why do Purkinje cells die so easily after global brain ischemia? Aldolase C, EAAT4, and the cerebellar contribution to posthypoxic myoclonus. Adv Neurol. 2002; 89: 331-359.
13. Ecker C, Suckling J, Deoni SC, Lombardo MV, Bullmore ET, Baron-Cohen S, et al. Brain anatomy and its relationship to behavior in adults with autism spectrum disorder: a multicenter magnetic resonance imaging study. Arch Gen Psychiatry. 2012; 69: 195-209.
14. Fletcher PT, Whitaker RT, Tao R, DuBray MB, Froehlich A, Ravichandran C, et al. Microstructural connectivity of the arcuate fasciculus in adolescents with high-functioning autism. Neuroimage. 2010; 51: 1117-1125.
15. Alexander AL, Lee JE, Lazar M, Boudos R, DuBray MB, Oakes TR, et al. Diffusion tensor imaging of the corpus callosum in Autism. Neuroimage. 2007; 34: 61-73.
16. Rojas DC, Camou SL, Reite ML, Rogers SJ. Planumtemporale volume in children and adolescents with autism. J Autism Dev Disord. 2005; 35: 479-486.
17. Abrams DA, Lynch CJ, Cheng KM, Phillips J, Supekar K, Ryali S, et al. Underconnectivity between voice-selective cortex and reward circuitry in children with autism. Proc Nat Acad Sci. 2013; 110: 12060-12065.
18. Bryant DM, Hoeft F, Lai S, Lackey J, Roeltgen D, Ross J, et al. Sex chromosomes and the brain: a study of neuroanatomy in XYY syndrome. Dev Med Child Neurol. 2012; 54: 1149-1156.
19. Lewis WW, Sahin M, Scherrer B, Peters JM, Suarez RO, Vogel-Farley VK, et al. Impaired language pathways in tuberous sclerosis complex patients with autism spectrum disorders. Cereb Cortex. 2013; 23: 1526-1532.
20. Moller A, Kern JK, Grannemann B. Are the non-classical auditory pathways involved in autism and PDD? Neurol Res. 2005; 27: 625-629.
21. Moller AR, Rollins PR. The non-classical auditory pathways are involved in hearing in children but not adults. Neurosci Lett. 2005; 319: 41-44.
22. Aylward EH, Minshew NJ, Goldstein G, Honeycutt NA, Augustine AM, Yates KO, et al. MRI volumes of amygdala and hippocampus in non-mentally retarded autistic adolescents and adults. Neurology. 1999; 53: 2145-2150.
23. Kemper TL, Bauman ML. The contribution of neuropathologic studies to the understanding of autism. Neurol Clin. 1993; 11: 175-187.
24. LeRoy CJ. The postnatal development of the human cerebral cortex. Cambridge (MA): Harvard University Press. 1993.
25. Jacobson M. Developmental neurobiology. New York: Plenum Press. 1991.
26. Bauman ML, Kemper TL. Neuroanatomic observations of the brain in autism. Bauman IML, Kemper TL, editors. In: The Neurobiology of Autism. Baltimore: Johns Hopkins UP. 1994.
27. Woodward CR, Goodwin MS, Zelazo PR, Aube D, Scrimgeour M, Osthothoff T, et al. A comparison of automic, behavioral, and parent-report measures of sensory sensitivity in young children with autism. Res Autism Spectrum Disord. 2012; 6: 1234-1246.
28. Beers AN, McBoyle M, Kakande E, Dar Santos RC, Kozak FK. Autism and peripheral hearing loss: a systematic review. Int J Pediatr Otorhinolaryngol. 2014; 78: 96-101.
29. Szymanski CA, Brice PJ, Lam KH, Hotto SA. Deaf children with autism spectrum disorders. J Autism Dev Disord. 2012; 42: 2027-2037.
30. Rosenhall U, Nordin V, Sandstrom M, Ahlsen G, Gillberg C. Autism and hearing loss. J Autism Dev Disord. 1999; 29: 349-357.

31. Joint Committee on Infant Hearing. Year 2007 position statement: Principles and guidelines for EHDI programs. Pediatrics. 2007; 120: 898-921.

32. Jure R, RapinI, Tuchman RF. Hearing-impaired autistic children. Dev Med Chil Neurol. 1991; 33: 1062-1072.

33. Vernon M, Rhodes A. Deafness and autistic spectrum disorders. Am Ann Deaf. 2009; 154: 5-14.

34. Gordon AG. Newborn encephalopathy, autism, and deafness. Dev Med Child Neurol. 2007; 49: 157-158.

35. Peterson CC, Wellman HM, Liu D. Steps in theory-of-mind development for children with deafness or autism. Child Dev. 2005; 76: 502-517.

36. Gordon AG. Ear disorders in autistic children. J Autism Dev Disord. 1989; 19: 470-473.

37. Smith DE, Miller SD, Stewart M, Walter TL, McConnell JV. Conductive hearing loss in autistic, learning-disabled, and normal children. J Autism Dev Disord. 1988; 18: 53-65.

38. Rafal Z. Conductive hearing loss in children with autism. Eur J Pediatr. 2013; 172: 1007-1010.

39. Danesh, AA, Andreassen W, Scott J, Kaf W, Bennett K, Flood B. Tinnitus and Hyperacusis in Autism Spectrum Disorders with Emphasis on Asperger's Syndrome (AS). Abstract & Oral presentation at the IXth International Tinnitus Seminar. Goteborg, Sweden. 2008: 15-18.

40. Gomes E, Rotta NT, Pedroso FS, Sleifer P, Danesi MC. Auditory hypersensitivity in children and teenagers with autism spectrum disorder. Arq Neuro-Psiquiatr. 2004; 62: 797-801.

41. Stiegler L. Understanding sound sensitivity in individuals with autism spectrum disorders. Focus on Autism and Other Developmental Disabilities. 2010; 25: 67-75.

42. Katzenell U, Segal S. Hiperacusis: review and clinical guidelines-invited comments. Oto Neurotol. 2001; 22: 326-327.

43. Nigam A, Samuel P. Hyperacusis and Williams syndrome. J Laryngol Otol. 1993; 108: 494-496.

44. Lucker JR, Doman A. Hypersensitivity and autism spectrum disorders: an emotional response. Autism Sci Digest. 2012; 4: 103-108.

45. Kushki A, Brian J, Dupuis A, Anagnostou E. Functional autonomic nervous system profile in children with autism spectrum disorder. Molecular Autism. 2014; 5: 39.

46. Hirstein W, Iversen P, Ramachandran VS. Autonomic responses of autistic children to people and objects. Proc R Sac Lond B. 2001; 268: 1883-1888.

47. Lucker JR. Helping children with APD: Individualized interventions through effective management, accommodation & treatment. Premier Publishing & Media. 2011.

48. Lucker JR. Auditory hypersensitivity in children with autism spectrum disorders. Focus on Autism & Other Dev Dis. 2013; 28: 184-191.

49. Thabet EM, Zaghloul HS. Auditory profile and high resolution CT scan in autism spectrum disorders children with auditory hypersensitivity. Eur Arch Otorhinolaryngol. 2013; 270: 2353-2358.

50. Giraud AL, Collet L, Chery-Croze S, Magnan J, Chays A. Evidence of a medial olivocochlear involvement in contralateral suppression of otoacoustic emissions in humans. Brain Res. 1995; 705: 15-23.

51. Khalfa S, Morlet T, Micheyl C, Morgon A, Collet L. Evidence of peripheral hearing asymmetry in humans: clinical implications. Acta Oto-laryngologica. 1997; 117: 192-196.

52. Gravel JS, Dunn M, Lee WW, Ellis MA. Peripheral audition of children on the autistic spectrum. Ear Hear. 2006; 27: 299-312.

53. Tas A, Yagiz R, Tas M, Esme M, Uzun C, Karasalihoglu AR. Evaluation of hearing in children with autism by using TEOAE and ABR. Autism. 2007; 11: 73-79.

54. Lau C. Audiology must take its rightful place on the autism team. Hearing Journal. 2012; 65: 18-20.

55. Molloy CA, Dietrich KN, Bhattacharya A. Postural stability in children with autism spectrum disorder. J Autism Dev Disord. 2003; 33: 643-652.

56. Minshew NJ, Sung K, Jones BL, Furman JM. Underdevelopment of the postural control system in autism. Neurology. 2004; 63: 2056-2061.

57. Kaf WA, Akhtar U, Barboa L. Audiological Evaluation & Speech Assessment in Children with Special Needs. Poster presentation at the ASHA Convention. Atlanta, GA. 2012; 15-17.

58. Cuvo AJ, Reagon AL, Ackerlund J, Huckfeldt R, Kelly C. Training children with autism spectrum disorders to be compliant with a physical exam. Res. Autism Spectr. Disord. 2010; 4: 168-185.

59. Madell JR. Behavioral Evaluation of Hearing in Infants and Children. New York, NY: Thieme Medical. 1998.

60. Collet L, Veuillet E, Bene J, Morgon A. Effects of contralateral white noise on click-evoked missions in normal and sensorineural ears: Towards an exploration of the medial olivocochlear system. Audiology. 1992; 31: 1-7.

61. Guinan JJ Jr. Physiology of olivocochlear efferents. Dallos P, Popper AN, Fay RR, editors. In: The Cochlea. Springer Handbook of Auditory Research. Springer, New York. 1996; 8: 435.

62. Danesh AA, Kaf WA. DPOAEs and contralateral acoustic stimulation and their link to sound hypersensitivity in children with autism. Int J Audiol. 2012; 51: 345-352.

63. Kaf WA, Danesh AA. Distortion-product otoacoustic emissions and contralateral suppression findings in children with Asperger's Syndrome. Int J Pediatr Otorhinolaryngol. 2013; 77: 947-954.

64. Khalfa S, Dubal S, Veuillet E, Perez-Diaz F, Jouvent R, Collet L. Psychometric normalization of a hyperacusis questionnaire. ORL J Otorhinolaryngol Relat Spec. 2002; 64: 436-442.

65. Egelhoff K, Lane AE. Brief report: preliminary reliability, construct validity and standardization of the Auditory Behavior Questionnaire (ABQ) for children with autism spectrum disorders. J Autism Dev Disord. 2013; 43: 978-84.

66. Khalfa S, Bruneau N, Roge B, Georgieff N, Veuillet E, Adrien JL, et al. Increased perception of loudness in autism. Hearing Research. 2004; 198: 87-92.

67. Rosenhall U, Nordin V, Brantberg K, Gillberg C. Autism and auditory brain stem responses. Ear&Hear. 2003; 24: 206-214.

68. Fujikawa-Brooks S, Isenberg AL, Osann K, Spence MA, Gage NM. The effect of rate stress on the auditory brainstem response in autism: a preliminary report. Int J Audiol. 2010; 49: 129-140.

69. Roberts TP, Cannon KM, Tavabi K, Blaskey L, Khan SY, Monroe JF, et al. Auditory magnetic mismatch field latency: a biomarker for language impairment in autism. Biol Psychiatry. 2011; 70: 263-269.

70. Roberts TP, Khan SY, Rey M, Monroe JF, Cannon K, Blaskey L, et al. MEG detection of delayed auditory evoked responses in autism spectrum disorders: towards an imaging biomarker for autism. Autism Res. 2010; 3: 8-18.

71. Donchin E, Ritter W, McCallum C. Cognitive psychophysiology: The endogenous componenets of the ERP. Callaway E, Tueting P, Koslow S, editors. In: Brain event-related potentials in man. New York: Academic Press; 1978; 349-441.

72. Novick B, Vaughan HG, Kurtzberg D, Simon R. An electrophysiologic indication of auditory processing defects in autism. Psychiat Res. 1980; 3: 107-115.

73. Rance G, Saunders K, Carew P, Johansson M, Tan J. The use of listening devices to ameliorate auditory deficit in children with autism. J Pediatr. 2014; 164: 352-357.

74. Schafer EC, Mathews L, Mehta S, Hill M, Munoz A, Bishop R, et al. Personal FM systems for children with Autism Spectrum Disorders (ASD) and/or attention-deficit hyperactivity disorder (ADHD): an initial investigation. J CommunDisord. 2013; 46: 30-52.

75. Tharpe AM, Fino-Szumski MS, Bess FH. Survey of hearing aid fitting practices

for children with multiple impairments. Am J Audiol. 2001; 10: 32-40.

76. Donaldson AI, Heavner KS, Zwolan TA. Measuring progress in children with autism spectrum disorder who have cochlear implants. Arch Otolaryngol Head Neck Surg. 2004; 130: 666-671.

77. Sinha Y, Silove N, Wheeler D, Williams K. Auditory integration training and other sound therapies for autism spectrum disorders. Archives of Disease in Childhood. 2006; 91: 1018-1022.

78. Sinha Y, Silove N, Hayen A, Williams K. Auditory integration training and other sound therapies for Autism Spectrum Disorders (ASD). The Cochrane Collaboration. 2011.

79. American Academy of Pediatrics. Auditory integration training and facilitated communication for autism. Pediatrics.1998; 102: 431-433.

80. Fey ME, Richard GJ, Geffner G, Kamhi AG, Medwetsky L, Paul D, et al. Auditory Processing Disorders and Auditory/Language Interventions: An Evidence-Based Systematic Review. Language, Speech and Hearing Services in Schools. 2011; 42: 246-264.

81. Case-Smith J, Weaver LL, Fristad, MA. A systematic review of sensory processing interventions for children with autism spectrum disorders. Autism. 2014; 19: 133-148.

82. Gravel JS. Auditory integration training: Placing the burden of proof. American Journal of Speech-Language Pathology. 1994; 3: 25-29.

83. Al-Ayadhi L, Al-Drees A, Al-Arfaj A. Effectiveness of auditory integration therapy in autism spectrum disorders-prospective study. Autism Insights. 2013; 5: 13.

84. Edelson SM, Arin D, Bauman M, Lukas SE, Rudy JH. Sholar M, et al. Auditory integration training: A double-blind study of behavioral, electrophysiological, and audiometric effects in autistic subjects. Focus on Autism and Other Developmental Disabilities. 1999; 14: 73-81.

85. Russo NM, Hornickel J, Nicol T, Zecker S, Kraus N. Biological changes in auditory function following training in children with autism spectrum disorders. Behav Brain Funct. 2010; 6: 60.

86. American Academy of Audiology (AAA). PositionStatement. Auditory integration training. 2010.

87. American Speech-Language-Hearing Association. Auditory integration training [Position Statement]. 2004.

88. Educational Audiology Association. Auditory integration training: Educational Audiology Association position statement. Educational Audiology Newsletter. 1997.

89. National Initiative for Autism: Screening and Assessment. National autism plan for children (NAPC). National Autistic Society, London. 2003.

90. New York State Department of Health. Clinical practice guideline: Report of the recommendations. Autism/pervasive developmental disorders, assessment and intervention for young children (age 0–3 years). 1999.

91. American Speech-Language-Hearing Association. ASHA Adopts AIT Policy. The ASHA Leader. 2003.

92. Kouijzer ME, Van Schie HT, Gerrits BJ, Buitelaar JK, de Moor JM. Is EEG-biofeedback an effective treatment in autism spectrum disorders? A randomized controlled trial. Appl Psychophysiol Biofeedback. 2013; 38: 17-28.

93. Haller S, Kopel R, Jhooti P, Haas T, Scharnowski F, Lovblad KO, et al. Dynamic reconfiguration of human brain functional networks through neurofeedback. Neuroimage. 2013; 81: 243-252.

Austin J Autism & Relat Disabil - Volume 1 Issue 1 - 2015
Submit your Manuscript | www.austinpublishinggroup.com

Citation: Danesh AA, Kaf WA, Abdelhakiem MK, Danesh D and Scott J. Auditory Manifestations and Intervention in Children with Autism Spectrum Disorders. Austin J Autism & Relat Disabil. 2015;1(1): 1005.