

Sequel of Auditory Dys-synchrony on Speech Production

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CHAPTER 1

INTRODUCTION

1
2
3
4 The disorder of auditory dys-synchrony (AD) is characterized by the absence
5 of auditory brainstem responses despite otoacoustic emissions and/or cochlear
6 microphonics being present (Sininger & Oba, 2001; Starr, Picton, Sininger, Hood, &
7 Berlin, 1996). At first, the disorder was termed as auditory neuropathy as majority of
8 the affected individuals were reported to have associated peripheral neuropathy. Later,
9 in view of the lesion being restricted to inner hair cells in some of the cases
10 (Miyamoto, Kirk, Renshaw, & Hussian, 1999), the term auditory dys-synchrony was
11 suggested (Berlin, Hood, Morlet, Rose, & Brashears, 2003). Hayes, Sininger and Starr
12 (2012) suggested the term auditory neuropathy spectrum disorder (ANSD)
13 considering that the site of damage is not confined to a particular locus in most of
14 these persons. Rather, there are different affected loci. Henceforth in this study, the
15 condition will be uniformly referred to as ANSD.

16
17 Speech identification abilities of individuals with auditory dys-synchrony are
18 disproportionate to the degree of their hearing loss (Starr et al., 1996; Zeng & Liu,
19 2006) and are the cardinal characteristics of persons with ANSD. Unlike cochlear
20 hearing loss, speech perception abilities in these individuals is believed to vary based
21 on the extent of distortion of temporal cues at suprathreshold levels rather than access
22 to speech spectrum (related to audibility), (Zeng, Oba, Garde, Sininger, & Starr, 1999;
23 Zeng et al., 2005). The psychoacoustical, neurophysiological and perceptual aspects
24 of individuals with ANSD are well established (Kumar & Jayaram, 2006; Norton &
25 Widen, 1990; Sininger, Hood, Starr, Berlin, & Picton, 1995; Sininger & Oba, 2001).

1 In general, studies report that both psychoacoustical abilities and speech perception in
2 ANSD are considerably poorer than that in cochlear hearing loss.

3

4 Research has revealed an alarming incidence and prevalence of ANSD among
5 individuals with hearing impairment. The incidence of ANSD in patients with
6 profound hearing loss is estimated to be 10% with a prevalence of 0.23% among high-
7 risk babies of **United States of America (USA)** (Kraus, Ozdamar, Stein, & Reed,
8 1984; Rance et al., 1999). In a hospital-based statistics, Rance et al. (1999) assessed
9 5199 'at risk' children for ANSD. The prevalence of ANSD among children at risk
10 was 1 in 433 (0.23%) and in children with hearing impairment, it was 1 in 9 (11.01%).
11 It was estimated that 2% to 15% of infants with hearing loss may exhibit ANSD
12 (Rance et al., 1999; Sininger, 2002).

13

14 Davis and Hirsh (1979) reported that 1 in 200 children with hearing
15 impairment **in USA** exhibit the clinical trait of ANSD. **Tang, McPherson, Yuen,**
16 **Wong, and Lee (2004)** examined the frequency of occurrence of ANSD in school-
17 aged children with hearing impairment and reported a prevalence of 2.44%. The
18 prevalence of ANSD in India has been reported to be 0.54% among individuals with
19 sensorineural hearing loss (Kumar & Jayaram, 2006).

20

21 Starr, Sininger, and Praat (2000) reported the occurrence of peripheral
22 neuropathy in ANSD among 80% of patients aged greater than 15 years. It was also
23 reported that ANSD in 96% of affected individuals is bilateral in nature and no gender
24 difference was noted. On the contrary, Narne, Prabhu, Chandan, and Deepthi (2014)
25 reported a female to male ratio of 1.25:1 in Indian population with ANSD.

26

1 Development of speech is primarily through auditory mode. Disruption in the
2 auditory feedback, as in instances of cochlear hearing loss, has been reported to have
3 deleterious influence on speech production (Culbertson & Kricos, 2002; Dunn &
4 Newton, 1986; Grover, 1998; Hudgins & Numbers, 1942; Jayaradha, 2001; Smith,
5 1982). This could be manifested either as delay or deviance in the domains of speech
6 and language. Specifically, with reference to speech, deficits are reported in
7 articulation, voice and fluency (Culbertson & Kricos, 2002; Dunn & Newton, 1986).
8 These speech production deficits are attributed to the defective auditory feedback
9 secondary to hearing loss (Binnie, Daniloff, & Buckingham, 1982; Cowie, Douglas-
10 Cowie, & Kerr, 1982) and are found to be directly related to the severity of hearing
11 loss and speech identification scores (Boothroyd, 1984; Perkell, Mathies & Lane,
12 1997; Smith, 1982).

13
14 Disruptions in the perception of temporal cues are demonstrated in children as
15 well as adults with ANSD (Kraus et al., 2000; Michalewski, Starr, Nguyen, Kong, &
16 Zeng, 2005; Rance, McKay, & Gradyen, 2004; Starr et al., 1991; Zeng et al., 1999;
17 Zeng, Kong, Michalewski, & Starr, 2005). In addition to the distortion of the spectral
18 information seen in individuals with cochlear hearing loss (Moore, 1995; Rance et al.,
19 2004), individuals with ANSD have relatively greater distortion in temporal
20 information (Kraus et al., 2000; Rance et al., 2004; Zeng et al., 1999; 2005). Hence,
21 the input signal in the auditory system is expected to be a lot more distorted in
22 individuals with ANSD compared to those with cochlear hearing loss. This is
23 supported by the findings of earlier studies that have reported speech perception in
24 individuals with ANSD (Kumar & Jayaram, 2006; Rance et al., 2004; Starr et al.,
25 1996, 2000; Zeng & Liu, 2006; Zeng, Oba, & Starr, 2001). However, speech
26 characteristics of adults with ANSD have not been systematically explored.

1 **1.1 Justification for the Study**

2 Speech characteristics of adults with ANSD have not been systematically
3 explored in any of the earlier western studies. However, Dayal and Maruthy (2009)
4 found that speech of ANSD is perceptually abnormal, more so in its prosody. They
5 also reported a significant high correlation between deficits in speech production and
6 speech perception scores. However, it was only a preliminary attempt and did not
7 include detailed evaluation of articulation, voice and fluency.

8
9 Detailed evaluation of speech characteristics will help enhance our
10 understanding of the influence of long term disruption in the temporal characteristics
11 of the input auditory signal, if any, on speech production in ANSD. This would
12 further help in verifying the Direction into velocities of articulators (DIVA) model of
13 speech production and will validate the findings of Dayal and Maruthy (2009). If
14 speech is found to be deviant, it will further stress on the need for early identification
15 and rehabilitation of speech related deficits of ANSD. The specific deviant
16 characteristics will guide us in understanding the auditory cues to speech production
17 relationship in a better way. These would further aid us to develop better management
18 strategies, thus improving the quality of life of individuals with ANSD.

19

20 **1.2 Objectives of the Study**

21 The objectives of the present study were:

- 22 1. To characterize speech production of individuals with ANSD in terms of
23 segmental and suprasegmental features.
- 24 2. To assess the relationship across auditory processing deficits, speech
25 perception deficits and speech production in individuals with ANSD.

26

1 Similar findings have been obtained in Sininger and Oba (2001) and Cheng et al.
2 (2005). Kumar and Jayaram (2006) reported absence of MEMRs in all of their
3 subjects. The absence of MEMRs has been attributed to the inability of afferent
4 pathway in generating sufficient synchronized neural discharge that trigger stapedius
5 muscle contraction (Starr et al., 1998). The presence of non-acoustic middle-ear
6 muscle reflexes in ANSD (Gorga, Stelmachowicz, Barlow, & Brookhouser, 1995;
7 Starr et al., 1998) suggests normal functioning of the efferent part of the MEMR arc.

8

9 ***2.1.3 Otoacoustic Emissions***

10 Persons with ANSD are found to have higher mean amplitude of TEOAEs
11 compared to their normal hearing controls (Hood, Berlin, Bordelon, & Rose, 2003;
12 Kumar & Jayaram, 2005). Higher amplitude is attributed to the lack of efferent
13 suppression in ANSD. However, the lack of efferent suppression and acoustic reflexes
14 which are thought to protect the cochlea from loud sounds may result in permanent
15 OHC damage over time (Berlin, Hood, Cecola, Jackson, & Szabo, 1993; Sininger et
16 al., 1995; Starr et al., 1996). Reduced OAE amplitude and deterioration of OAEs
17 have been found in persons with longstanding ANSD (Deltenre et al., 1999). These
18 findings have been speculated to be the result of hearing aid use or the effect of OTOF
19 (Otoferlin) mutation in OHCs (Rodriguez-Ballestros et al., 2003). Nonetheless,
20 researchers have reported that the presence or absence of OAE does not relate to
21 speech perception in persons with ANSD (Rance et al., 1999; Starr et al., 2000).

22

23 ***2.1.4 Auditory Evoked Potentials***

24 Auditory brainstem responses (ABRs) are known to be absent or abnormal in
25 persons with ANSD. While most show absent ABRs, a few of them show present but

1 abnormal ABRs. Starr et al. (2000) reported that 73% of the patients tested had absent
2 ABRs, whereas 21% showed a fifth peak with reduced amplitude and 6% of them had
3 the third and fifth peak.

4
5 Electrocochleography (EcochG) is recommended in ANSD to confirm the
6 peripheral functions (Arslen, Turrini, Lupi, Genovese, & Orzan, 1997; Kraus et al.,
7 1984). The presence of summing potential in EcochG indicates normal functioning
8 of inner hair cells (Durrant, Wang, Ding, & Salvi, 1998). Shi, Kempfle, and Edge
9 (2012) reported that the input-output (I/O) function of cochlear microphonics helps in
10 differentiating the site of lesion in persons with ANSD. If the I/O function of cochlear
11 microphonics shows good nonlinearity, it indicates that the site of lesion could be
12 either inner hair cells, synapse between IHCs and eighth nerve, or at the eighth nerve
13 itself. On the contrary, reduced nonlinearity in the I/O function of cochlear
14 microphonics indicates that the site of lesion could be at the synapse between IHCs
15 and eighth nerve or at the eighth nerve itself.

16
17 Satya-Murti, Wolpaw, Cacace, and Schaffer (1983) observed cortical auditory
18 evoked potentials (CAEPs) for the first time in six individuals in whom the ABR was
19 absent. Starr et al. (1996) could detect N1 and P2 components of CAEPs in three out
20 of five individuals with ANSD. Rance, Cone-Wesson, Wunderlich, and Dowel (2002)
21 reported the presence of CAEPs in 50% (9 out of 18) of individuals with ANSD.
22 Since the CAEPs do not depend on the neural synchrony as much as the earlier
23 potentials, the effect of temporal disruption on the cortical potentials is minimal
24 (Hood, 1998; Rapin & Gravel, 2003). Kumar and Jayaram (2005) reported the
25 presence of P1 and N1 in 10 out of 14 individuals with ANSD being tested whereas

1 P2 and N2 components were present in all the 14 individuals. In their study, mismatch
2 negativity was also recordable in 9 out of 14 participants. Furthermore, there was no
3 significant difference in the mismatch negativity between normals and persons with
4 ANSD even though persons with ANSD were not able to discriminate the stimulus
5 contrast behaviorally. On the contrary, delay in the late latency responses has been
6 reported for tonal (Starr et al., 2004), click stimuli (Narne & Vanaja, 2008a) as well as
7 gaps in noise (Michalewski et al., 2005) stimuli. Compared to controls, a delay is
8 reported in individuals with ANSD, ranging between 10 ms to 80 ms depending on
9 the different stimuli used as listed above.

10

11 Stimulus characteristics are also reported to influence the cortical responses.
12 In normal hearing individuals, cortical response to unvoiced stimulus has two peaks;
13 one corresponds to the burst/aspiration (usually labelled as P1' in recording) and the
14 second corresponds to the onset of voicing (Sharma & Dorman, 1999). The early
15 peaks were not detected in ANSD (Kraus et al., 2000). The absence of P1' suggests
16 that the transient cues, plausibly related to stimulus burst, are poorly represented.

17

18 The relationship between the CAEPs and speech perception abilities in ANSD
19 has also been investigated. Kumar and Jayaram (2005) reported that there is no
20 correlation of speech perception with the latency or amplitude of CAEPs in
21 individuals with ANSD. However, when participants with ANSD were grouped based
22 on their speech identification scores (SIS) as good (SIS>50%) and poor performers
23 (SIS<50%), the amplitude of N1-P2 complex was found to be lower for poor
24 performers compared to good performers (Narne & Vanaja, 2009).

1 Recording late latency responses is also suggested to predict the speech
2 perception score in ANSD. Rance et al. (2002) correlated the aided speech perception
3 scores of individuals with ANSD with their late latency responses. They found that
4 children with ANSD who had measurable speech recognition scores showed good late
5 latency responses that positively correlated with the aided performance. Those
6 individuals who showed presence of CAEPs had an average speech perception score
7 of 60%, while those without CAEPs had an average perception score of only 6%. The
8 authors hypothesized that preserved synchrony at the cortical level may be the
9 contributing factor for better speech perception. Similar findings were reported in
10 children using cochlear implants. Alvarenga et al. (2012) reported the presence of P1
11 in 12 of 14 (85%) children using cochlear implants and concluded that the P1
12 component can be an indicator of central auditory cortical development and a
13 predictor of speech perception in implanted children with ANSD.

14

15 **2.2 Age of Onset of ANSD**

16 Berlin et al. (2010) studied the occurrence of ANSD in 260 patients and
17 reported that 85.76% of their participants had an onset below the age of 12 years. A
18 very few of them had an onset during puberty and adulthood. On the contrary, the
19 other studies indicate the onset to be in the second decade of life. Rance (2005) found
20 that symptoms started after 15 years of age in nearly 80% of individuals with ANSD,
21 whereas Wang, Gu, Han, and Yang (2003) reported late onset ANSD in their study.
22 The onset of ANSD in Indian scenario is reported to be between 10 and 20 years (Jijo
23 & Yathiraj, 2012), more frequently between 10 and 14 years of age (Kumar &
24 Jayaram, 2006). Similar findings were reported by Prabhu, Avilala, and Manjula
25 (2012), and Shivashankar, Satishchandra, Shashikala, and Gore (2003).

1 **2.3 Aetiology and Pathophysiology of ANSD**

2 The etiological factors of ANSD include genetic, infectious, toxic-metabolic
3 (hypoxia, hyperbilirubinemia) and immunological disorders (drug reaction,
4 demyelination). In most cases, the origin of ANSD is reported to be idiopathic in
5 nature (Berlin et al., 2010; Starr et al., 2000; Starr, Zeng, Michalewski, & Moser,
6 2008). Conditions such as hyperbilirubinemia, ototoxic drug regimen, low birth
7 weight, low APGAR scores, exposure to aminoglycosides, hyponatremia, anoxia and
8 family history of deafness are also found to be the causative factors (Berlin et al.,
9 2003). Leonardis et al. (2000) reported a gypsy family with hereditary motor and
10 sensory neuropathy (Lom HMSN-L) associated with ANSD. Similarly, X-linked
11 recessive inheritance and autosomal recessive inheritance are also reported in
12 individuals with ANSD (Wang et al., 2003).

13

14 The conditions usually associated with ANSD include Charcot Marie Tooth
15 disease, Friedrich Ataxia, Rufson syndrome and Gullian Barre syndrome (Starr et al.,
16 1996) and multiple sclerosis (Cevette, Robinette, Carter, & Knops, 1995). Friedrich's
17 ataxia (FRDA) is a neurodegenerative condition restricted to the brainstem and
18 cerebellar parenchyma (Rance, 2005) and reported to be due to mutations in the FXN
19 gene (Durr et al., 1996). Histological evidence shows spared cochlear structure and
20 damage to the cochlear nerve in FRDA, hence showing the features of ANSD
21 (Spendlin, 1974).

22

23 ANSD is also reported to be associated with other syndromes such as Harding
24 disease, multiple sclerosis-like conditions which are caused by mutation of
25 11778mtDNA, (Berlin, Morlet, & Hood, 2003) and syndromes affecting

1 mitochondrial enzymes (Deltenre, Mansbach, Bozet, Clercx, & Hecox, 1997; Corley
2 & Crabbe, 1999). The isolated case of ANSD is associated with rare genetic disorders
3 such as Ehlers-Danlos syndrome (Sininger & Oba, 2001) and Stevens-Johnson
4 syndrome (Doyle, Sininger, & Starr, 1998).

5
6 Hyperbilirubinemia is known to be one of the most prevalent causative factors
7 of ANSD (Kraus et al., 1984; Rance et al., 1999). The excessive amount of bilirubin
8 usually causes damage to the CNS and peripheral nervous system, especially the
9 cochlear nucleus (Chisin, Perlman, & Sohmer, 1979; Kraus et al., 1984). Sustained
10 hypoxia is reported to be the other etiology of ANSD (Delterne et al., 1997; Rance et
11 al., 1999). In prolonged hypoxia, inner hair cells are more prone to damage than the
12 outer hair cells (Shirane & Harrison, 1987). Apart from these more prevalent
13 causative associations, ANSD can occur secondary to mitochondrial disorders (Corley
14 & Crabbe, 1999; Delterne et al., 1997), childhood measles/mumps (Prieve, Gorga, &
15 Neely, 1991), and acute lead poisoning (Starr et al., 2000). Among non-syndromic
16 late onset ANSD, the causative factors are reported to be the hormonal, genetic and
17 idiopathic conditions (Prabhu et al., 2012).

18
19 The possible site of lesion of ANSD includes inner hair cell (IHC), synapse
20 between IHC and the eighth nerve, and the eighth nerve itself (Berlin, Hood, & Rose,
21 2001; Starr et al., 1996). Other possible location of dysfunction in ANSD include
22 generation of receptor potential by IHC, transmitter release from IHC, nerve impulse
23 generation in eighth nerve dendrites, and the eighth nerve ganglion cell dysfunction
24 (Starr et al., 1998). ANSD is reported to be mainly of two types. Type I ANSD, which
25 is postsynaptic, may have an associated peripheral neuropathy, either hereditary or

1 inflammatory in origin (Butinar et al., 1999; Starr et al., 1996; Starr et al., 2001). On
2 the other hand, in type II ANSD, hearing loss is not confined to the eighth nerve but
3 lesion sites may also involve IHCs and synapse of IHC with auditory nerve (Starr et
4 al., 2001).

5
6 Starr et al. (2003) conducted a histopathological investigation of the cochlea
7 and auditory nerve in an individual with ANSD. It revealed normal organ of corti in
8 the basal turn with nearly 30% loss of outer hair cells at the apex of the cochlea. There
9 was a significant loss of ganglion cells despite normal inner hair cells throughout the
10 length of the cochlea. The proximal part of the eighth nerve showed a considerable
11 decrease in the number of auditory fibers. Furthermore, thin myelin sheath on the
12 surviving auditory nerve fibers indicated incomplete myelination. McDonald (1980)
13 reported that in demyelinating neuropathy, the conduction velocity through the nerve
14 slows down once the neural impulses pass through a demyelinated segment of the
15 axon and then regain normal speed when that segment is passed. Thus, demyelination
16 of varying degrees in different nerve fibers carry neural impulses at different
17 velocities and results in neuronal de-synchrony. Demyelination is reported to result in
18 an increase in membrane capacitance and decrease in membrane resistance, leading to
19 a delayed excitation, reduction in the velocity of action potential propagation, and an
20 increase in conduction vulnerability (McDonald & Sears, 1970; Pender & Sears,
21 1984; Rasminsky & Sears, 1972). The dys-synchronous firing of auditory neurons
22 disrupts the ABR waveform along with auditory perception which depends on
23 temporal cues (Kraus et al., 2000; Starr et al., 1991; Zeng et al., 1999, 2005).

24

1 Barman (2007) assessed the temporal processing in ANSD by means of
2 psychophysical methods and reported temporal processing deficits in individuals with
3 ANSD. Studies have also reported normal or near normal temporal integration in
4 ANSD (Zeng et al., 1999). They inferred that the perceptual deficits in ANSD are
5 mostly caused by the demyelination or axonal loss of auditory nerve. McMahon,
6 Pattuzi, Gibson, and Sanli (2008), based on their findings of EcochG, and the eABR
7 after cochlear implantation, reported the existence of pre and postsynaptic ANSD. Out
8 of the 14 subjects they tested, seven showed EcochG with delayed summing
9 potential (with or without CAEP) and superior eABR consistent with a pre-synaptic
10 lesion, whereas six subjects with normal summing and dendritic potential showed
11 poor morphology of eABR or absent eABR consistent with a postsynaptic lesion.

12
13 A presynaptic form of ANSD may be the result of mutation of OTOF gene,
14 which is important for membrane trafficking known to affect the release of
15 neurotransmitter (Rodríguez-Ballesteros et al., 2003; Roux et al., 2006; Varga et al.,
16 2003). The OTOF plays an important role in synaptic vesicle trafficking and/or fusion
17 to the plasma membrane (Yasunaga et al., 2000). Wang et al. (2005) reported OTOF
18 mutation in four out of 73 ANSD subjects (5.5%) in Chinese population. The OTOF
19 mutation in p1515t has also been found in temperature-sensitive ANSD (Varga et al.,
20 2003). In case of demyelinating neuropathy, locus of the gene is reported to be on
21 chromosome number 8 (8q24). Due to MPZ gene mutation, ANSD can have
22 peripheral as well as vestibular neuropathy (Starr et al., 2003). Further, mutation of
23 ANUAI gene is reported to be responsible for an autosomal dominant form of ANSD
24 (Kim et al., 2004). ANSD may also result from a genetic disorder affecting peripheral
25 myelin protein 22 (PMP-22) on chromosome 7p11.2 (Kovach et al., 1999).

1 Impaired perception of high frequency information in ANSD is reported to be
2 due to the limitation of the neural refractory period (Rance, 2005) whereas, the
3 impaired low frequency hearing may be due to the poor timing accuracy in
4 representing the low frequency information. Kumar and Jayaram (2006) opined that
5 the longest auditory nerve fibres which innervate the apical region are more prone to
6 get disrupted due to the longer course. Shortest fibres exit from the second half of the
7 cochlea and mediate mid frequency. Fibres which supply the basal part of cochlea
8 have length in between the former two fibres. Hence, mid frequencies are less
9 affected compared to low and high frequencies (Starr et al., 2001).

10

11 Temperature-dependent disorder of auditory function is reported in ANSD. It
12 is reported to be caused due to conduction block rather than disruption of timing
13 (Marsh, 2002). This kind of pathology is consistent with demyelinating neuropathies
14 (Starr et al., 1998). Starr et al. (1998) recorded nerve conduction velocity on sural,
15 peroneal and median nerve on both sides at normal body temperature and also at
16 39°C. The results showed a normal velocity at increased temperature, indicating the
17 absence of other neuropathic conditions. Authors opined that maintenance of nerve
18 conduction in the paranodal region of demyelinated axons is temperature dependent.
19 With slight increment in temperature, the voltage-gated Na⁺ channels become
20 inactivated more rapidly compared to normal temperature, resulting in failure of
21 impulse transmission. Moreover, authors suspect both conduction block and deafness
22 with elevated body temperature in individuals with ANSD.

23

24 In persons with ANSD, ABR in the affected ear is either absent or abnormal
25 because of the paucity of neural element or disruption of temporal integrity. In the

1 former case, as in the case of anti-neoplastic drugs (carboplatin), there is selective
2 damage of IHCs and hence, volume conducted neural activity is too low to be
3 detected by scalp electrode (Rance, 2005). In the latter case, ABR is absent or grossly
4 abnormal due to compromised neural synchrony (Berlin et al., 2001). The ABR peaks
5 represent the synchronous spike discharge at the neural tracts whereas the cortical
6 potentials correspond to the summation of excitatory postsynaptic potentials. The unit
7 contribution of ABR is biphasic and of shorter duration, and hence it tends to cancel
8 when the response occurs at a difference of fraction of milliseconds in individuals
9 with ANSD (Kraus et al., 2000).

10

11 **2.4 Psychoacoustic Abilities in ANSD**

12 The subjects with ANSD are reported to show marked deficits in their ability
13 to resolve rapid stimulus changes (Michalewski et al., 2005; Starr et al., 1991; Zeng et
14 al., 1999, 2005). The studies (Zeng et al., 2005; Zeng et al., 1999, 2001) that
15 measured gap detection thresholds (GDT) have shown that normal hearing individuals
16 could perceive silent periods of less than 5 ms within a continuous signal, whereas
17 individuals with ANSD required a gap of 20 ms or more. This inability to perceive
18 small gaps in speech signal was reported to affect the perception of brief vowel
19 feature such as third formant onset frequency. On similar lines, discrimination of
20 manner of articulation of consonants which is based on the small difference in voice
21 onset time is reported to be affected secondary to reduced GDT in ANSD.

22

23 Kumar and Jayaram (2005) estimated the temporal modulation transfer
24 function (TMTF) in normal hearing individuals and individuals with ANSD. They
25 reported that individuals with ANSD required significantly higher modulation depth

1 to detect the modulations compared to normal hearing individuals. Further, they found
2 that at higher modulation frequencies, individuals with ANSD were unable to detect
3 the modulation even with 100% modulation depth. Similarly, studies have reported
4 that individuals with ANSD experience difficulty to follow faster as well as slow (<10
5 Hz) amplitude envelope changes over time (Rance et al., 2004; Zeng et al., 1999,
6 2005). It has been reported that individuals with ANSD perform poorly on tasks
7 involving timing cues and they found a correlation between temporal processing
8 abnormalities and speech perception abilities. The impaired temporal processing is
9 reported to hamper the ability to effectively handle the dynamic nature of speech
10 signal causing speech perception deficits in ANSD.

11

12 Psychophysical evidence has shown that subjects with ANSD have more
13 problems with simultaneous and non-simultaneous masking compared to normal
14 listeners (Kraus et al., 2000; Vinay & Moore, 2007; Zeng et al., 2005). Kraus et al.
15 (2000) and Zeng et al. (2005) studied temporal processing in individuals with ANSD
16 using forward and backward masking experiments. Results showed that the
17 perception of short duration signals was affected even with masker to signal delays of
18 100 ms whereas normal hearing subjects showed limited masking effects beyond 10
19 to 20 ms of the masker. When tested on masking level difference, individuals with
20 ANSD had little or no masking release (Berlin et al., 1993; Starr et al., 1996). This
21 was inferred as the inability to combine the neural code from the two ears in ANSD.
22 Poor backward masking thresholds were seen in ANSD, indicating that they are
23 poorer than normal at separating noise and sounds in time. Kraus et al. (2000) found
24 that persons with ANSD had poorer ability to separate a brief tone from a noise which
25 is remote from the frequency of the tone, making them a poor listener in the noisy

1 environment. They were also found to show abnormal temporal measures such as
2 GDT, TMTF (Rance et al., 2004), wider temporal window in forward-backward
3 masking (Kraus et al., 2000; Zeng et al., 2005), and abnormal binaural processing
4 (Zeng et al., 1999). The authors also opined that, in ANSD, location-based binaural
5 timing cues was poorly perceived, but the perception of inter-aural intensity
6 difference required for the judgment of lateralization was preserved.

7

8 Kumar and Jayaram (2011) examined the effect of lengthened transition
9 duration on speech perception and Just Noticeable Difference (JND) in transition
10 duration of stop consonants in individuals with ANSD. Results revealed a significant
11 difference in JND between normal and ANSD groups. Improvement in the perception
12 of place of articulation of phonemes was noted with lengthened transition duration of
13 the stimuli. The results of sequential information analysis (SINFA) showed that there
14 was better transmission of the place information compared to voicing information
15 when transition duration was increased. It was also noted that JND of individuals with
16 ANSD was almost 3 to 4 times longer than that of normal hearing individuals
17 indicating impaired temporal processing in ANSD. The authors hypothesized that
18 longer transition duration would have reduced the modulation frequency without
19 affecting modulation depth or overall spectrogram of the signal. Moreover,
20 individuals with ANSD have difficulty following faster modulation. Hence, the
21 decrease in modulation frequency (by lengthening the transition duration) was
22 reported to augment their speech perception as the modulation detection is better at
23 lower frequency compared to higher modulation frequencies. Other studies also
24 reported JND of individuals with ANSD to be approximately 4.5 times longer than
25 normal hearing individuals (Starr et al., 1991; Zeng et al., 2001).

1 For the steady state pure-tone of 4 kHz or higher, frequency discrimination is
2 primarily cued by the place of excitation on the basilar membrane (Moore, 1973;
3 2008). On the contrary, frequencies less than 4 kHz are discriminated based on the
4 temporal cues. Zeng et al. (2001) found abnormal frequency discrimination at low
5 frequencies while the discrimination was normal at higher frequencies. Rance et al.
6 (2004) found a strong direct relationship between difference limen of frequency and
7 speech perception in ANSD. Abdala, Sininger, and Starr (2000) generated DPOAE
8 suppression tuning curves in individuals with ANSD and their controls, by
9 systematically changing the level and frequency of the ipsilateral noise. The
10 suppression tuning curve thus obtained in ANSD was similar to normal, suggesting
11 normal cochlear level frequency selectivity in individuals with ANSD. Hence, it can
12 be inferred that individuals with ANSD exhibit normal frequency resolution and
13 intensity discrimination, but impaired temporal resolution. On the contrary,
14 individuals with cochlear hearing loss demonstrate normal temporal resolution and
15 impaired frequency resolution (Hassan, 2011).

16

17 **2.5 Speech Perception in ANSD**

18 The cardinal feature of ANSD is the poor speech perception that does not
19 relate to their degree of hearing loss (Starr et al., 1996; Starr et al., 2000; Zeng et al.,
20 2001). The poor speech perception in ANSD is known to be due to the impaired
21 ability to process the dynamic cues of speech. Earlier studies have shown that the
22 disrupted neural synchrony in individuals with ANSD impairs their ability to use
23 envelope cues as well as spectral cues of speech (Rance, 2005; Zeng et al., 1999).

24

1 The speech perception in ANSD is reported to further deteriorate in adverse
2 listening conditions such as in the presence of background noise (Kraus et al., 2000;
3 Shallop, 2002; Starr et al., 1998). The drastic reduction in speech perception ability in
4 the presence of noise is known to be due to the "line busy effect" in which the noise
5 activates the auditory nerves and reduces the response to other signals (Derbyshire &
6 Davis, 1935; Powers, Salvi, Wang, Spongr, & Qiu, 1995; Spreng, 2000). The auditory
7 perceptual deficits in subjects with ANSD are reported to be mainly due to the
8 disruption of temporal cues (Kraus et al., 2000; Starr et al., 1991) and are found to
9 correlate with their abnormal temporal and masking functions (Vinay & Moore, 2007;
10 Zeng et al., 1999).

11
12 In individuals with ANSD, fricatives are perceived better compared to the
13 other consonant groups due to the preserved high frequency discrimination (Hassan,
14 2011). The perception of nasal consonants is known to be affected in them which are
15 attributed to their impaired ability to use low frequency spectral cues (Narne &
16 Vanaja, 2008a). Narne and Vanaja (2008a) also reported place errors for stops as a
17 major concern in ANSD. This was suggested to be due to the impairment in utilizing
18 the burst amplitude and formant transition that contribute mainly to the perception of
19 stop consonants. Kumar and Jayaram (2011, 2013) also reported impaired perception
20 of voice onset time, burst and formant transitions, resulting in poor perception of
21 stops. They attributed it to the impaired temporal processing in individuals with
22 ANSD. Zeng et al. (1999) stated that individuals with ANSD have impaired
23 perception of fast modulation of speech. This results in the poor perception of burst
24 duration and transition duration which are crucial in the perception of stops.

25

1 Synchrony at the level of eighth nerve and brainstem that play a major role in
2 speech perception is affected in individuals with ANSD. In addition, they fail to make
3 use of the neural mechanism that represents the temporal fine structure of the
4 stimulus, which is important for speech perception in noise (Kraus et al., 2000).
5 Difficulty understanding speech in background noise has been attributed to the
6 impaired ability to process the envelope of the signal (Houtgast & Steeneken, 1985).
7 The perception of auditory signals during simultaneous masking is found to be more
8 affected in ANSD compared to individuals with normal hearing (Kraus et al., 2000;
9 Zeng et al., 2005). Excessive masking effect that is 10-20 dB higher than normal has
10 been reported in this population (Kraus et al., 2000). The findings also suggested that
11 some form of central masking mechanism exists in ears with normal OAEs, as is the
12 case in ANSD. Overall, the forward and backward masking experiments showed that
13 a short signal with proximity of 100 ms to the masker is difficult to perceive in
14 individuals with ANSD. This is likely to deleteriously affect perception of the running
15 speech.

16
17 Typically in ANSD, speech perception is poorer than that seen in cochlear
18 hearing loss. But not all individuals with ANSD show unusually poor speech
19 identification scores in quiet. This may be due to the fact that in some individuals with
20 ANSD, the disease process may be less severe (Rance, 2005). Some of the factors
21 contributing to poor speech perception in ANSD include reduced ability to follow fast
22 and slow temporal modulation as evidenced by TMTF, reduced gap detection and
23 affected frequency discrimination at low frequency (Rance et al., 2004; Starr et al.,
24 1996). Rance et al. (2004) also reported a strong correlation between speech
25 perception and temporal modulation in ANSD. Shanon, Zeng, Kamath, Wygonski,

1 and Ekelid (1995) reported that the reduced ability of individuals with ANSD to
2 perceive cues contained in the temporal envelope results in poor speech in noise
3 perception. They also found that the peak sensitivity for modulation detection in
4 ANSD was -3.4dB for individuals with SIS less than 30%, and -14.3dB for
5 individuals with SIS of more than 30%.

6
7 Drullman, Festen, and Plomp (1994) studied speech perception in normal
8 hearing individuals by reducing the modulation depth, degrading the amplitude
9 modulation and flattening the spectral change in the auditory stimulus. It was found
10 that individuals with normal hearing experience difficulty in extracting the salient
11 cues for consonant-vowel distinction and spectral contrast. This was comparable to
12 perceptual deficits seen in ANSD. Narne and Vanaja (2008a) reported that in
13 individuals with ANSD, voicing cues are poorly perceived compared to place or
14 manner of articulation. Gnanateja and Barman (2011) studied the perception of place,
15 manner, and voicing in individuals with cochlear hearing loss and ANSD and reported
16 that all the three cues are poorly perceived in ANSD compared to those with cochlear
17 hearing loss. They also reported that in individuals with ANSD, manner cues were
18 perceived better than place and voicing cues. Rance and Barker (2008) compared the
19 perception of vowels, diphthongs and semivowels in ANSD and cochlear hearing
20 loss. Their results revealed that perception of vowels was similar in both the groups,
21 whereas the perception of diphthongs and semivowels were poorer in persons with
22 ANSD compared to cochlear loss.

23
24 Prabhu, Avilala and Barman (2011) found no difference in the perception of
25 unfiltered and low pass filtered speech with a cutoff frequency of 1700Hz in

1 individuals with ANSD. It may be attributed to the low frequency hearing loss in
2 ANSD, caused by poor phase locking of low frequency information by Type I fibers.
3 The authors opined that greater loss at low frequency leads to increased temporal
4 asynchrony and poor perception of low-pass filtered speech in ANSD. They
5 concluded that individuals with ANSD may not make use of phase locking cues due
6 to neural dys-synchrony but make use of high frequency information for
7 understanding speech.

8

9 **2.6 Relationship between Speech Perception and Production**

10 Speech perception and speech production skills share a close relationship
11 (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967). Auditory feedback of
12 one's own speech helps to map speech sounds accurately in relation to the articulatory
13 activity, whereas listening to the speech of others primarily help in establishing and
14 storing the meaning of the sounds (Fowler & Saltzman, 1993). The auditory feedback
15 is essential to monitor and maintain a fairly intelligible speech. Given the intimate
16 relationship between hearing and speech, language, and communication, hearing loss
17 in early years of life can have major detrimental effects on these areas of development
18 (Culbertson & Kricos, 2002; Dunn & Newton, 1986; Hudgins & Numbers, 1942;
19 Smith, 1982). These effects are observed as delayed or deviant language skills and
20 defective speech in terms of articulation, fluency and voice. Apart from the segmental
21 aspects, the suprasegmental features of speech are also found to be affected. Thus, a
22 defective auditory feedback secondary to hearing loss is considered to be the cause of
23 poor segmental and suprasegmental speech characteristics (Binnie et al., 1982; Cowie,
24 et al., 1982; Elman, 1981; Kirchner & Suzuki, 1968; Penn, 1955; Ramsden, 1981;
25 Zimmermann & Rettaliata, 1981).

1 Several articulatory errors are reported in individuals with cochlear hearing
2 loss. Deletion of initial and final consonants, consonant cluster errors, voicing and
3 nasality errors, consonant substitutions, and vowel distortions are few of the common
4 errors observed in children with hearing impairment (Angelocci, Kopp, & Holbrook,
5 1964; Boone, 1966; Geffner, 1980; Hudgins & Numbers, 1942; Markides, 1970;
6 Nober, 1967). A reduced vowel triangle space or phonological space and more
7 centralized vowel production is reported in individuals with hearing loss when
8 compared to those with normal hearing skills (Angelocci et al., 1964; Monsen, 1976).
9 Boone (1966) reported a lowered second formant frequency in children with hearing
10 impairment. In addition to the misarticulated vowels, consonants are also found to be
11 equally affected. Markides (1970) reported an error rate of 26% to 72% on consonant
12 production in children with partial hearing to complete hearing loss. The most
13 commonly misarticulated sounds were /s/, /f/ and /n/. Geffner (1980) reports more
14 errors on consonants than vowels in these children. The overall speech intelligibility
15 was also found to have a significant correlation with the severity of hearing loss
16 (Boothroyd, 1984; Perkell et al., 1997; Smith, 1982).

17

18 According to the acoustic theory of speech production, speech signal is
19 processed and represented as an internal map which may get distorted if the acoustic
20 patterns are not received adequately during the input process. Input process could be
21 assumed to be compromised secondary to hearing loss, which in turn causes an
22 incorrect mapping resulting in distorted or deleted speech sounds during production
23 (Stevens, 2002). This impaired hearing ability correlates well with the compromised
24 speech intelligibility (Kuhl, 1981; Stevens, 2002). For example, children with mild to
25 moderate degrees of loss develop fairly intelligible speech, but still make articulatory

1 errors while producing affricates, fricatives and blends (Elfenbein, Hardin-Jones, &
2 Davis, 1994). On the other hand, children with severe to profound loss have severely
3 compromised speech intelligibility as they have articulatory difficulties with
4 consonants, vowels and diphthongs, as well as abnormal voice (Culbertson & Kricos,
5 2002).

6
7 Along with the segmental errors discussed above, the suprasegmental features
8 of speech are also reported to be affected in the speech of hearing impaired
9 individuals especially those with severe to profound loss (Dunn & Newton, 1986).
10 The typical suprasegmental errors observed include slow speech rate, slow
11 articulatory transitions, poor breath control, inappropriate stress patterns, and poor
12 resonance.

13
14 The individuals with hearing loss are usually considered to have flat and
15 monotonous intonation contour (Hood & Dixon, 1969). Some investigators have
16 reported a restricted or reduced range of pitch variations in these individuals (Hood,
17 1966; Voelker, 1935), while few others report intonation variations in the form of
18 excessive and inappropriate changes in fundamental frequency (Monsen, 1979; Smith,
19 1975; Stevens, Nickerson, & Rollins, 1978). Angelocci et al., (1964) and Martony
20 (1968) attribute these errors to limited/no control of voice frequency (particularly for
21 vowels of long duration) in these individuals. An attempt to quantitatively classify the
22 intonation patterns in children with hearing impairment was made by Monsen (1979)
23 who reported four different patterns including falling, short-falling, falling-flat and a
24 changing contour. According to him, the type of contour appeared to be an important
25 characteristic in separating the better from the poorer hearing-impaired speakers.

1 Susman and Hernandez (1979) studied intonation control in ten hearing impaired
2 subjects. Subjects were instructed to read three sentence pairs, each with a declarative
3 and interrogative form. The results revealed terminal fall in mean F0 for both the
4 sentence types. Indira (1981) examined the intonation patterns of normal hearing and
5 hearing impaired subjects using a story reading task. The findings revealed a
6 difference in the rise and fall patterns across the two groups. The hearing impaired
7 group had restricted pitch variations when compared to normal subjects. It was also
8 found that the duration of the speech segment was longer for the hearing impaired
9 subjects. This was also considered to be the reason for minimal changes in the
10 intonation patterns observed in subjects with hearing impairment. In contrast, sharp
11 changes in intonation patterns of the normal hearing subjects were observed.

12

13 The studies on stress indicate that children with hearing impairment
14 demonstrate marked deficits in the production of stress. It has been found that the
15 durations of unstressed and stressed syllables produced by these children do not differ
16 significantly (Angelocci, 1962; Nickerson, 1975) giving an impression that
17 individuals with hearing impairment produce only stressed speech (Boone, 1966).
18 McGarr and Osberger (1978) report production of equal stress on each word followed
19 by equal pause as the most common prosodic error in individuals with hearing
20 impairment while saying a sentence. Another study investigating the production of
21 stress in Tamil speaking hearing impaired children reported improved stress
22 production with increase in age (Sarumathi & Savithri, 1993).

23

24 Savithri, Johnsi, and Agarwal (2007) studied speech rhythm in hearing
25 impaired children using picture description and story narration tasks. They used

1 pairwise variability index (PVI) to assess rhythm. The findings revealed a significant
2 difference between groups on rPVI (intervocalic) and nPVI (vocalic) values. Both
3 rPVI and nPVI were higher in children with hearing impairment when compared to
4 typically developing normal hearing children indicating the significant difficulties in
5 them to perceive and process normal rhythmic patterns.

6
7 Although the impact of hearing loss is more severe in the early years of life,
8 several perceptual studies suggest that long-term acquired loss might result in flat,
9 unmodulated and dysprosodic voice along with deterioration of segmental speech
10 (Binnie et al., 1982; Cowie et al., 1982; Elman, 1981; Kirchner & Suzuki, 1968; Penn,
11 1955; Ramsden, 1981). Ramsden (1981) reported deterioration of speech secondary to
12 long-term hearing loss, emphasizing the role of auditory information in maintenance
13 of normal speech. This deterioration of speech as a sequel of long term auditory
14 deprivation is attributed to the overlearned motor patterns (errors in articulation or
15 production without the knowledge of errors occurring) which take place after several
16 instances of production exceeding the standard range of variability (Zimmerman &
17 Rettaliata, 1981). Altered or impaired auditory feedback could also result in changes
18 in individual sound production leading to misarticulation (Houde & Jordan, 2002).
19 These findings are in consensus with other acoustic studies, which report higher
20 speaking fundamental frequency (Leder, Spitzer, & Kirchner, 1987), greater intensity
21 (Leder et al., 1987b) and lower speaking rate (Leder et al., 1987a) than that of age-
22 matched, normal hearing subjects. Longer sentence duration is another common
23 finding reported in individuals with post-lingual loss (Kirk & Edgerton, 1983; Lane &
24 Webster, 1991). This prolonged sentence duration is a cumulative effect of longer
25 syllables (Lane & Webster, 1991; Leder et al., 1987), pause duration (Lane &

1 Webster, 1991), and vowel duration (Waldstein, 1990) observed in this population. In
2 summary, these findings emphasize the role of feedback in speech production and
3 support the closed loop models.

4
5 In contrast to the studies discussed above, Leder and Spitzer (1990) and Goehl
6 and Kaufman (1984) reported no significant deterioration of speech sound production
7 in their subjects with long term hearing loss. These findings suggest that mature
8 phonemic motor patterns are robust and do not rely on auditory feedback, reflected
9 through good speech intelligibility seen in individuals with profound postlingual
10 hearing loss. These researchers and their findings support the open loop speech motor
11 control system, which suggest that sensory feedback is not necessary for the execution
12 of normal speech and posits that the speech movements are preprogrammed.
13 Therefore, the effector units (speech musculature) in open loop models do not rely on
14 sensory information to perform accurate movements but rather play out a
15 predetermined neural code (Matthies, Svirsky, Perkell, & Lane, 1996).

16
17 In the context of Indian studies, Grover (1998) reported a slow rate of speech
18 in individuals with hearing impairment. Speed of transition was also reported to be
19 significantly reduced in individuals with hearing impairment compared to individuals
20 with normal hearing (Jayaradha, 2001). The slow transition rate was attributed to
21 sluggish tongue movements and imprecise articulatory movements. The extent of
22 speech deterioration is determined by the age of onset of hearing loss. In other words,
23 earlier the onset, greater is the impact of hearing loss on speech intelligibility (Binnie
24 et al., 1982; Cowie et al., 1982).

25

1 An insight to the literature on ANSD reveals that these individuals have more
2 severe deficits in speech processing and perception as proven by several
3 psychophysical and perceptual studies. Based on the aforementioned literature,
4 individuals with long standing cochlear loss are prone to speech deterioration
5 secondary to prolonged auditory deficits. Thus, it can be speculated that individuals
6 with ANSD who have poorer speech identification than individuals with cochlear loss
7 will exhibit speech production deficits. Some support can be drawn for this
8 speculation from the study by Rance, Barker, Sarant, and Ching (2007) reporting
9 delayed spoken language development in children with ANSD compared to children
10 with normal hearing.

11
12 Dayal and Maruthy (2009) made one of the first attempts to investigate the
13 speech perception characteristics in adults with long term ANSD. They analyzed both
14 perceptual and acoustic characteristics of the speech of individuals with ANSD.
15 Perceptual rating was done for all the parameters (voice, articulation, prosody, rate of
16 speech & overall intelligibility) and compared between individuals with ANSD and
17 normal hearing. Similarly acoustic analysis comparing the temporal parameters of
18 speech (word duration, voice onset time, burst duration, transition duration and speed
19 of transition, preceding and following vowel duration) between the two groups was
20 carried out. The findings suggested perceptually abnormal speech on all the
21 parameters, although prosody was found to be maximally affected. The overall speech
22 intelligibility was also found to be poor and had a significant correlation with their
23 speech identification scores. It supports the notion that the auditory feedback is
24 essential for normal speech production and long standing auditory deprivation could
25 have detrimental effects on speech. The segmental aspects of speech were found to be

1 relatively better than prosody. While the former involves other sensory cues/feedback
2 such as tactile and visual, the latter depends completely on auditory feedback making
3 it more prone to disruption. Further, the acoustic analysis revealed significant
4 differences for all the temporal parameters of speech between individuals with ANSD
5 and normal hearing. A good correlation was also established between the perceptual
6 and acoustic characteristics of speech of individuals with ANSD. These findings are
7 in consensus with earlier studies (Houde & Jordan, 2002; Binnie et al., 1982;
8 Zimmerman & Rettaliata, 1981) supporting the closed loop models. The temporal
9 parameters measured provide important place and manner of articulation cues
10 (Kumar, 2006), thus the increased duration was attributed to be a form of
11 compensatory production or modifications made by individuals with ANSD to avail
12 better feedback on place and manner of articulation.

13

14 However, it was only a preliminary attempt and did not include detailed
15 evaluation of various segmental and suprasegmental aspects of speech. Therefore, the
16 present study aimed to explore and understand the segmental and suprasegmental
17 characteristics of speech in individuals with ANSD.

18

CHAPTER 3

METHODS

The study aimed to assess the speech production characteristics of individuals with auditory neuropathy spectrum disorder (ANSD) and correlate these with their auditory profile. The study used a standard group comparison research design and was executed in two phases. Phase I comprised of preparation and compilation of test stimuli, while Phase II involved data collection and analyses.

3.1 Participants

The study included two groups of participants; individuals with ANSD and individuals with normal auditory abilities (NAA). The ANSD group had 30 participants in the age range of 18 to 40 years (Mean age: 26.03 years) and had visited the department of Audiology, All India Institute of Speech and Hearing (AIISH), Mysore, once earlier. They were contacted through letters and calls and were requested to visit AIISH for a follow-up evaluation. ANSD was diagnosed by qualified audiologists based on the criteria recommended by Starr et al. (2000). All of them had sensorineural hearing loss, and the degree of hearing loss ranged from minimal to profound hearing loss.

The speech identification scores of participants with ANSD in quiet ranged from 0% to 96% in the two ears (Right ear: Mean = 44.82%, SD = 34.80 and Left ear: Mean = 43.17%, SD = 34.87). The minimum duration of ANSD in these participants was five years, and the maximum duration was up to 20 years. All of them had acquired ANSD post-lingually.

1 The presence of external or middle ear pathology was ruled out by an
2 experienced otologist, and normal middle ear functioning was further confirmed with
3 immittance evaluation. They had normal outer hair cell function revealed by the
4 presence of transient otoacoustic emissions (OAEs) (amplitude > 6 dB SPL) or
5 cochlear microphonics. They had absent ABR indicative of neuronal dys-synchrony.
6 All the participants had also undergone neurological examination to rule out the
7 presence of space occupying lesion. Neurological evaluation included clinical
8 examination, CT scan and/or MRI as recommended by the neurologist.

9

10 The NAA group included 30 participants in the same age range as ANSD
11 group, i.e., 18 to 40 years (Mean age: 21.9 years). The participants in the NAA group
12 had normal hearing sensitivity (less than 15 dB HL at octave frequencies from 250 Hz
13 to 8000 Hz). They were all screened using WHO ten questions disabilities screening
14 checklist (cited in Singhi, Kumar, Malhi, & Kumar, 2007) to rule out history of any
15 neurological, speech-language and hearing disorders. All the participants in this group
16 had normal OAEs and normal ABRs. Speech identification scores were within normal
17 limits in both quiet and in the presence of noise at 0 dB SNR. These individuals
18 reported no past/present history of any neurological or otologic complaints.

19

20 Participants in both the groups were native speakers of Kannada language. All
21 the participants had a minimum education of 10th standard and could comprehend,
22 speak and read Kannada proficiently. All of them resided in and around Mysore
23 district. Based on the developmental history, all the participants had normal speech
24 and language milestones. All the participants were subjected to an oral mechanism
25 examination to rule out the presence of any structural abnormalities. A written

1 informed consent regarding willingness to participate in the study was obtained from
2 all the participants. The methods adopted were approved by the AIISH ethical
3 committee for bio-behavioral research in human subjects (Basavaraj & Venkatesan,
4 2009).

5

6 **3.2 Test Stimuli**

7 The speech production characteristics were measured in terms of segmental
8 and suprasegmental features. The details of stimuli used to assess the same are given
9 below (refer to Appendix I for stimuli).

10

11 ***3.2.1 Stimuli to assess segmental characteristics***

12 a) *Vowels:* Three short vowels /a/, /ɪ/ and /ʊ/ of Kannada language were
13 considered. A list of nine words was prepared to assess the segmental features
14 of these vowels. There were three words to assess each of the vowels.

15 b) *Plosives:* Eight plosives including four voiced (/g/, /d/, /ɖ/, /b/) and four
16 unvoiced (/k/, /t/, /t̪/, /p/) phonemes were considered as targets. A list of 16
17 words was prepared with each of the target plosives in initial and medial word
18 position.

19 c) *Fricatives:* Three fricatives /s/, /ʃ/ and /f/ were considered. A list of six words
20 was prepared in which the three fricatives occurred in initial and medial
21 positions in one word each.

22 d) *Voice:* Segmental analysis also included subjective and objective assessment
23 of voice. A phonation sample of vowel /a/ and a reading sample obtained
24 using **standard Kannada passage (Shashidhara, 1984)** developed at AIISH,
25 Mysore, served as the voice samples. Subjective assessment was carried out

1 using Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V)
2 (American Speech-Language and Hearing Association, 2002) scale, while for
3 the objective analysis, the recorded samples were analyzed using Vagmi
4 Diagnostics software version 9.1 (Voice & Speech Systems, 2018).

5

6 **3.2.2 Stimuli to assess suprasegmental characteristics**

- 7 *a) Emphasis:* Ten adjective-noun phrases adopted from Ananthi and Savithri
8 (2002) were used to assess emphasis. In each of the phrases, the target word
9 (i.e., the adjective) to be emphasized was highlighted (bold and underlined).
- 10 *b) Rhythm:* Kannada sentences were adopted from Santosh and Sahana (2012) and
11 were given to five experienced Speech-Language Pathologists (SLPs) for rating
12 based on their meaningfulness and grammaticality. They were instructed to rate
13 the stimuli on a 2-point rating scale, where 1 indicated ‘appropriate’ and 0
14 indicated ‘inappropriate’. The SLPs were also asked to suggest the
15 correction/modification for the inappropriate sentences. The suggestions were
16 incorporated, and the five most appropriate sentences in each of the two
17 categories (interrogatives & declaratives) were included as the final set of
18 stimuli to assess rhythm.
- 19 *c) Intonation:* Ten sentences (5 declaratives & 5 interrogatives) adopted for the
20 assessment of rhythm were used as stimuli to assess intonation.

21

22 **3.3 Instrumentation**

23 Following instruments were used for the assessment in the present study:

- 1 ▪ A calibrated two channel diagnostic audiometer (Audiostar pro) was used for the
2 estimation of pure tone thresholds, speech recognition threshold, speech
3 identification scores, speech in noise scores, and gap detection thresholds
- 4 ▪ A calibrated tympanometer (GSI tymptstar) was used to assess middle ear function
- 5 ▪ ILO (version 6) Otodynamics audiology system was used to record otoacoustic
6 emissions
- 7 ▪ Biologic Navigator Pro (version 7.2.1) AEP system was used to record auditory
8 brainstem and late latency responses
- 9 ▪ Sony digital voice recorder- IC recorder ICD-UX81 was used to record the speech
10 samples
- 11 ▪ A computer with Praat software (version 5.1.2.9) (Boersma & Weenink, 2011)
12 was used for acoustic analysis and
- 13 ▪ MATLAB (MathWorks Inc. Natick, USA, R2010a) installed in the same
14 computer was used for administering gap detection test and for analyzing rhythm.

15

16 **3.4 Test Procedure**

17 Each participant was individually tested in one or more sessions to assess their
18 audiological and speech production characteristics.

19

20 **3.4.1 Audiological Profiling**

21 The participants were profiled in terms of their pure-tone thresholds,
22 tympanometry, otoacoustic emissions, auditory evoked potentials, speech perception
23 and gap detection thresholds (Refer to Appendix II).

24

1 *Pure-tone thresholds* were estimated using modified Hughson and Westlake
2 procedure. Pure-tone thresholds were estimated at octave frequencies between 250 Hz
3 and 8000 Hz in air conduction, and between 250 Hz and 4000 Hz in bone conduction
4 mode. *Speech recognition thresholds* were obtained monaurally in the two ears using
5 paired-words in Kannada, developed in the department of Audiology, AIISH, Mysore.
6 *Speech identification score* was obtained monaurally at Most Comfortable Loudness
7 levels for phonetically balanced words developed by Yathiraj and Vijayalakshmi
8 (2005). *Speech perception in noise (SPIN)* was assessed using the word list given by
9 Manjula, Antony, Kumar, and Geetha (2015). The presentation level was 40 dB SL
10 and the SPIN was tested at 2 SNRs (0 dB & 10 dB).

11

12 *Tympanogram and acoustic reflex thresholds* were measured using 226 Hz
13 probe tone using the standardized procedure. A calibrated GSI-Tympstar, version-2
14 middle ear analyzer was used for the purpose. Ipsilateral and contralateral acoustic
15 reflex thresholds were measured at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz in the two
16 ears.

17

18 *Gap Detection Threshold (GDT)* was measured using noise bursts of 750 ms
19 duration. The onset and offset of the noise bursts was linearly ramped for 20 ms. The
20 gaps/silences were introduced at the temporal center of the noise bursts. A three
21 interval three alternate forced choice procedure was used to estimate the minimum
22 gap that the participant can detect. The noise bursts without gap served as a reference
23 while the noise bursts with gap served as the target stimuli. Every trial involved the
24 presentation of the three noise bursts in which two were the standard stimuli and one
25 was the variable or target stimulus. The task of the participants was to identify the

1 noise bursts in which a gap was present. The order of presentation of reference and
2 target stimuli was randomized. The duration of the gap was varied in a two-down one-
3 up procedure to estimate the 70.7% point on the psychometric function. A total of 12
4 reversals were obtained. Initial gap size was 20ms which was then altered in 5ms
5 step sizes for the first two reversals. The subsequent reversals were then altered in
6 steps of 1ms gap size. The test was performed through the MLP tool box implemented
7 in MATLAB by Grassi and Soranzo (2009). The average of the last eight reversals
8 was considered for calculating the gap detection threshold.

9

10 *Auditory brainstem responses (ABRs) and auditory late latency responses*
11 *(ALLRs)* were recorded using Biologic Navigator evoked potential system (version
12 7.2.1). Each recording was repeated to ensure reproducibility of the responses. The
13 stimulus and acquisition parameters used to record ABR and LLR are given in Table
14 3.1.

15

16 *Transient evoked otoacoustic emissions (TEOAEs)* were measured for clicks at
17 80 dB +/-5 dB peak SPL using ILO V6 Echoport (version 6.40.0.0) equipment.
18 TEOAEs were considered to be present if the waveform reproducibility was more
19 than 75% and the overall amplitude was more than 6 dB in at least 3 consecutive
20 frequencies of measurement.

21

22

23

24

25

1 Table 3.1

2 *Stimulus and acquisition parameters used to record click evoked ABR and ALLR*

Stimulus Parameters			Acquisition Parameters		
	ABR	LLR		ABR	LLR
Stimulus	Clicks	500 Hz Tone Burst & /da/ of 40ms	Filter	100- 3000 Hz	0.1- 100Hz
Polarity	Rarefaction	Alternating	Window	10.6 ms	533 ms
Level	90 dB nHL	80 dB nHL	Montage	Cz-M1 and Cz- M2, Nasion-ground	
Duration	100 μ s	60 ms			
Number of sweeps	2000	500			
Rate	11.1/s and 90.1/s	1.1/s			
Artifact rejection	+/- 22 μ V	+/- 30 μ V			
Transducer	ER 3A Inserts ear phones				

3

4 **3.4.2 Profiling of Speech Production**

5 *Recording of Speech Samples:* The speech samples of the participants were recorded
6 in a sound treated room as per the ANSI standards. The recording was done using a
7 Sony digital voice recorder (IC recorder ICD-UX81) with an omnidirectional
8 microphone with a distance of six inches between the microphone and the speaker's
9 mouth. The recorded files were transferred to a personal laptop in .wav format and
10 were further analyzed using Praat software at a sampling frequency of 44,100 Hz.

11

12 All the participants were instructed to produce three trials per target item and
13 the best out of the three was selected for analysis. The words used in segmental
14 analysis i.e., target words for vowels, plosives and fricatives were embedded in a

1 common carrier phrase. Participants were instructed to embed the target word into the
2 carrier phrase “/nāno īga (Target word) hēlōttēne/” and say the complete sentence.
3 Example, “/nāno īga kəbbu hēlōttēne/”.

4

5 **3.5 Analyses**

6 **3.5.1 Analysis of the Audiological Characteristics**

7 The audiogram of the participants was analyzed in terms of pure tone average.
8 If the pure tone thresholds indicated the presence of hearing loss, the degree of
9 hearing loss, type of hearing loss and the configuration of audiogram were interpreted.
10 The speech identification in quiet and noise was analyzed in terms of percentage of
11 correct identification.

12

13 The electrophysiological recordings were visually inspected by two
14 audiologists with rich experience in the domain. ABRs were analyzed in terms of the
15 presence or absence of wave I, III and V. In instances of presence of these waves, the
16 latency and amplitude of the waves were noted down to infer the presence or absence
17 of space occupying or diffuse lesions of the brainstem. ALLR recordings, when
18 present, were also visually inspected to locate P1, N1, P2 and N2 waves. The latency
19 and amplitude of the waves present were noted down.

20

21 **3.5.2 Analysis of Speech Samples**

22 The speech samples of the participants were subjected to both perceptual and
23 acoustical analyses. The recorded samples of each participant were acoustically
24 analyzed to obtain both spectral and temporal parameters. Praat software was used to
25 analyze the acoustic characteristics of vowels and consonants (plosives and
26 fricatives), emphasis and pitch variations in intonation.

1 **3.5.2.1 Segmental characteristics**

2 The acoustic analysis for vowels and consonants was carried out using Praat
 3 software, wherein the segment representing the target phoneme was selected and the
 4 acoustic parameters were derived / computed. The parameters are listed and the
 5 method of measurement is described in Table 3.2. A visual representation of these
 6 measures is provided as Figure 1.

7

8 Table 3.2

9 *Summary of acoustic parameters measured*

Target sound	Acoustic Parameters	Method of measure
Vowels		
Short vowels (/a/, /ɪ/, /ʊ/)	Fundamental Frequency (F ₀) Formants (F ₁ , F ₂) Formant bandwidths (F ₁ BW, F ₂ BW)	Steady state of the target vowel was selected and the parameters were extracted by Praat software.
	Vowel duration	The vowel duration (in milliseconds) was measured from the onset of the steady state of the vowel to the offset of the steady state of the vowel.
Consonants		
Plosives (/k/, /g/, /t/, /d/, /t̚/, /d̚/, /p/, /b/)	Voice onset time (VOT)	VOT was defined as the time interval between the release of a stop consonant and the onset of voicing, and was measured in milliseconds (ms). While measuring VOT for the unvoiced plosive, phonation is initiated after the stop release and the VOT value is written with a positive (+) sign.

	<p>This duration was selected for the target phonemes and VOT was calculated.</p> <p>In case of voiced plosives, the voicing is initiated prior to release of the plosive. Hence, a negative (-) sign preceded the VOT value indicating early initiation of phonation.</p>
Burst duration (BD)	<p>Release burst is usually seen as a vertical spike following the silent gap which is usually more intense for unvoiced stops than their voiced cognates. The segment marking the start and end of this vertical spike was selected and the duration for the same was measured (in ms).</p>
Closure duration (CD)	<p>Closure duration is also known as ‘silent gap’. It is a result of the “hold” period in articulation, during which the articulators involved form a complete obstruction and there is no flow of air out of the vocal tract. This can be measured only in case of the stop/plosive in the medial or final position. In this study, closure duration (in ms) was calculated for the words with the plosive in the medial position.</p>
Transition duration (TD)	<p>The formant frequencies change during the transition from one speech sound to another, referred to as formant transitions. The time taken (in ms) for this transition is labeled as the transition duration. It was measured as the time interval between the F₂ onset to offset or to the start of steady state of the following vowel considering CV as the syllable structure in which the target consonant was a plosive.</p>
Extent of transition (EoT)	<p>The difference in frequencies between the onset and offset of F₂ (in Hz) determined the EoT.</p>
Speed of	<p>The SoT was estimated by dividing the value</p>

	transition (SoT)	representing the EoT (in Hz) by the transition duration (in ms). The same can be represented as the following formula: $\text{SoT (Hz/ms)} = \text{EoT(Hz)} / \text{TD (ms)}$
Fricatives (/s/, /ʃ/, /f/)	Frication duration (FD)	The duration for which the frication noise prevails is labeled as Frication duration. The segment representing the onset and offset of frication was highlighted and the duration (in ms) was estimated.
	Transition duration (TD)	Transition duration was measured as the time interval (in ms) between the F ₂ onset to offset or to the start of steady state of the following vowel considering CV as the syllable structure, where the target consonant was a fricative.
	Extent of transition (EoT)	The difference in frequencies between the onset and offset of F ₂ (Hz) determined the EoT.
	Speed of transition (SoT)	The SoT was estimated by dividing the value representing the EoT (in Hz) by the transition duration (in ms). The same can be represented as the following formula: $\text{SoT (Hz/ms)} = \text{EoT(Hz)} / \text{TD (ms)}$
Voice		
Measure	Parameters	Method of measure
Subjective	Roughness Breathiness Strain Pitch Loudness Overall severity	Perceptual rating was done using CAPE-V. Additional features (if any) were also noted for each participant.
Objective	F ₀ F ₀ range Jitter I ₀	Extracted for phonation (/a/ vowel) and reading sample using Vagmi software.

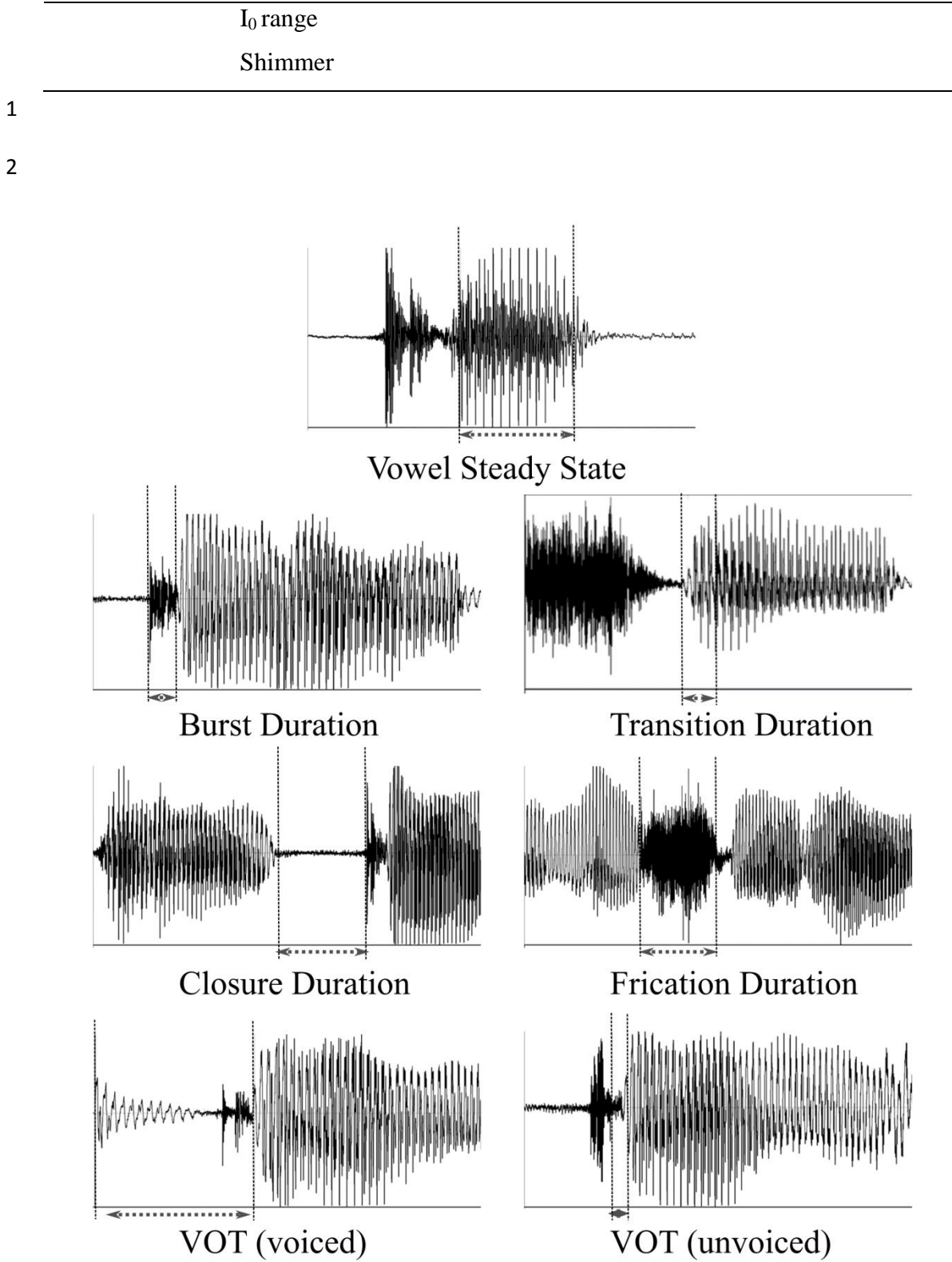


Figure 3.1: Representation of temporal measures considered in the study

1 **3.5.2.2 Suprasegmental characteristics**

2 a) *Emphasis*: All the subjects were asked to produce ten adjective-noun phrases
3 adopted from Ananthi and Savithri (2002). The stimuli were given in written form
4 with the target adjectives highlighted (bold and underlined). Participants were
5 instructed to read the target phrases once with emphasis on the adjective and once
6 without any emphasis. The recorded samples were opened in Praat software and
7 the target word i.e., the ‘adjective’ was selected and the following acoustic
8 parameters were extracted:

- 9 • Fundamental frequency (F_0)
- 10 • Mean Intensity (I_0) and
- 11 • Mean duration (D_0)

12

13 b) *Rhythm*: Each participant was asked to read five Kannada sentences adapted from
14 Santosh and Sahana (2012). The recorded samples were analyzed using Envelope
15 Modulation Spectrum (EMS) which is a MATLAB based script.

16

17 *Envelope Modulation Spectrum (EMS)*

18 EMS represents the gradual modulations or variations in the signal amplitude.
19 It depicts the distribution of energy in the amplitude fluctuations across frequencies.
20 The speech signal is subjected to a series of filtering and down-sampling using fast
21 fourier transform following which six EMS metrics (Peak frequency, Peak amplitude,
22 Energy 3-6 Hz, Energy 0-4 Hz, Energy 4-10 Hz, & Energy Ratio) were computed
23 from the resulting spectrum for the full signal (Liss, LeGendre, & Lotto, 2010). EMS
24 can be considered as an effective measure of rhythm over the traditional rhythm
25 analysis measures as it doesn’t demand identification of vowels and consonant

1 intervals, is completely automated in MATLAB, and takes into account the probable
2 pauses and non-phonetic elements in the sample (Liss et al., 2010). Several
3 researchers have proposed it to be useful in analyzing the rhythm metrics of speech
4 (Drullman et al., 1994; Greenberg, Arai, & Silipo, 1998). EMS has been used
5 successfully and proven to be effective in measuring rhythm in individuals with
6 dysarthria (Liss et al., 2010; Liss et al., 2009) and stuttering (Dechamma & Santosh,
7 2018).

8

9 *c) Intonation:* Each participant was asked to produce the five declaratives and five
10 interrogatives chosen as stimuli for the analysis of intonation. The recorded
11 responses of each participant were analyzed for the presence or absence of
12 intonation. Further, the pattern of intonation for each of the target stimuli was also
13 noted, and classified as rising, falling or level.

14

15 **3.6 Statistical Analyses**

16 Statistical Package for Social Sciences (SPSS) (Version 21) (SPSS Inc,
17 Chicago) was used for statistical analyses.

18

CHAPTER 4

RESULTS

The study aimed to characterize the speech production of individuals with Auditory Neuropathy Spectrum Disorder (ANSD) in terms of their segmental and suprasegmental features. The relationship of the speech production characteristics with their auditory processing deficits was explored. The results obtained are reported under the following major headings:

1. Auditory abilities in individuals with ANSD
2. Speech production characteristics in individuals with ANSD
3. Relationship between auditory abilities and speech production characteristics of ANSD

4.1 Auditory Abilities in individuals with ANSD

The auditory abilities of individuals with ANSD were compared with that of the individuals with normal auditory abilities (NAA) using Mann-Whitney U test **owing to the non-normal distribution of the data**. Table 4.1 shows the median and range of Speech Identification Scores (SIS) and Gap Detection Thresholds (GDT) in the two ears, in the two groups of participants. Typically, the median SIS was lesser and the median GDT was higher in the ANSD group compared to NAA group. Results of Mann-Whitney U test (Table 4.2) showed a significant difference between the two groups in SIS (in quiet as well as in noise) and GDT. This was true for both the ears.

1 Table 4.1

2 *Median and range of SIS (in quiet as well as in noise) and GDT obtained in ANSD*
 3 *and NAA groups*

Measure	Group	Right Ear		Left Ear	
		Median	Range	Median	Range
SIS (%)	ANSD	42	0-96	52	0-88
	NAA	100	100	100	100
SPIN (%) at 10dB SNR	ANSD	0	0-72	0	0-76
	NAA	100	84-100	100	92-100
SPIN (%) at 0dB SNR	ANSD	0	0-52	6	0-56
	NAA	100	88-100	100	92-100
GDT (ms)	ANSD	21.65	5.21-64.50	21.58	2.95-57.21
	NAA	2.72	1.65-4.37	2.87	2.15-5.21

4

5 Table 4.2

6 *Results of Mann-Whitney U test comparing the ANSD and NAA groups in terms of*
 7 *their SIS (in quiet as well as in noise) and GDT*

Measures	Ear	
	Right	Left
SIS (%)	7.126*	7.127*
SPIN (%) at 10 dB SNR	6.818*	7.052*
SPIN (%) at 0 dB SNR	6.997*	7.068*
GDT (ms)	6.657*	6.465*

8 *Note: *p < 0.001*

9

10 The scores of SIS (in quiet as well as in noise) and GDT obtained in the two
 11 ears of participants with ANSD were compared using Wilcoxon Signed-rank test.
 12 Results showed a significant difference between the two ears in SPIN at 10dB SNR
 13 (Z=3.211, p<0.05) and SPIN at 0dB SNR (Z=2.412, p<0.05) while there was no

1 significant difference between the two ears in GDT ($Z=0.623$, $p>0.05$) and SIS in
2 quiet ($Z=0.488$, $p>0.05$).

3

4 **4.2 Speech Production Characteristics in individuals with ANSD**

5 Both segmental and suprasegmental characteristics of the speech production of
6 individuals with ANSD were assessed. The segmental characteristics were assessed
7 separately in vowels and consonants. The suprasegmental aspects assessed included
8 emphasis, rhythm and intonation. The results of each of these parameters are reported
9 separately.

10

11 **4.2.1 Vowel production characteristics in individuals with ANSD in comparison** 12 **to individuals with NAA**

13 The results in this section address the vowel production characteristics in
14 individuals with ANSD and NAA. As mentioned in the earlier chapter, three words
15 were considered for each of the three short vowels (/a/, /ɪ/ & /ʊ/) and the following
16 acoustic characteristics were measured: Fundamental frequency (F_0), first formant
17 (F_1), first formant bandwidth (F_1BW), second formant (F_2), second formant bandwidth
18 (F_2BW), and vowel duration (VD). An average of the three words was computed for
19 each of these measures for each vowel. The mean and standard deviation of the
20 acoustic parameters in the two study groups are presented in Table 4.3. Considering
21 that the acoustic parameters will significantly vary between males and females, the
22 data are presented separately for the two genders.

23

24

25

1 Table 4.3

2 *Mean and standard deviation (SD) of the acoustic parameters of vowels in the two*
 3 *study groups*

Vowel	Parameter	ANSD Group (N = 30)				NAA Group (N = 30)			
		Male (N = 10)		Female (N = 20)		Male (N = 10)		Female (N = 20)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
/a/	F ₀ (Hz)	138.8	27.02	220.7	32.10	122.3	16.52	202.4	15.20
	F ₁ (Hz)	612.1	125.1	693.9	84.21	706.4	59.98	694.1	50.57
	F ₁ BW (Hz)	258.4	159.1	255.6	205.5	446.4	25.2	137.1	43.88
	F ₂ (Hz)	1380.5	220.7	1571.4	108.2	1443.7	127.5	1605.6	77.89
	F ₂ BW (Hz)	254.1	111.0	280.2	275.8	476.0	252.2	157.3	54.88
	VD (ms)	64.83	11.48	70.51	16.77	68.53	13.69	69.43	9.99
/ɪ/	F ₀ (Hz)	151.9	36.61	229.9	31.89	131.2	15.75	220.6	18.12
	F ₁ (Hz)	543.8	371.7	430.1	94.26	1351.5	636.8	423.0	54.63
	F ₁ BW (Hz)	301.1	373.7	173.7	105.7	283.3	173.1	190.3	169.1
	F ₂ (Hz)	2084.8	148.3	2437.5	156.0	2447.4	197.2	2511.3	116.7
	F ₂ BW (Hz)	256.3	202.1	642.2	813.0	310.1	135.8	299.6	167.7
	VD (ms)	64.30	18.51	68.41	26.62	67.96	21.56	61.10	13.07
/ʊ/	F ₀ (Hz)	147.2	30.60	232.16	21.99	131.86	19.13	213.93	31.24
	F ₁ (Hz)	513.0	183.0	515.4	126.8	783.1	231.5	474.6	69.28
	F ₁ BW (Hz)	262.3	155.8	224.7	216.3	309.9	118.5	224.4	158.4
	F ₂ (Hz)	1558.0	310.6	1451.2	191.4	2012.8	382.9	1455.1	114.5
	F ₂ BW (Hz)	403.4	335.0	331.2	318.5	497.3	239.5	457.4	241.8
	VD (ms)	59.20	28.13	55.98	21.12	58.00	14.57	58.55	10.63

Note: F₀ (Hz) – Fundamental frequency; F₁(Hz) – First formant; F₁BW (Hz) – Bandwidth of first formant;

5 F₂ (Hz) – Second formant; F₂BW (Hz) – Bandwidth of second formant; VD (ms) – Vowel duration

6

7 The data were subjected to a normality check for each gender in each of the
 8 groups using Shapiro-Wilk's test of normality. Normal distribution of data were found
 9 (p > 0.05) and hence parametric tests were carried out. The effect of gender was tested

1 using an independent t-test and the results revealed significant difference between the
 2 two genders in both ANSD and NAA groups (Table 4.4).

3

4 Table 4.4

5 *Results of independent t-test comparing two genders for their vowel production*
 6 *characteristics*

Vowel	Parameter	ANSD Group (N=30)	NAA Group (N=30)
/a/	F ₀ (Hz)	6.92**	13.22**
	F ₁ (Hz)	2.12*	0.59
	F ₁ BW (Hz)	0.03	5.35**
	F ₂ (Hz)	3.20*	4.32**
	F ₂ BW (Hz)	0.28	5.48**
	VD (ms)	0.96	0.20
/ɪ/	F ₀ (Hz)	6.01**	13.26**
	F ₁ (Hz)	1.30	6.58**
	F ₁ BW (Hz)	1.43	1.40
	F ₂ (Hz)	5.92**	1.11
	F ₂ BW (Hz)	1.46	0.17
	VD (ms)	0.43	1.08
/ʊ/	F ₀ (Hz)	8.73**	7.58**
	F ₁ (Hz)	0.04	5.56**
	F ₁ BW (Hz)	0.48	1.50
	F ₂ (Hz)	1.16	6.08**
	F ₂ BW (Hz)	0.57	0.42
	VD (ms)	0.35	0.11

7 *Note: *p < 0.05; **p < 0.001; df = 28*

8 *Note: F₀ (Hz) – Fundamental frequency; F₁(Hz) – First formant; F₁BW (Hz) – Bandwidth of first*
 9 *formant; F₂ (Hz) – Second formant; F₂BW (Hz) – Bandwidth of second formant; VD (ms) – Vowel*
 10 *duration*

11

12 Owing to significant differences between the two genders, the ANSD and NAA
 13 groups were compared with each other using an independent t-test, separately in
 14 males and females. The results of male participants revealed significantly lower F₁
 15 (for /a/, /ɪ/ & /ʊ/), F₂ (for /ɪ/ & /ʊ/), and F₂ bandwidth (for /a/) in ANSD group
 16 compared to NAA group (Table 4.5). On the contrary, in the female participants, the

1 results showed significantly higher F_0 (for /a/ & /ʊ/), and F_1 bandwidth in ANSD
 2 group compared to NAA group (Table 4.5).

3

4 Table 4.5

5 *Results of independent t-test comparing NAA and ANSD groups for their vowel*
 6 *production characteristics, in the two genders*

Parameters	Male (df=18)			Female (df=38)		
	/a/	/ɪ/	/ʊ/	/a/	/ɪ/	/ʊ/
F_0 (Hz)	1.64	1.64	1.34	2.30*	1.13	2.13*
F_1 (Hz)	2.14*	3.46*	2.89*	0.01	0.28	1.26
F_1 BW (Hz)	1.97	0.13	0.76	2.52*	0.37	0.01
F_2 (Hz)	0.78	4.64**	2.91*	1.14	1.69	0.07
F_2 BW (Hz)	2.54*	0.69	0.72	1.95	1.84	1.41
VD (ms)	0.65	0.40	0.12	0.24	1.10	0.48

7 *Note: * $p < 0.05$; ** $p < 0.001$*

8 *Note: F_0 (Hz) – Fundamental frequency; F_1 (Hz) – First formant; F_1 BW (Hz) – Bandwidth of first*
 9 *formant; F_2 (Hz) – Second formant; F_2 BW (Hz) – Bandwidth of second formant; VD (ms) – Vowel*
 10 *duration*

11

12 **4.2.2 Consonant production characteristics in individuals with ANSD in** 13 **comparison to individuals with NAA**

14 In the study, segmental characteristics of consonants were explored for
 15 plosives (/k/, /g/, /t/, /d/, /t̪/, /d̪/, /p/, /b/) and fricatives (/s/, /ʃ/, /f/). The results are
 16 reported separately for the two classes of consonants.

17

18 *Results of Plosives:* The acoustic parameters measured in plosives included- voice
 19 onset time (VOT), burst duration (BD), transition duration (TD), extent of transition
 20 (EoT), speed of transition (SoT) and closure duration (CD). The mean and standard
 21 deviation of the target measures are presented in Table 4.6.

22

23

1 Table 4.6
 2 Mean and Standard Deviation (SD) of the acoustic parameters measured in plosives,
 3 in the two study groups

Consonant	Parameter	Initial position				Medial position			
		ANSD group (N = 30)		NAA group (N = 30)		ANSD group (N = 30)		NAA group (N = 30)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
/p/	BD (ms)	10.60	4.70	8.63	4.00	10.80	5.83	8.76	3.79
	CD (ms)					99.10	34.69	99.36	29.39
	VOT (ms)	17.23	12.74	14.06	17.02	15.40	9.52	16.13	25.92
	TD (ms)	17.33	6.31	19.96	7.95	20.53	9.41	18.86	6.66
	EoT (Hz)	174.6	103.5	119.6	60.73	242.1	163.3	169.6	102.7
	SoT (Hz/ms)	11.52	8.83	6.62	3.97	13.70	11.23	10.01	7.18
	/b/	BD (ms)	9.13	3.36	9.93	3.94	9.93	4.76	8.66
CD (ms)						60.56	26.74	71.33	15.17
VOT (ms)		62.16	22.43	79.63	25.71	64.96	20.50	75.80	16.16
TD (ms)		19.30	5.98	19.66	6.42	18.83	6.81	17.60	6.68
EoT (Hz)		172.0	98.56	142.5	91.83	216.8	146.2	141.1	61.83
SoT (Hz/ms)		9.29	5.10	7.43	4.58	11.59	6.67	8.51	4.14
/t/		BD (ms)	9.80	4.38	8.30	3.01	11.70	4.92	10.00
	CD (ms)					88.56	30.02	88.36	16.95
	VOT (ms)	13.56	6.19	9.86	4.22	14.83	8.92	11.23	8.04
	TD (ms)	18.36	8.48	22.70	11.42	16.00	6.45	16.86	6.85
	EoT (Hz)	213.8	135.9	194.5	126.5	188.0	129.0	139.1	92.53
	SoT (Hz/ms)	12.82	7.75	9.62	5.69	13.66	10.67	9.28	7.48
	/d/	BD (ms)	10.36	4.73	8.83	3.67	9.76	4.84	8.96
CD (ms)						56.86	32.56	55.80	15.36
VOT (ms)		67.53	23.39	84.46	26.97	59.20	28.08	62.10	12.35
TD (ms)		17.90	6.82	19.86	10.64	18.50	5.06	15.93	6.62
EoT (Hz)		181.5	90.12	158.8	110.8	184.1	162.8	137.1	73.39
SoT (Hz/ms)		11.23	6.26	9.23	6.46	11.20	12.99	9.23	5.61
/t/		BD (ms)	7.33	2.66	6.43	1.67	8.06	2.59	6.96
	CD (ms)					73.60	22.03	80.90	18.41
	VOT (ms)	11.00	7.26	6.83	2.10	11.20	5.04	6.63	1.93
	TD (ms)	18.93	8.76	22.03	11.00	17.80	8.89	16.63	6.50
	EoT (Hz)	199.1	123.1	220.4	118.3	195.3	116.0	147.5	84.40
	SoT (Hz/ms)	11.67	8.21	11.19	5.77	12.69	8.76	9.21	5.49
	/d/	BD (ms)	8.50	3.44	7.06	3.11	8.03	3.65	6.43
CD (ms)						35.90	27.46	31.66	18.17
VOT (ms)		64.10	23.80	81.40	24.40	37.33	17.17	35.30	13.91

	TD (ms)	13.13	4.51	14.50	4.84	15.63	7.82	17.33	8.23
	EoT (Hz)	118.9	96.05	147.5	88.82	151.4	96.75	148.8	88.01
	SoT (Hz/ms)	10.42	11.02	10.10	4.56	11.21	8.13	9.80	6.16
	BD (ms)	20.36	7.79	20.00	6.72	18.26	7.94	18.70	5.47
	CD (ms)					96.33	39.17	90.70	21.74
	VOT (ms)	19.23	8.60	13.80	5.95	18.76	9.29	16.13	7.70
/k/	TD (ms)	17.63	6.62	18.66	9.61	16.90	7.57	17.76	6.59
	EoT (Hz)	109.7	61.81	130.5	97.18	149.7	119.4	133.0	110.9
	SoT (Hz/ms)	6.46	3.12	7.36	4.32	9.18	5.61	7.89	7.70
	BD (ms)	15.30	7.07	18.00	6.34	14.66	8.53	14.30	5.36
	CD (ms)					45.66	18.01	52.83	12.11
	VOT (ms)	58.93	25.60	83.90	23.08	56.80	25.09	67.53	10.62
/g/	TD (ms)	17.73	6.01	18.86	6.96	17.46	7.28	17.26	6.16
	EoT (Hz)	130.2	88.53	112.8	52.01	126.7	102.4	108.3	69.37
	SoT (Hz/ms)	7.90	5.61	6.41	3.50	7.34	4.49	6.22	3.60

1 *Note: BD - Burst duration; CD - Closure duration; VOT - Voice onset time; TD - Transition duration;*
2 *EoT- Extent of transition; SoT - Speed of transition.*

3

4 Owing to the normal distribution of the data (assessed using Shapiro-Wilk's
5 test of normality), the two study groups were compared for their segmental
6 characteristics of plosives using independent t -test. The results revealed significant
7 differences between two groups for VOT of /b/, /t/, /d/, /t/, /d/, /k/, and /g/ in initial
8 position, and VOT of /b/, /t/, and /g/ in medial position. It was observed that
9 participants in ANSD group had longer VOT for unvoiced plosives and shorter VOT
10 for voiced plosives in comparison to NAA group.

11

12 A significantly longer BD was observed for /d/ in medial position in
13 individuals with ANSD. Further, the EoT was significantly longer for /p/ in initial
14 position, and /p/ and /b/ in medial position in ANSD group when compared to NAA
15 group. Significantly longer SoT was also observed for /p/ in initial position and /b/ in
16 medial position in individuals with ANSD when compared to individuals with NAA.

1 No significant group differences were observed in other acoustic parameters (Table
2 4.7).

3

4 Table 4.7

5 *Results of independent t-test comparing two groups for the production characteristics*
6 *of plosives*

Consonant	Position	Parameters					
		BD (ms)	CD (ms)	VOT (ms)	TD (ms)	EoT (Hz)	SoT (Hz/ms)
/p/	Initial	1.74		0.81	1.42	2.51*	2.76*
	Medial	1.60	0.03	0.14	0.79	2.05*	1.51
/b/	Initial	0.84		2.81*	0.22	1.19	1.49
	Medial	1.03	1.91	2.27*	0.70	2.61*	2.14*
/t̪/	Initial	1.54		2.70*	1.66	0.56	1.82
	Medial	1.46	0.03	1.64	0.50	1.68	1.84
/d̪/	Initial	1.40		2.59*	0.85	0.87	1.21
	Medial	0.69	0.16	0.51	1.68	1.44	0.76
/t̪/	Initial	1.56		3.01*	1.20	0.68	0.26
	Medial	1.82	1.39	4.63**	0.58	1.82	1.84
/d̪/	Initial	1.69		2.77*	1.13	1.19	0.14
	Medial	2.09*	0.70	0.50	0.82	0.11	0.75
/k/	Initial	0.19		2.84*	0.48	0.98	0.93
	Medial	0.24	0.68	1.19	0.47	0.56	0.74
/g/	Initial	1.55		3.96**	0.67	0.92	1.23
	Medial	0.19	1.80	2.15*	0.11	0.81	1.07

7 Note: * $p < 0.05$; ** $p < 0.001$; $df = 58$

8 Note: BD - Burst duration; CD - Closure duration; VOT - Voice onset time; TD - Transition duration;
9 EoT - Extent of transition; SoT - Speed of transition.

10

11 *Results of Fricatives:* The acoustic parameters measured in fricatives included -
12 frication duration (FD), transition duration (TD), speed of transition (SoT) and extent
13 of transition (EoT). The mean and standard deviation of the target measures are
14 presented in Table 4.8.

15

1 Table 4.8
 2 *Mean and Standard Deviation (SD) of the acoustic parameters measured in fricatives,*
 3 *in the two study groups*

Consonant	Parameter	Initial Position				Medial Position			
		ANSD Group (N = 30)		NAA Group (N = 30)		ANSD Group (N = 30)		NAA Group (N = 30)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
/s/	FD (ms)	115.8	28.59	118.3	25.09	115.1	27.11	112.5	15.01
	TD (ms)	22.31	9.35	18.60	4.15	22.62	9.39	20.70	5.34
	EoT (Hz)	205.2	154.1	158.8	126.6	188.8	116.7	180.8	72.36
	SoT (Hz/ms)	9.48	5.53	9.28	8.25	8.95	5.65	8.97	3.43
/ʃ/	FD (ms)	132.6	36.25	118.0	24.85	122.2	27.91	114.3	16.66
	TD (ms)	20.55	5.83	17.56	4.91	22.13	8.49	20.83	7.98
	EoT (Hz)	212.6	125.2	208.9	165.7	202.2	159.5	181.9	82.82
	SoT (Hz/ms)	10.96	6.24	12.33	8.51	9.33	6.73	9.52	4.87
/f/	FD (ms)	109.5	40.25	116.9	28.96	91.65	35.90	108.8	21.46
	TD (ms)	25.17	9.43	21.83	7.52	21.86	8.24	25.26	11.96
	EoT (Hz)	206.8	109.7	192.1	133.6	277.9	172.6	289.5	174.8
	SoT (Hz/ms)	8.80	5.48	9.21	5.36	12.76	6.01	11.32	4.77

4 *Note: FD – Frication duration; TD – Transition duration; EoT – Extent of transition; SoT – Speed of*
 5 *transition*

6
 7 The data were found to adhere to normal distribution (assessed using Shapiro-
 8 Wilk’s test of normality), and hence the two groups were compared for the segmental
 9 characteristics of fricatives using an independent t-test. The results revealed
 10 significantly longer TD of /ʃ/ in initial position in the ANSD group compared to NAA
 11 group. Significantly shorter FD was also observed for /f/ in medial position in
 12 individuals with ANSD when compared to NAA group. No significant group
 13 differences were observed in other acoustic parameters (Table 4.9).

14

15

1 Table 4.9

2 *Results of independent t-test comparing two groups for the production characteristics*
 3 *of fricatives*

Consonant	Position	Parameters			
		FD (ms)	TD (ms)	EoT (Hz)	SoT (Hz/ms)
/s/	Initial	0.35	1.98	1.26	0.10
	Medial	0.45	0.97	0.31	0.02
/ʃ/	Initial	1.80	2.12*	0.09	0.70
	Medial	1.32	0.60	0.61	0.12
/f/	Initial	0.80	1.50	0.46	0.28
	Medial	2.23*	1.26	0.25	1.01

4 *Note: *p < 0.05; df = 58, FD – Frication duration; TD – Transition duration; EoT – Extent of*
 5 *transition; SoT – Speed of transition*

6

7 **4.2.3 Voice production characteristics in individuals with ANSD in comparison**
 8 **to individuals with NAA**

9 The voice characteristics were assessed using a standard perceptual rating
 10 scale – CAPE-V and an objective analysis of phonation and reading sample using the
 11 Vagmi Diagnostics software tool. The median and range of the CAPE-V measures are
 12 presented in Table 4.10. The table presents the data of male and female participants
 13 separately in view of the known characteristic differences of voice in the two genders.

14

15 Table 4.10

16 *Median and range for CAPE-V perceptual rating scale in the two study groups*

Parameter (in %)	ANSD Group (N = 30)				NAA Group (N = 30)			
	Male (N = 10)		Female (N = 20)		Male (N = 10)		Female (N = 20)	
	Med	Range	Med	Range	Med	Range	Med	Range
Roughness	17.50	0-20	17.50	0-25	0.00	0-5	0.00	0-5
Breathiness	7.50	0-20	0.00	0-20	0.00	0-5	0.00	Nil
Strain	15.00	0-25	17.50	0-35	0.00	Nil	0.00	Nil
Pitch	15.00	0-15	0.00	0-25	0.00	Nil	0.00	0-5
Loudness	0.00	0-30	0.00	0-20	0.00	Nil	0.00	Nil
Overall severity	17.50	0-25	25.00	0-25	0.00	0-5	0.00	0-5

17 *Note: Med = Median*

1 Shapiro-Wilk's test of normality revealed non-normal distribution ($p < 0.05$)
 2 of the data in both male and female groups, and thus non-parametric test was carried
 3 out. Perceptual rating of CAPE-V was compared between ANSD and NAA groups
 4 using Mann-Whitney U test. Results revealed significant difference between male
 5 participants of the two groups for Roughness, Breathiness, Strain, Pitch, Loudness,
 6 and the Overall severity. Similar results were obtained on comparison of female
 7 participants between the groups, who were found to differ in all five parameters and
 8 the overall severity (Table 4.11).

9
 10 Table 4.11

11 *Results of Mann-Whitney U test comparing two groups for their perceptual rating of*
 12 *voice using CAPE-V*

Parameter (in %)	Male	Female
Roughness	3.07*	3.80**
Breathiness	2.14*	2.86*
Strain	3.42*	4.22**
Pitch	3.43*	2.42*
Loudness	2.16*	2.86*
Overall severity	3.60**	4.63**

13 *Note: * $p < 0.05$; ** $p < 0.001$*

14 Apart from statistically comparing the median percentage of perceptual
 15 deviance between the two study groups, an attempt was made to assign the degree of
 16 deviance (as standardized in CAPE-V) based on the perceptual rating. All the
 17 individuals with NAA had received percentage of deviance of less than 5%. Table
 18 4.12 presents the distribution of participants of ANSD group across the different
 19 degrees of perceptual deviance as rated on CAPE-V scale. In all the parameters most
 20 of the individuals with ANSD obtained perceptual rating within normal limits (less
 21 than 10%), whereas only few of them were rated as mildly deviant as presented in the
 22 table.

1 Table 4.12

2 *Distribution of participants of ANSD group across the different degrees of perceptual*
3 *deviance as rated on CAPE-V scale*

Parameter	Normal	Mildly deviant	Moderately deviant	Severely deviant
Roughness	24	6		
Breathiness	27	3		
Strain	22	8		
Pitch	27	3		
Loudness	28	2		
Overall severity	24	6		

4

5 The median and range of the acoustic measures extracted from Vagmi in the
6 two groups of individuals are presented in Table 4.13. The table presents the data of
7 male and female participants separately owing to the known characteristic differences
8 of voice in the two genders. The derived acoustic parameters were compared between
9 the two groups (ANSD & NAA) using Mann-Whitney U test. The results of male
10 participants showed significantly higher F_0 range ($|z| = 3.51, p < 0.001$) and jitter ($|z| =$
11 $3.30, p = 0.001$) in ANSD group compared to NAA group during phonation task.
12 Similarly, in female participants, the F_0 range ($|z| = 3.40, p = 0.001$), I_0 range ($|z| =$
13 $3.73, p < 0.001$), and shimmer ($|z| = 3.80, p < 0.001$) were significantly higher in
14 ANSD group during phonation task (Table 4.14).

15

1 Table 4.13

2 *Median and range for acoustic parameters extracted from Vagmi in the two study*
 3 *groups*

Task	Parameters	ANSD Group (N = 30)				NAA Group (N = 30)			
		Male (N = 10)		Female (N = 20)		Male (N = 10)		Female (N = 20)	
		Med	Range	Med	Range	Med	Range	Med	Range
Phonation	Mean F ₀ (Hz)	135.8	107.4-169.6	215.8	158.7-290.9	125.5	104.7-163.7	215.0	184.0-273.8
	F ₀ Range (Hz)	23.54	9.19-112.7	15.43	4.74-146.7	6.90	3.01-12.55	8.60	4.20-20.03
	Mean I ₀ (dB)	109.6	104.7-119.6	112.5	104.3-117.7	110.1	104.07-111.1	111.2	105.7-115.0
	I ₀ range (dB)	6.00	1.05-18.30	6.61	1.78-20.24	4.18	1.67-8.71	3.26	1.08-26.83
	Jitter (%)	1.84	1.02-4.45	1.50	0.67-13.08	0.87	0.49-1.67	1.12	0.34-3.20
	Shimmer (dB)	0.70	0.36-1.35	0.64	0.25-0.97	0.55	0.37-1.40	0.39	0.15-0.85
Reading	Mean F ₀ (Hz)	141.9	109.7-182.3	234.8	209.3-270.0	125.9	112.8-148.9	224.5	197.0-259.6
	F ₀ Range (Hz)	62.79	40.75-98.49	117.4	45.65-196.7	64.30	43.60-82.14	134.1	100.1-183.3
	Mean I ₀ (dB)	103.7	99.60-109.3	109.7	97.82-109.0	102.1	98.26-103.2	105.1	100.1-110.3
	I ₀ Range (dB)	32.74	23.26-35.23	31.32	26.44-36.95	31.35	25.57-33.30	30.81	19.53-37.06

4 *Note: Med = Median; F₀ – Fundamental frequency; I₀ – Intensity*

5 Table 4.14

6 *Results of Mann-Whitney U test comparing two groups for acoustic parameter of*
 7 *voice extracted from Vagmi*

Task	Parameter	Male	Female
Phonation	Mean F ₀ (Hz)	1.63	0.13
	F ₀ Range (Hz)	3.51**	3.40*
	Mean I ₀ (dB)	0.57	0.93
	I ₀ range (dB)	1.14	3.73**
	Jitter (%)	3.30*	1.79
	Shimmer (dB)	0.32	3.80**
Reading	Mean F ₀ (Hz)	0.89	1.59
	F ₀ Range (Hz)	1.87	1.05
	Mean I ₀ (dB)	0.73	1.78
	I ₀ Range (dB)	1.06	0.37

8 *Note: *p < 0.05; **p < 0.001; F₀ – Fundamental frequency; I₀ – Intensity*

1 **4.2.4 Emphasis production characteristics in individuals with ANSD in**
 2 **comparison to individuals with NAA**

3 Production of emphasis was assessed using three parameters – F_0 , I_0 , and D_0 . A
 4 total of ten adjective-noun phrases served as the stimuli. An average of the ten phrases
 5 was obtained and the averaged data was subjected to statistical analysis. The data in
 6 these parameters were found to be normally distributed, as tested using Shapiro-
 7 Wilk’s test. The mean and standard deviation of the acoustic parameters of emphasis
 8 in the two study groups are presented in Table 4.15. The data are presented separately
 9 for males and females in view of known differences in the suprasegmental speech
 10 characteristics between males and females.

11
 12 Table 4.15
 13 *Mean and Standard Deviation (SD) of emphasis production*

Condition	Parameter	ANSD Group (N = 30)				NAA Group (N = 30)			
		Male (N = 10)		Female (N = 20)		Male (N = 10)		Female (N = 20)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
With emphasis	F_0 (Hz)	169.3	20.87	254.7	22.76	165.6	20.09	259.2	24.38
	I_0 (dB)	82.48	3.05	80.88	2.34	80.11	3.30	81.59	3.02
	D_0 (ms)	485.6	178.9	594.9	121.4	593.4	114.0	514.4	60.48
Without emphasis	F_0 (Hz)	170.4	47.33	244.0	25.35	151.1	21.78	232.2	233.2
	I_0 (dB)	79.83	6.18	80.11	3.04	76.37	3.92	119.1	176.4
	D_0 (ms)	388.1	146.8	443.1	98.66	410.3	123.0	373.5	43.95

14
 15 The results of independent t-test showed no significant difference between the
 16 ANSD and NAA groups in male participants for any of the parameters. However, in
 17 females, there was a significantly longer D_0 in ANSD group compared to that of NAA
 18 group. Such a difference was not present in F_0 and I_0 in females. The result was true
 19 both in without- and with-emphasis conditions (Table 4.16).

1 Table 4.16

2 *Results of independent t-test comparing the acoustic parameters of emphasis the two*
3 *study groups separately in males and females*

Condition	Parameter	Male (df=18)	Female (df=18)
With emphasis	F ₀ (Hz)	0.40	0.61
	I ₀ (dB)	1.66	0.82
	D ₀ (ms)	1.60	2.65*
Without emphasis	F ₀ (Hz)	1.17	1.58
	I ₀ (dB)	1.49	0.98
	D ₀ (ms)	0.36	2.88*

4 *Note: *p < 0.05*

5 **4.2.5 Speech rhythm characteristics in individuals with ANSD in comparison to** 6 **individuals with NAA**

7 Speech rhythm was documented using automated analysis of envelope
8 modulation spectra (EMS), which was extracted for full band signal. From the
9 extracted envelope, six predictor variables (peak frequency, peak amplitude, energy in
10 the region 3-6 Hz, energy in spectrum from 0-4 Hz, energy in spectrum from 4-10 Hz,
11 & ratio of energy below 4Hz/above 4Hz) were computed. When tested with the
12 Shapiro-Wilk's test of normality, the data were found to be normally distributed ($p >$
13 0.05). The mean and standard deviation of the six predictor variables are presented in
14 Table 4.17.

15
16 Table 4.17

17 *Mean and Standard Deviation (SD) of the six EMS predictor variables of rhythm in*
18 *the two study groups*

Parameter	ANSD		NAA	
	Mean	SD	Mean	SD
Peak frequency	0.84	0.43	1.16	0.86
Peak Amplitude	1.46	0.09	1.36	0.06
Energy 3-6 Hz	0.30	0.01	0.30	0.01
Energy 0-4 Hz	0.44	0.01	0.42	0.01
Energy 4-10 Hz	0.55	0.01	0.57	0.01
Energy Ratio	0.81	0.05	0.74	0.03

1 Comparison of the two study groups (ANSD & NAA) on independent t-test
 2 showed a significant difference in peak amplitude [$t(56) = 4.87, p < 0.001$], energies
 3 in the region of 0-4 Hz [$t(56) = 5.95, p < 0.001$] and 4-10 Hz [$t(56) = 5.11, p < 0.001$],
 4 and energy ratio [$t(56) = 5.70, p < 0.001$]. However, there was no significant
 5 difference in peak frequency [$t(56) = 1.78, p > 0.05$] and energy in 3-6 Hz region
 6 [$t(56) = 0.06, p > 0.05$]. **These significant differences between the two groups reflect**
 7 **the deviant rhythm characteristics in individuals with ANSD.**

8
 9 **4.2.6 Intonation characteristics in individuals with ANSD in comparison to**
 10 **individuals with NAA**

11 The intonation was perceptually analyzed for its presence, and if present, the
 12 pattern of intonation was identified. It was found that all the individuals with NAA
 13 showed intonation patterns in both declarative and interrogative sentences. However,
 14 only some of the participants with ANSD showed presence of intonation. The details
 15 of the number of individuals who showed intonation patterns (gender-wise data) in
 16 their speech and the respective pattern of intonation are given in Table 4.18.

17 Table 4.18

18 *Distribution of participants of ANSD group based on presence of intonation pattern*
 19 *for the two sentence types (Appendix 1)*

Sentence	Male (N = 10)	Female (N = 20)	Total (N = 30)	Pattern when present (both genders together)	
				Rise	Fall
Declarative 1	3	9	12	4	8
Declarative 2	4	8	12	0	12
Declarative 3	4	7	11	0	11
Declarative 4	3	7	10	0	10
Declarative 5	5	9	14	2	12
Interrogative 1	6	9	15	3	12
Interrogative 2	6	9	15	6	9
Interrogative 3	5	7	12	5	13

Interrogative 4	4	11	15	7	8
Interrogative 5	6	12	18	4	8

1

2 The data showed that most of the individuals with ANSD were monotonous.
3 This was true in both males and females. The observation of intonation patterns, when
4 present, reveals that correct pattern was followed for declarative sentences in most
5 instances. On the contrary, falling pattern was seen for interrogative sentences instead
6 of the typical rising pattern in most instances. Table 4.18 represents the instances of
7 wrong patterns through shading of the cells.

8

9 **4.3 Relationship between auditory abilities and speech production characteristics**
10 **of ANSD**

11 The purpose of this analysis is to determine the relationship between auditory
12 abilities and speech production characteristics in individuals with ANSD. To do this,
13 in the ANSD group, individuals with good auditory abilities were compared to those
14 with poor auditory abilities. The good and poor auditory ability was defined
15 relatively. The auditory abilities considered were pure tone average, SIS in quiet,
16 SPIN, GDT, presence/absence of speech evoked ALLR and presence/absence of tone
17 burst elicited ALLR. Only those parameters of speech that were found to be
18 significantly deviant in ANSD group compared to the control group were considered
19 for comparison in this section.

20

21 **4.3.1 Comparison between individuals in whom ALLR was present and ALLR**
22 **was absent**

23 The speech production characteristics of individuals with ANSD in whom
24 ALLR was present were compared with those without ALLR. This was done

1 separately for speech elicited ALLR and tone burst elicited ALLR, and also separately
 2 for the two ears.

3 **a. Results of ALLR for speech**

4 Among the participants with ANSD, nine of them had presence while 21 had
 5 absence of ALLR for speech in the right ear. Similarly, there were six participants
 6 with present and 24 participants with absent ALLR for speech in the left ear. All the
 7 parameters of speech that showed significant deviance between ANSD and NAA
 8 groups were compared between those who had and those who did not have speech
 9 elicited ALLR. The median VOT (when /k/ is in the initial position), and BD (when
 10 /d/ is in medial position) was higher in those with absent ALLR compared to those
 11 with presence of ALLR. The results of Mann-Whitney U test showed a significant
 12 difference between the two groups of ANSD in the parameters given in Table 4.19.

13
 14 Table 4.19

15 *The results of Mann-Whitney U test for parameters of speech that showed significant*
 16 *difference between participants with and without speech elicited ALLR*

Ear	Parameter of speech	Participants with	Median	Range	Z
Right	VOT /k/ initial	Presence of ALLR	10.00	5-36	3.354*
		Absence of ALLR	22.00	7-31	
	BD /d/ medial	Presence of ALLR	6.00	5-9	2.393*
		Absence of ALLR	7.00	4-23	
Left	VOT /k/ initial	Presence of ALLR	11.50	5-36	2.337*
		Absence of ALLR	21.50	7-32	

17 Note: * $p < 0.05$

18
 19 **b. Results of ALLR for tone burst**

20 Among the participants with ANSD, ten of them had presence while 20 had
 21 absence of ALLR for tone burst in the right ear. Similarly, there were eight
 22 participants with present and 22 participants with absent ALLR for tone burst in the

1 left ear. Results of Mann-Whitney U test revealed significant difference between
 2 participants with and without tone burst elicited ALLR only for VOT (when /k/ is in
 3 the initial position) (Table 4.20). The median VOT (when /k/ is in the initial position)
 4 was significantly prolonged in individuals with absent ALLR compared to those with
 5 ALLR. These significant differences between the two groups reflect that the
 6 individuals with poorer ALLR produce longer VOT and BD in order to enhance their
 7 feedback of self-produced speech.

8

9 Table 4.20

10 *The results of Mann-Whitney U test for parameters of speech that showed significant*
 11 *difference between participants with and without tone burst elicited ALLR*

Ear	Parameter of speech	Participants with	Median	Range	Z
Left	VOT /k/ initial	Presence of ALLR	12.00	5-36	2.043*
		Absence of ALLR	21.50	5-32	

12 *Note: *p<0.05*

13

14 **4.3.2 Comparison between individuals with good and poor auditory abilities**

15 In order to derive the relationship between auditory abilities and speech
 16 production characteristics in individuals with ANSD, the participants were divided
 17 into ‘Good’ and ‘Poor’ performers based on their SIS, SPIN (at 0 & 10 dB SNR),
 18 GDT and PTA. The confidence intervals were derived from the ANSD group in each
 19 of these auditory measures. The scores of only the left ear were considered for this
 20 analysis as the deviation based on the left ear scores gave equivalent number of
 21 participants in the good and poor performer groups. The participants with a score
 22 equal to or more than the upper bound were grouped as ‘Good performers’, while
 23 those with a score equal to or less than the lower bound were grouped as ‘Poor
 24 performers’ in SIS and SPIN. Vice-versa was the definition of the good and poor

1 performers in GDT and PTA. Subsequently, the speech production characteristics
2 were compared between the good and poor performers. Only those parameters of
3 speech that showed a significant deviance between ANSD and NAA groups were
4 considered for such comparisons.

5

6 **a. Comparison between participants with good and poor SIS**

7 Based on the SIS of participants with ANSD, the upper bound score was 55
8 and the lower bound score was 29. Accordingly, there were 13 good performers and
9 13 poor performers. The results of Mann-Whitney U test showed that there was no
10 significant difference in any of the parameters of speech between good and poor
11 performers.

12

13 **b. Comparison between participants with good and poor SPIN at 10 dB SNR**

14 Based on the SPIN scores obtained in participants with ANSD at 10 dB SNR,
15 the upper bound score was 24 and the lower bound score was 7. Accordingly, there
16 were eight good performers and 17 poor performers. Table 4.21 gives the results of
17 Mann-Whitney U test comparing between good and poor performers in terms of their
18 speech production characteristics. The results showed a

19 a) significantly prolonged median VOT (in /t/ in initial position) in the group of poor
20 performers compared to the group of good performers

21 b) significantly different peak amplitude, energy 0-4 Hz, energy 4-10 Hz and energy
22 ratio of speech rhythm in the group of poor performers compared to the group of
23 good performers.

24

25

1 Table 4.21

2 *The results of Mann-Whitney U test comparing between good and poor performers*
3 *(classified based on their SPIN score at 10 dB SNR) in terms of their speech*
4 *production characteristics*

Parameter of speech	Participants with	Median	Range	Z
VOT /t/ initial	Good performance	9.50	8-16	2.40*
	Poor performance	14.00	8-34	
Peak amplitude full band	Good performance	1.40	1.33-1.76	2.15*
	Poor performance	1.48	1.39-1.67	
Energy 0-4 Hz @ FB	Good performance	0.43	0.43-0.50	2.56*
	Poor performance	0.44	0.43-0.47	
Energy 4-10 Hz @ FB	Good performance	0.56	0.50-0.57	2.56*
	Poor performance	0.55	0.53-0.57	
Energy Ratio @ FB	Good performance	0.77	0.75-0.98	2.56*
	Poor performance	0.81	0.75-0.90	

5 *Note: *p < 0.05*

6

7 **c. Comparison between participants with good and poor SPIN at 0 dB SNR**

8 Based on the SPIN scores obtained in participants with ANSD at 0 dB SNR,
9 the upper bound score was 23 and the lower bound score was 8. Accordingly, there
10 were nine good performers and 16 poor performers. Table 4.22 gives the results of
11 Mann-Whitney U test comparing between good and poor performers in terms of their
12 speech production characteristics. The results showed the following in the group of
13 poor performers compared to the group of good performers.

14 a) significantly prolonged median VOT (in /t/ in initial position & /t/ in medial
15 position)

16 b) significantly shorter median EoT (in /p/ in medial position)

17 c) significantly shorter median FD (in /f/ in medial position)

18 d) significantly higher breathiness rating for voice

19

20

1 Table 4.22

2 *The results of Mann-Whitney U test comparing between good and poor performers*
3 *(classified based on their SPIN score at 0 dB SNR) in terms of their speech*
4 *production characteristics*

Parameter of speech	Participants with	Median	Range	Z
VOT /t/ initial	Good performance	9.00	5-16	2.76*
	Poor performance	14.00	8-34	
VOT /t/ medial	Good performance	8.00	6-14	1.91*
	Poor performance	10.50	7-25	
EoT /p/ medial	Good performance	253.80	163-654	2.32*
	Poor performance	124.20	17.40-563	
FD /f/ medial	Good performance	103.00	35-134	2.20*
	Poor performance	70.00	22-139	
Breathiness	Good performance	0.00	0-10	2.03*
	Poor performance	10.00	0-50	

5 *Note: *p < 0.05*

6

7 **d. Comparison between participants with good and poor GDT**

8 Based on the GDT obtained in participants with ANSD, the upper bound score
9 was 31 and the lower bound score was 19. Accordingly, there were 13 good
10 performers and 12 poor performers. Table 4.23 gives the results of Mann-Whitney U
11 test comparing between good and poor performers in terms of their speech production
12 characteristics. The results showed a significantly prolonged VOT (in /t/ in initial
13 position) in the group of poor performers compared to the group of good performers.

14

1 Table 4.23

2 *The results of Mann-Whitney U test comparing between good and poor performers*
3 *(classified based on their GDT) in terms of their speech production characteristics*

Parameter of speech	Participants with	Median	Range	Z
VOT /t/ initial	Good performance	8.00	5-14	2.09*
	Poor performance	11.50	7-45	

4 *Note: *p < 0.05*

5

6 **e. Comparison between participants with good and poor hearing sensitivity**

7 Based on the pure tone average of participants with ANSD (only the left ear),
8 the upper bound was 47 dB and the lower bound was 33 dB. Accordingly, there were
9 nine good performers and eight poor performers. Table 4.24 gives the results of
10 Mann-Whitney U test comparing between good and poor performers in terms of their
11 speech production characteristics. The results showed a significantly shorter VOT (in
12 /g/ in initial position) in the group of poor performers compared to the group of good
13 performers. The results of CAPE-V showed a significantly higher rating for
14 breathiness and strain in voice in the group of poor performers compared to the group
15 of good performers.

16

17 Table 4.24

18 *The results of Mann-Whitney U test comparing between good and poor performers*
19 *(classified based on their PTA) in terms of their speech production characteristics*

Parameter of speech	Participants with	Median	Range	Z
VOT /g/ initial	Good performance	61.00	37-114	1.97*
	Poor performance	39.50	21-70	
Breathiness	Good performance	0.00	0-10	2.55*
	Poor performance	15.00	0-50	
Strain	Good performance	10.00	0-50	2.09*
	Poor performance	27.50	0-50	

20 *Note: *p < 0.05*

1 **4.3.3 Correlation between auditory and speech production measures**

2 The speech production measures were assessed for their correlation with
 3 auditory measures in participants with ANSD, using Spearman’s rank correlation test.
 4 Only the scores of left ear were considered for this purpose. The results showed that
 5 there was no significant correlation of any of the speech production measures with
 6 that of SIS and SPIN at 0 dB SNR. However, SPIN at 10 dB SNR, GDT and PTA
 7 showed a significant correlation with some of the speech production measures. The
 8 results showed that

- 9 • the SPIN at 10 dB SNR showed a significant negative correlation with VOT of
 10 /t/ and /t/, and some of the parameters of rhythm (Peak amplitude, Energy in 0-
 11 4 Hz & the Energy ratio) as given in Table 4.25.
- 12 • the SPIN at 10 dB SNR showed a significant positive correlation with Energy
 13 in 0-4 Hz (Table 4.25).
- 14 • GDT scores significantly correlated with Energy in 0-4 Hz ($r = 0.38, p =$
 15 0.03), Energy in 4-10 Hz ($r = -0.38, p = 0.03$) and the Energy ratio ($r = 0.38, p$
 16 $= 0.03$).
- 17 • PTA significantly correlated with VOT of /g/ in initial position ($r = -0.37, p =$
 18 0.039) and breathiness rated in CAPE-V ($r = 0.53, p = 0.002$)

19
 20 Table 4.25

21 *The results of Spearman’s rank correlation showing significant correlation between*
 22 *SPIN score at 10 dB SNR and some of the measures of speech production*

Parameter	VOT /t/ initial	VOT /t/ initial	PA	Energy 0-4 Hz	Energy 4-10 Hz	Energy Ratio
<i>r</i>	-0.46	-0.26	-0.44	-0.47	0.47	-0.47
<i>p</i>	0.010	0.15	0.015	0.008	0.008	0.008

23 *Note: VOT = Voice Onset Time; PA – Peak Amplitude*

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CHAPTER 5
DISCUSSION

Auditory Neuropathy Spectrum Disorder (ANSD) is known to result in distortion of the auditory feedback owing to its temporal processing and speech perception deficits. Based on the available literature in sensorineural hearing loss, one can expect individuals with long-standing ANSD to show deviations in their speech production characteristics. Therefore, in the present study, it was attempted to study the characteristics of speech production in individuals with long-standing ANSD. Attempt was also made to study the relationship between their speech production characteristics and the auditory abilities. Overall, the results support presence of deviations in speech production which appear to relate to their temporal processing deficits. The specific findings are discussed under the following headings:

- 5.1 Auditory abilities of individuals with ANSD
- 5.2 Speech production of individuals with ANSD
- 5.3 Relationship between auditory abilities and speech production in individuals with ANSD

5.1 Auditory abilities of individuals with ANSD

Speech perception and the gap detection thresholds (GDTs) were assessed in the study. It was found that individuals with ANSD performed significantly poorer compared to individuals with normal auditory abilities (NAA) in both these measures. Reduced speech perception, both in quiet and in noise, is known to be characteristic of ANSD and is primarily the result of a deficit in temporal processing (Zeng et al., 1999; 2005). GDTs reflect temporal resolution abilities of an individual, and it was

1 found that temporal resolution is significantly poorer in individuals with ANSD. The
2 results are in agreement with all the previous studies (Kraus et al, 2000; Michalewski
3 et al., 2005; Rance et al., 2004; Zeng et al., 1999; 2005) wherein consistent evidence
4 for deficit in temporal processing has been shown. The temporal resolution is
5 important for speech perception both in quiet and in noise. It helps the individual to
6 perceive the modulations in speech, helps in segmentation and deriving speech related
7 cues in the presence of noise. Therefore, deviant temporal resolution abilities in these
8 individuals are likely to result in speech perception deficits.

9

10 Poor temporal processing and speech perception is likely to negatively
11 influence the auditory feedback of these individuals. This is true both in cases of
12 listening to others' speech and listening to their own speech. It is important to note
13 that the speech perception deficits in these individuals is a lot more in severity than
14 what could be expected of their hearing thresholds. Therefore, it is expected that the
15 distortion in the auditory feedback in these individuals is much more than that of
16 cochlear pathology.

17

18 **5.2 Speech production of individuals with ANSD**

19 Earlier studies have shown that perception of speech in individuals with
20 ANSD improves with temporal enhancement (Narne & Vanaja, 2008a, 2008b). When
21 the cues of speech such as burst and transition were increased in their duration, or
22 when the temporal envelope was enhanced, it was found that the speech identification
23 and its accuracy improved (Narne & Vanaja, 2008b). Based on these findings, one can
24 expect that there would be compensatory modifications in the speech of ANSD in
25 order to facilitate correct feedback of their own speech. In a preliminary study, Dayal

1 and Maruthy (2009) found deviations in the speech production characteristics of
2 individuals with ANSD. However, they did not characterize it in terms of the acoustic
3 measures of speech. Therefore, in this study, an attempt was made to characterize the
4 speech both perceptually and acoustically.

5
6 The study hypothesized that long-standing speech perception deficits could
7 result in speech production deficits as in case of cochlear hearing loss (Culbertson &
8 Kricos, 2001; Dunn & Newton, 1986; Hudgins & Numbers, 1942; Smith, 1982). The
9 results of the study revealed that speech production characteristics of ANSD are
10 deviant compared to individuals with NAA, both for vowels and consonants.
11 However, the extent of deviation observed was more for consonants.

12 13 **5.2.1 Segmental characteristics of speech in ANSD**

14 Analyses of vowel production revealed significant differences for spectral
15 measures between males and females in both ANSD and NAA groups. Gender
16 differences observed are attributed to the differences in the vocal tract characteristics
17 of males and females (Pèpiot, 2015; Simpson, 2009). In males, the ANSD group had
18 significantly lower F_1 (for /a/, /ɪ/ & /ʊ/), F_2 (for /ɪ/ & /ʊ/), and F_2 bandwidth (for /a/).
19 On the contrary, among the females, those in the ANSD group had significantly
20 higher F_0 (for /a/ & /ʊ /), and F_1 bandwidth compared to NAA group. As stated
21 previously, studies on speech production characteristics in ANSD are sparse.
22 However, literature on individuals with cochlear hearing loss provides evidence of
23 deviant spectral characteristics when compared to normal hearing individuals
24 (Culbertson & Kricos, 2002; Dunn & Newton, 1986). The researchers have attributed
25 deviant production to the deficits in perception and auditory feedback. The present

1 study also reports similar trend in ANSD group which could be attributed to the
2 disrupted auditory feedback in these individuals.

3

4 In case of plosives, individuals with ANSD significantly differed from
5 individuals with NAA on temporal measures like Voice onset time (VOT), Burst
6 duration (BD), extent of transition (EoT), and speed of transition (SoT). Though there
7 are limited studies reporting deviant acoustic characteristics in the speech of
8 individuals with ANSD, there exists a vast body of literature reporting significant
9 deficits in their perception. To reiterate, individuals with ANSD are reported to have
10 relatively greater deficits in temporal processing when compared to spectral
11 processing. A study by Kumar and Jayaram (2006) revealed increased just noticeable
12 differences in VOT, BD and TD in individuals with ANSD. Based on these findings,
13 it is speculated that long standing temporal processing deficits could be reflected as a
14 distortion or disruption of the temporal measures of speech such as VOT and BD.
15 These findings are in consensus with the findings of Dayal and Maruthy (2009)
16 reporting lengthened temporal cues in the speech of individuals with ANSD. The
17 findings suggest that individuals with ANSD exhibit increased temporal measures of
18 speech as a compensatory strategy to perceive their own speech better.

19

20 Another set of sounds considered was fricatives and the findings of the study
21 revealed significantly longer transition duration (TD) of /f/, and significantly shorter
22 frication duration (FD) of /f/ in the ANSD group compared to NAA group. These
23 findings also support the deviations of speech production in ANSD.

24

1 On comparison of the three classes of speech sounds considered in the present
2 study, it was found that more number of measures were deviant in plosives when
3 compared to vowels and fricatives. This could be due to the transient nature of
4 plosives. As discussed earlier, individuals with ANSD are known to have significant
5 temporal processing deficits. In such instances, perception of plosives is more prone
6 to disruption when compared to vowels and fricatives which are longer in duration.
7 Speech perception based studies in ANSD have consistently revealed plosives to be
8 maximally difficult compared to other classes of speech sounds (Narne et al., 2015).
9 Considering that the consonants are more dynamic in nature, one can assume that the
10 distorted auditory perception found in ANSD has greater negative influence on the
11 dynamic phonemes than the static phonemes. Perceptually, individuals with ANSD
12 showed more deviance in consonants. Greater deviation in the production of
13 consonants hints at the direct relationship between perception and production.

14

15 ***5.2.2 Voice characteristics in ANSD***

16 The voice was characterized perceptually as well as acoustically in the present
17 study. On the perceptual scale, it was found that all individuals in NAA group were
18 rated normal on all the parameters of CAPE-V. The samples of the NAA group and
19 the ANSD group were randomly presented to the listeners, and the findings are true in
20 spite of the listeners being blinded to the samples being presented. The deviations
21 were observed in Roughness, Strain and the overall severity in both males and
22 females with ANSD. Additionally, in males, the deviations were also found in the
23 pitch of the voice. The findings are in agreement with **Maruthy, Rallapalli, Shukla, &**
24 **Priya (2019)** who reported deviations in Roughness, Strain and Breathiness of the
25 voice in a different group of 11 individuals with ANSD. However, from the present

1 findings, it is not plausible to speculate whether these deviations are secondary to the
2 reduced hearing sensitivity or compromised auditory processing or both.

3

4 The acoustic analysis of voice revealed that individuals with ANSD have
5 higher F_0 range, I_0 range, jitter and shimmer in their voice compared to the NAA
6 group. These findings reflect poor control of voice, probably attributed to the
7 compromised auditory feedback. The deviations in the voice observed in the
8 perceptual analysis could be partly explained by the deviations in F_0 range and jitter.

9

10 ***5.2.3 Suprasegmental characteristics of speech in ANSD***

11 Analysis of emphasis production characteristics revealed significantly longer
12 D_0 in females of ANSD group compared to the NAA group. This was true for both
13 with-emphasis and without- emphasis conditions. This suggests that individuals with
14 ANSD are prolonging the emphasis on a particular utterance which could be probably
15 to facilitate the feedback of the emphasis intended.

16

17 The findings also revealed deviant speech rhythm characteristics in individuals
18 with ANSD when compared to the individuals in the NAA group. These findings are
19 objective evidence to the preliminary investigations of Dayal and Maruthy (2009)
20 who reported deviant prosody based on the perceptual ratings of the speech of
21 individuals with ANSD. The peak amplitude of the modulation spectra, energy in 0-4
22 Hz and 4-10 Hz region, and the energy ratio was found to be higher in the speech of
23 individuals in ANSD group compared to the NAA group. The deviant peak amplitude
24 and the amplitude in different frequency bands reflect the deviant rhythm
25 characteristics in individuals with ANSD. However, the specific deviations within

1 rhythm cannot be derived from the present findings. In a recent investigation, Priya,
2 Seth, and Maruthy (2018) reported lengthened temporal cues as characteristic feature
3 of speech of ANSD at the segmental level. However, the pattern of variation of
4 amplitude spectra across the frequency bands remained similar between the two
5 groups. This suggests that the rate of speech was unaltered in individuals with ANSD.
6 The lack of significant difference in the peak frequency of envelope spectra is an
7 additional evidence for this inference. Taken together, the existing evidence indicates
8 lengthened temporal cues without significant alteration in the rate of speech.

9

10 The increase in the amplitude of the envelope spectra in individuals with
11 ANSD suggests that the spectral variations in the amplitude of the envelope are larger
12 in these individuals compared to that of controls. The individuals with ANSD are
13 known to have deficits in processing the temporal modulations and need more
14 modulation depth compared to controls (Kumar & Jayaram, 2005) for perception.
15 Therefore, the modulation spectra are being enhanced in their own utterances,
16 probably as compensatory mechanism to facilitate perception of the self-uttered
17 speech. The findings support closed-loop models of speech production. The
18 deviations in rhythm production observed in the study could be either due to the
19 hearing loss and/or temporal processing deficits. The relative role of the two variables
20 in the resultant rhythm deviations needs to be explored in future studies.

21

22 Dayal and Maruthy (2009) have reported deviations in the prosody of
23 individuals with ANSD. The current findings are in agreement and show absence of
24 intonation in most instances. In instances when the intonation was present, most often
25 it was erroneous. The deviations in the intonation were primarily seen in

1 interrogatives sentences, wherein a falling pattern of intonation was seen instead of
2 rising. The interrogative sentences require more variations in the pitch compared to
3 the declarative sentences, and this could be the possible reason for finding the
4 deviations primarily in interrogative sentences. Poor control of the vocal parameters,
5 as found in the acoustical analysis of voice, may have contributions to the poor
6 intonation patterns observed. The poor intonation patterns observed is likely to make
7 the speech of ANSD sound less natural and hinder the effective communication of
8 their emotions or the intent.

9

10 Overall, speech production characteristics of individuals with ANSD reflect
11 poor control of the vocal parameters, prolongation of the temporal characteristics of
12 speech, deviations in the segmental and suprasegmental aspects. The observed
13 characteristics, although, have many deviations in common with that of adventitious
14 cochlear hearing loss, they are not totally same. **This warrants a detailed assessment
15 of speech characteristics in individuals with ANSD at regular intervals, and early
16 intervention, if deviations are found.**

17

18 **5.3 Relationship between Auditory Abilities and Speech Production in** 19 **Individuals with ANSD**

20 It was in the interest of the present study to statistically verify whether the
21 deviations observed in the speech production characteristics are related to their
22 auditory abilities. The findings showed support for the relationship between the two,
23 i.e. those with poor auditory abilities were found to be more deviant in their speech
24 production characteristics. Such a relationship was found to exist with hearing
25 sensitivity, temporal processing, speech perception in noise, and the characteristics of

1 late latency responses. In general, the vocal parameters, the temporal characteristics in
2 terms of VOT, and speech rhythm were found to be significantly deviant in
3 individuals with poor auditory abilities compared to those with better auditory
4 abilities. Earlier studies (Dayal & Maruthy, 2009; Maruthy et al., 2019) have also
5 reported significant relationship between perception and production attributes.
6 However, the current study projects a lot more detailed analysis of both perceptual
7 and speech production attributes compared to the previous studies.

8

9 It is important to note that the relationship between auditory abilities and
10 speech production was verified in two different ways. First, by comparing between
11 individuals with good and poor auditory abilities, and second, through correlation
12 analysis. Both kinds of statistical analyses support a significant relationship between
13 the two. Taken together, the findings suggest that poorer the auditory abilities, more
14 deviant the speech production characteristics are likely to be. These results support
15 the closed loop models of speech production highlighting the importance of auditory
16 feedback and its role in speech production.

17

CHAPTER 6

SUMMARY AND CONCLUSIONS

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3
4 Individuals with Auditory Neuropathy Spectrum Disorders (ANSD) are
5 known to have speech perception poorer than what could be accounted by their
6 hearing sensitivity. Deficits in temporal processing are known to be the primary
7 reason for their poor speech perception. Therefore, one can expect speech production
8 characteristics to be deviant and unique in these individuals compared to those with
9 adventitious cochlear hearing loss. Hence, the primary aim of the study was to profile
10 the speech production characteristics of individuals with ANSD, and assess its
11 relationship with their auditory abilities.

12
13 Thirty individuals diagnosed to have ANSD participated in the study. They
14 were assessed for their auditory abilities in terms of hearing sensitivity, speech
15 perception (in quiet & in noise), gap detection thresholds, and late latency responses.
16 Their speech production characteristics were profiled in terms of segmental and
17 suprasegmental aspects. Segmental aspects included acoustic analysis of vowels,
18 plosives, fricatives, and voice characteristics while suprasegmentals included
19 emphasis, rhythm, and intonation. The auditory abilities and the speech characteristics
20 of individuals with ANSD were compared with those of individuals with normal
21 auditory abilities (NAA). Attempts were also made to statistically analyze the
22 relationship between auditory abilities and speech production characteristics of
23 individuals with ANSD.

24

1 Results revealed significantly poorer auditory abilities in individuals with
2 ANSD compared to NAA. The speech production characteristics were deviant in
3 segmental as well as suprasegmental aspects. The temporal cues showed a
4 characteristic prolongation in the speech of individuals with ANSD. The perceptual
5 and acoustic analysis of voice hinted at poor control of the vocal parameters.
6 Deviations were also seen in the parameters of emphasis, rhythm, and intonation.
7 Further, the deviations seen in the speech production were related to the auditory
8 abilities of individuals with ANSD.

9
10 The findings suggest that the compromised auditory processing in
11 ANSD has negative impact on speech production owing to the compromised auditory
12 feedback. The close association of the deviations observed with that of the auditory
13 processing measures, indicate that the deviations seen in speech production cannot be
14 solely attributed to the reduced hearing sensitivity. The findings support the closed
15 loop model of speech production. **This calls for a detailed assessment of speech
16 characteristics of individuals with ANSD at regular intervals. Further, it is
17 recommended to identify and rehabilitate ANSD at the earliest possible to minimize
18 the negative impact on speech production.**

19
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APPENDIX I

TEST STIMULI

I. Segmental aspects of speech

A. Wordlist to assess the acoustic characteristics of vowels

Sl. No.	Target word	IPA
1.	ಕಬ್ಬು	/kəbbu/
2.	ದಪ್ಪ	/d̪əppa/
3.	ಸರ	/səra/
4.	ಕಿವಿ	/kivi/
5.	ದಿಂಬು	/d̪imb̪u/
6.	ಸಿಹಿ	/sihi/
7.	ಕುರಿ	/kuri/
8.	ದುಂಬಿ	/d̪uɒmbi/
9.	ಸುಖ	/sok ^h a/

B. Wordlist to assess the acoustic characteristics of plosives

Sl. No.	Target word	IPA	Sl. No.	Target word	IPA
1.	ಕಾರು	/kāru/	9.	ಆಕ	/āka/
2.	ಗಾರೆ	/gāre/	10.	ಆಗ	/āga/
3.	ಟಾರು	/tāru/	11.	ಆಟ	/āṭa/
4.	ಡಬ್ಬಿ	/d̪əbbi/	12.	ಆಡ	/āḍa/
5.	ತಾರು	/tāru/	13.	ಆತ	/āṭa/
6.	ದಾರಿ	/d̪āri/	14.	ಆದ	/āḍa/
7.	ಪಾರು	/pāru/	15.	ಆಪ	/āpa/
8.	ಬಾರಿ	/bāri/	16.	ಆಬ	/āba/

C. Wordlist to assess the acoustic characteristics of fricatives

Sl. No.	Target word	IPA	Sl. No.	Target word	IPA
1.	ಸರ	/səra/	1.	ಆಸೆ	/āse/
2.	ಶಂಖ	/ʃəŋk ^h a/	2.	ಆಶ	/āʃa/
3.	ಫ್ಯಾನು	/fænu/	3.	ಕಾಫಿ	/kāfi/

D. Reading passage for voice analysis

ಬೆಂಗಳೂರು ನಮ್ಮ ರಾಜ್ಯದ ಒಂದು ದೊಡ್ಡ ಊರು. ಈ ಊರನ್ನು ನಮ್ಮ ರಾಜ್ಯದ “ಬೊಂಬಾಯಿ” ಎನ್ನುವರು. ಇಂಡಿಯಾದ ದೊಡ್ಡ ನಗರಗಳಲ್ಲಿ ಇದೂ ಒಂದು. ಈ ಊರನ್ನು ನೋಡಲು ಜನರು ಬೇರೆ ಬೇರೆ ಊರುಗಳಿಂದ ಬರುವರು. ಇದಲ್ಲದೆ ನಮ್ಮ ರಾಜ್ಯದಲ್ಲಿರುವ ಬೇಲೂರು, ಜೋಗ್, ನಂದಿ, ಇವುಗಳನ್ನು ನೋಡಲು ಜನರು ಬರುವರು. ಈ ನಾಡಿನಲ್ಲಿ ರೇಷ್ಮೆಯನ್ನು ಬೆಳೆಯುವರು.

/beŋgə|ūru nəmma rādʒjəɖɖa oŋɖ u ɖɖoɖɖa ūru/ /i ūrənnu nəmma rādʒjəɖɖa bomb āji
ennuvəru/ /ɪndʒjādɖa ɖo ɖɖa nəgərəgəɖɖəlli ɪɖū oŋɖ u/ /i ūrənnu nōɖəɖu ɖʒənəru bēre bēre
ūruɡəɖɖa b əruvəru/ /ɪɖəɖəɖɖə nəmma rādʒjəɖɖəɖɖəɖɖə bēlūru, ɖʒoɡ, nəŋɖi, ɪvugəɖɖənnu
nōɖəɖu ɖʒənəru bəruvəru/ /i nəɖɪnəɖɖə rēʃmējənnu bēɖjəɖɖəɖɖə/

II. Suprasegmental aspects

A. Noun-Adjective phrases to assess emphasis

Sl. No.	Target word	IPA	Sl. No.	Target word	IPA
1.	ಚಿಕ್ಕ ಅಂಗಡಿ	/ʃɪkka əŋgəɖɖi/	6.	ದಪ್ಪ ಮನುಷ್ಯ	/ɖɖəppa mənuʃja/
2.	ನೀಲಿ ಬಸ್ಸು	/nīli bəssu/	7.	ಕೆಂಪು ಗುಲಾಬಿ	/kempu guɭābi/
3.	ಹಸಿರು ಬೆಟ್ಟ	/həsɪru bətta/	8.	ದೊಡ್ಡ ಮರ	/ɖɖoɖɖa məra/
4.	ಬಿಳಿ ಬುಟ್ಟಿ	/biɭi buɖɖi/	9.	ಕೆಂಪು ಪೆನ್ನು	/kempu pənnu/
5.	ಪುಟ್ಟ ಗೊಂಬೆ	/puɖɖa goŋbɛ/	10.	ಕಪ್ಪು ಶೂ	/kəppu ʃū/

B. Sentences to assess rhythm

i. ಈ ಕಾಲದಲ್ಲಿ ಒಳ್ಳೆ ತಳಿಯ ಕಾಶ್ಮೀರದ ಸೇಬುಗಳು ಮಾರುಕಟ್ಟೆಯಲ್ಲಿ ಸಿಗುವುದು ಕಷ್ಟ ಮತ್ತು ದುಬಾರಿ ಕೂಡ.

/i kāləɖɖəɖɖə oɭɭə ʃəɭɭə kəʃmīrəɖɖə sēbuɡəɖɖə mərukeɖɖəɖɖəɖɖə siɡuvuɖɖə kəʃɪə məɖɖə
ɖubāri kūɖə/

ii. ಊರಿನ ಹುಡುಗಿಯರು ಮನೆಯ ಬಳಿ ಇರುವ ಮರದ ಅಡಿಯಲ್ಲಿ ಕುಳಿತುಕೊಳ್ಳುವರು.

/ūrina huɖuɡiəru mənejə bəɭi ɪruvə mərəɖɖə əɖɖəɖɖə kʊɭɪtuɖɖəɖɖə/

- iii. ಭ್ರಷ್ಟಾಚಾರವನ್ನು ಹೋಗಲಾಡಿಸಲು ಅಣ್ಣಾಹಜಾರೆಯವರು ನಡೆಸಿದ ಉಪವಾಸವು ಜನರಲ್ಲಿ ಭಾರೀ ಪ್ರಮಾಣದ ಜಾಗೃತಿಯನ್ನು ಬೆಳೆಸಿತು.

/b^hrəʃʈrāʃvāraḥaḥarēyavaru naḍesiḍa upavāsava dʒanarəlli
b^hāri pramāṇaḍa dʒāgruḥjāṇnu beḷesiḥu/

- iv. ನನ್ನ ಸ್ನೇಹಿತೆ ಮೊದಲ ಸಂಬಳ ಪಡೆದ ಸಂತೋಷಕ್ಕಾಗಿ ತನ್ನ ತಾಯಿಗೆ ಒಂದು ಸುಂದರವಾದ ಸೀರೆಯನ್ನು ಉಡುಗೊರೆಯಾಗಿ ಕೊಟ್ಟಳು.

/nanna snēhiḥe moḍala s ambaḷa paḍeḍa saṇṭōʃakkāgi ḥanna ḥjāḥige onḍu
suṇḍarāvāda sīreḥannu uḍugoreḥāgi koḥḥuḷu/

- v. ನಾವು ಏಳು ಜನ ಎರಡು ಆಟೋಗಳಲ್ಲಿ ರಾತ್ರಿ ಸಿನೆಮಾ ನೋಡಲು ಹೋದೆವು.

/nāvu ēḷu dʒana eraḍu aḥḥōgaḷḷi rāḥḥi sinemā nōḍalu hōḍevu/

C. Sentences to assess intonation

(a) Interrogatives

- i. ಸೂರ್ಯನ ಸುತ್ತ ಎಷ್ಟು ಗ್ರಹಗಳು ಸುತ್ತುತ್ತವೆ?

/sūrjana sutḥa ēʃḥu grahaḥaḷu sutḥutḥave?/

- ii. ನಾಳೆ ನೀವು ಎಲ್ಲಿಗೆ ಹೋಗುತ್ತೀರಾ?

/nāḷe nīvu eḷḷige hōguttīrā?/

- iii. ಕರ್ನಾಟಕ ರಾಜ್ಯದ ಮುಖ್ಯಮಂತ್ರಿ ಯಾರು?

/karnāḥka rāḍyjaḍa muk^hjamāṇḥri jāru?/

- iv. ನಿಮ್ಮ ತಂದೆಯ ಹೆಸರು ಏನು?

/nimma ḥṇḍeja hesaru ēnu?/

- v. ನಿಮಗೆ ತುಂಬಾ ಇಷ್ಟವಾದ ತಿಂಡಿ ಯಾವುದು?

/nimage ḥumbā ḥḥḥvāḍa ḥṇḍi jāvuḍu?/

Appendix II

Demographic details and audiological findings of participants with ANSD

Sl. No	Age (yrs) / Gender	Duration of loss (yrs)	PTA (dB HL)		SIS (%)		Tymp	Reflex		OAE	ABR	LLR
			R	L	R	L	R/L	Ipsi	Contra	R/L	R/L	R/L
1	31 / M	6	40.00	36.25	35	35	A/A	NR	NR	P/P	NR	P/NR
2	18/M	7	60.00	73.33	CNT	CNT	A/A	NR	NR	P/P	NR	NR
3	34/M	5	22.50	18.75	30	25	A/A	NR	NR	P/P	NR	NR
4	23/M	6	17.50	15.00	35	25	A/A	NR	NR	P/P	NR	P/P
5	19/F	8	41.25	38.75	45	45	A/A	NR	NR	P/P	NR	P/P
6	26/F	14	58.33	76.67	CNT	CNT	A/A	NR	NR	P/P	NR	NR
7	22/F	7	33.70	35.00	65	60	A/As	NR	NR	P/P	NR	P/P
8	26/F	9	36.25	41.25	50	60	A/A	NR	NR	P/P	NR	P/P
9	19/M	8	77.50	58.75	CNT	CNT	A/A	NR	NR	P/P	NR	NR
10	18/F	6	36.25	28.75	35	30	A/A	NR	NR	P/P	NR	NR
11	40/F	10	35.00	38.33	CNT	CNT	As/As	NR	NR	P/P	NR	NR
12	29/F	8	50.00	37.50	55	50	C/A	NR	NR	P/P	NR	P/P
13	19/F	7	18.75	37.50	25	45	A/A	NR	NR	NR/P	NR	NR
14	29/M	5	5.00	21.25	15	25	Ad/A	NR	NR	P/P	NR	P/P
15	23/F	12	28.75	50.00	45	60	A/A	NR	NR	NR/P	NR	NR
16	18/F	7	20.00	11.67	55	50	A/As	NR	NR	P/P	NR	NR
17	27/F	15	90.00	90.00	CNT	CNT	A/A	NR	NR	P/P	NR	NR
18	21/F	8	30.00	26.25	CNT	CNT	As/As	NR	NR	NR	NR	P/P
19	28/F	7	23.33	33.33	25	30	A/A	NR	NR	P/P	NR	P/P
20	18/F	7	21.25	16.25	25	30	A/A	NR	NR	P/P	NR	P/P
21	32/F	9	28.75	27.50	35	30	A/A	NR	NR	P/P	NR	NR/P
22	19/F	6	18.75	48.75	20	70	As/As	NR	NR	P/P	NR	P/NR
23	40/F	14	65.00	36.67	65	40	A/A	NR	NR	P/P	NR	P/P
24	22/F	8	36.20	42.50	45	65	A/A	NR	NR	P/P	NR	NR
25	38/F	5	21.25	23.75	20	15	A/As	NR	NR	P/P	NR	NR
26	20/M	8	56.67	85.00	70	CNT	A/A	NR	NR	P/P	NR	NR
27	25/M	9	28.33	46.67	CNT	CNT	Ad/Ad	NR	NR	P/P	NR	NR/P
28	34/F	7	36.67	48.30	45	55	A/A	NR	NR	P/P	NR	NR
29	34/F	9	31.67	40.00	45	50	Ad/A	NR	NR	P/P	NR	P/P
30	23/M	7	70.00	66.25	75	65	Ad/Ad	NR	NR	P/P	NR	NR/P

Note: yrs-years, PTA-Puretone average, SIS- Speech identification score, Tym- Tympanometry, Reflex- Acoustic reflex, OAE- Otoacoustic emissions, CM- Cochlear microphonics, ABR- Auditory brainstem response, LLR- Late latency response, NR- No response, P- Present, R- Right ear, L- Left ear, Ipsi- Ipsilateral, Contra- Contralateral, F- Female, M- Male, CNT- Could not test.

Glossary

Abbreviation	Expansion
ABR	Auditory brainstem response
AD	Auditory Dys-synchrony
ALLR	Auditory late latency response
ANSD	Auditory neuropathy spectrum disorder
ASHA	American Speech-Language and Hearing Association
BD	Burst duration
CAEP	Cortical auditory evoked potential
CAPE-V	Consensus Auditory–Perceptual Evaluation of Voice
CD	Closure duration
D_0	Mean duration
DIVA	Direction into velocities of articulators
DPOAE	Distortion product otoacoustic emission
EcochG	Electrocochleography
EMS	Envelope modulation spectra
EoT	Extent of transition
F_0	Mean fundamental frequency
F_1	First formant
F_1 BW	First formant bandwidth
F_2	Second formant
F_2 BW	Second formant bandwidth
FD	Frication duration
FRDA	Friedrich's ataxia
GDT	Gap detection threshold
I_0	Mean intensity
IHC	Inner hair cells
JND	Just noticeable difference
MEMR	Middle ear muscle reflex
NAA	Normal auditory abilities

nPVI	Normalized pairwise variability index
OAE	Otoacoustic emission
OHC	Outer hair cells
PTA	Pure tone average
rPVI	Raw pairwise variability index
SINFA	Sequential information analysis
SIS	Speech identification score
SoT	Speed of transition
SPIN	Speech in noise
TB	Tone burst
TD	Transition duration
TEOAE	Transient evoked otoacoustic emission
TMTF	Temporal modulation transfer function
VD	Vowel duration
VOT	Voice onset time
WHO	World Health Organization
