Cortical Auditory Evoked Potentials and the Neural Processing of Speech Stimuli in Cochlear Implant Users

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ABSTRACT

With the current implementation of newborn hearing screening in New Zealand, rising numbers of prelingual children receiving hearing aids and cochlear implants are expected. An objective measure for speech perception would be invaluable clinically for these children and other difficult-to-test populations. Cortical Auditory Evoked Potentials (CAEPs) offer insight into detection and processing in the cortices, and the potential for evaluation of hearing aids and cochlear implants (Dillon, 2005; Kelly, Purdy, & Thorne, 2005). Thus CAEPs from speech stimuli have been investigated (Agung, Purdy, McMahon, & Newall, 2006), with the hope that it can be useful in the assessment of speech detection and processing abilities.

The current study recorded CAEPs in response to /m/, /g/, and /t/, spanning the speech frequency spectrum, in adults (part I) and children (part II) from the New Zealand Northern Cochlear Implant programme. The aim was to investigate differences in the neural processing of these phonemes, and their relationship with speech perception tests. The change CAEPs were recorded in the children as they adapted to their implant over 3 months. Repeated measures analyses explored the interactions between effects of stimuli, speech perception, and adaptation to the implant over time, as a function of chronological age and CI experience. Furthermore, CAEPs were recorded with two evoked potential systems to compare artefact minimisation strategies.

Different CAEPs were observed across stimuli for N1 waveforms in the adults and P1 waveforms in the children. The CAEPs for low frequency /m/ was later and larger than the high frequency /t/ stimulus, suggesting neural differentiation between the most spectrally diverse phonemes. Longer P2 latency was correlated with better speech perception, and this warrants further investigation of the relationship between behavioural phoneme discrimination and P2. CAEPs changes over time appear to be larger in younger children, for the high frequency /t/ stimulus. This is supportive of the sensitive period of maximal plasticity and the urgency in early intervention. Future research should consider that age of implantation was joined by other interacting factors such as length of auditory deprivation, deafness characteristics and CI experience in determining P1 maturation.
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INTRODUCTION

The ability to hear across the speech spectrum is imperative for the development of speech and language in a young child. For severe to profoundly deaf individuals today, a cochlear implant referral is made when these goals are no longer achieved with a hearing aid. Cochlear implants (CI) are auditory prostheses which delivers the sound via electrical simulation directly to the auditory nerve. Through appropriate auditory rehabilitation and training, the brain learns to interpret and discriminate the electrical input spectrally and temporally.

There is growing evidence for a sensitive period in the development of the auditory system when neural pathways are maximally plastic. Research suggests that early cochlear implantation prior to the cessation of this period, allows the hearing impaired child to catch up on a stimulation-driven neural development (Sharma, Dorman, & Spahr, 2002b). These early amplification goals have lead to the implementation of universal newborn hearing screening in New Zealand (Kelly, Purdy, Pokorny, Hann, & McNeur, 2010). As a downstream effect, the number of pre-lingual children receiving cochlear implants after unsuccessful hearing aid use is expected to rise.

As with hearing aid fitting in young children and other difficult-to-test populations, prescription of amplification and programming strategies for cochlear implant users can vary widely. Furthermore, behavioural responses can often be unreliable in these populations. Thus an objective measure of the neural encoding and detection of speech sounds from higher auditory centres would be clinically advantageous for cochlear implant users.

Electrophysiological responses to auditory stimulation and its trafficking throughout the neural pathways can be recorded by electrodes placed on the scalp. These are known as Auditory Evoked Potentials which reflect the summation of voltage differences across the auditory pathway during the relay of the auditory signal. Over the past decade, Cortical Auditory Evoked Potentials (CAEPs) have increased in popularity as growing evidence suggest its potential use for evaluation of hearing aids and cochlear implants (Dillon, 2005; Kelly, et al., 2005). CAEPs offer insight into detection and processing in the cortices, and can also be recorded from speech stimuli. Thus allowing a more complete depiction of the signal arriving in the higher order neural processes, spectrally and temporally.
The seven Ling sounds, representative of the speech frequency spectrum, have elicited different CAEPs in normal hearing adults (Agung, et al., 2006). This has lead to the belief that CAEPs may have the capacity to reflect complex neural signalling during speech discrimination. Variations in the stimulus duration and inter-stimulus intervals have also demonstrated significant differences in their cortical responses (Beukes, Munro, & Purdy, 2009). Accumulating evidence for phonemes /m/, /g/, and /t/, with spectral energy concentrations in low, mid, and high frequencies respectively, have been identified to reliably elicit CAEPs in young infants (Golding et al., 2007; Purdy & Gardner-Berry, 2009). This study undertakes further investigations to understand the processing of these stimuli through cochlear implant in adult and child users as this objective test may be invaluable to both populations. Indisputably, this objective verification of speech detection and perception would also be highly assistive throughout the journey of rehabilitation for CI users.

CAEP recordings from cochlear implant users face a unique but incessant problem of artefact contamination. Previous work on minimization of the stimulus-associated artefact has been promising, although impractical for clinical use. Friesen and Picton (2010) have recently employed a subtraction method which utilises a deduction of waveforms elicited via stimuli with varying inter-stimulus intervals (Friesen & Picton, 2010). This study also seeks to investigate the clinical usefulness for this method, and the characterisation of artefact morphology form recordings in CI users.
CHAPTER 1 REVIEW OF LITERATURE

1.1 Cochlear implants

Cochlear implants are currently one of the most popular options for people with bilateral profound deafness. The purpose of the cochlear implant can be very different for users: many seek a device which provides adequate speech perception abilities, while some rely on the environmental acoustic cues it provides for sound awareness. Nonetheless, the impact on the user is valuable and continues to improve as the technology advances.

The technology and designs have always been driven by research and industrial intentions. Four main companies produce cochlear implants for clinical use to date, each having collaborated with research at some stage. In 1961, two deaf adults reported auditory and tonal perception after a single-channel stimulation device implanted by House and colleagues (Fretz & Fravel, 1985) in the USA. Throughout the 1970s, numerous people were implanted by this single-channel device which was marketed as the 3M/ House implant at the time. The University of California in San Francisco also began developing single and four channel analogue devices in the early 1970s. This device was not successfully commercialized by the research group, but was the basis for the Clarion cochlear implants which are produced by Advanced Bionics Corporation today.

During the 1970s, the first multi-electrode device was successfully implanted by Graeme Clark, whose research saw the rapid development of speech processing technologies which enabled much improved speech discrimination in many users. In 1995 Cochlear™ stemmed from this Nucleus Limited device, and became one of the largest providers, enveloping several other multi-electrode and research designs of the bionic ear (Patrick & Clark, 1991). Currently, all government-funded cochlear implants in New Zealand are Nucleus® devices from Cochlear™. The recipients are therefore users of this family; and have external parts belonging to the ESPrit™, Freedom™ and CP510 series, and internal parts from the Nucleus 22, 24 and 5 series.
1.1.1 Hardware components of a cochlear implant

The modern implant consists of an external and an internal component which are connected via percutaneous magnets. The internal components are surgically inserted, while the external parts are usually worn and maintained similarly to a behind-the-ear hearing aid. As technology develops and new devices are introduced, users may decide to upgrade the external part, capable of adapting to older technology without re-implanting the internal part (Patrick, Busby, & Gibson, 2006). Figure 2 below shows the external and internal components from the Freedom™ and Nucleus 24 family widely used today.

The main goal of the external CI is to process the acoustic stimuli into digital signals which can be filtered and processed to be encoded as electrical stimuli. It also acts as a source of power supply for the entire device. The behind-the-ear controller comprises of the receiver, digital signalling processing units (DSP), amplifiers, and a battery compartment. The DSP unit is responsible for storage of maps and speech processing strategies which enable the incoming sound to be translated and filtered. The processed signal is delivered to a coil which acts as a radio frequency transmitter situated about the temporal portion of the mastoid, which converts the electrical input into a radio frequency (Patrick, et al., 2006, p. 176).

The internal components are responsible for decoding the radio frequency back to the electrical signal to be delivered along the electrode array which enters the cochlea via either the round window or a cochleostomy. Any flaw along the signal transmission within both external and internal components of the device can result in a poorer degraded signal at the electrical-biological interface (Wilson & Dorman, 2008). Electrical stimulation via the intracochlear array is placed specifically along the tonotopically mapped cochlea; which replaces the frequency-specific populations of hair cells and neurons along the spiral, that are responsible for excitation of the auditory nerve in normal hearing.
1.1.2 Speech processing in cochlear implants

To convey speech signals from the electrode array upon the ascending auditory neural pathway, has always been a challenge. A speech signal typically carries complex characteristics such as voicing, place of articulation, and aspiration which are difficult to translate into electrical information. Encoding thus requires a combination of temporal and spectral components. Spectral translation depends on the electrical stimulation delivered to the specific site along the electrode array, and the summation of inputs across the different channels. Temporal information conveying the timing of signal is regulated by the amplitude of the stimulus through a particular channel at any one time (Cochlear™, 2006). Several variables control the temporal and spectral encoding: total charge, current, pulse width, rate and level of technology (Cochlear™, 2006).
Electrical stimuli are delivered via biphasic phases (see Figure 3); they are comprised of a positive voltage and negative voltage in short succession that is applied over an active and an indifferent or ground electrode. The total charge is represented by the manipulation of both amplitude and width of the 2 pulses. Subsequently, the pulse width dictates the amount of time which current flows through the channel. This is coupled with the pulse rate which is number of stimuli delivered in one second per channel (pps/ch) (Cochlear™, 2006).

Audiologists are able to manipulate parameters via the fitting software. The effects of parametric variables on speech recognition and understanding has been previously reported (Loizou, Poroy, & Dorman, 2000). Loizou et al. (2000) reported that a wider pulse width had a positive effect on consonant recognition, while higher stimulation rates were found in those who performed better overall in speech recognition. However, the effects seen in these signal parameters can vary across devices, as new features of external and internal hardware, as well as processing strategies are developed.

1.1.2.1 Electrode array

The Nucleus family has adopted the design of an array of banded, platinum electrodes which are encased in a silicone carrier (Cochlear™, 2005). The array is inserted into the Scala tympani and curls towards the apex of the cochlea for approximately one and a half turns (Wilson & Dorman, 2008). The Nucleus® CI 22 device (Patrick & Clark, 1991) was the very
first commercialized implant consisting of 22 electrodes which all positioned within the cochlea. In 1998, the Nucleus® CI24 series was introduced with additional electrodes positioned outside the cochlea, and a ‘pre-coiled’ electrode array logarithmically spaced along the scala tympani, which enabled optimal stimulation to the auditory nerve fibres (Patrick, et al., 2006). The additional extracochlear electrodes comprised of a plate electrode attached to the internal receiver coil casing, and a ball electrode extended via a lead wire from the extra cochlear array and positioned beneath the temporalis muscle. They play an important role in introducing a monopolar mode of stimulation, which offers advantages in mapping and programming the speech processor.

1.1.2.2 Modes of stimulation

Three modes of stimulation currently exist for internal implants of the Nucleus family. The original Nucleus 22 device (CI22M) was capable of both common ground and bipolar stimulation, whilst monopolar stimulation is only achievable with the Nucleus 24 series (CI 24M and CI 24RE) and the newest Nucleus 5 addition.

**Bipolar stimulation (BP)**

Bipolar stimulation is achieved via pairing of electrodes either adjacent or close to one another. The most basal of the pair is named the active electrode; and the other termed the indifferent or ground electrode. The current path flows between the active and indifferent electrode and produces electrical stimulation based on the dynamic range between C (comfortable) and T (threshold) levels, influenced by the amount of current flow per electrode (Cochlear™, 2006). Subjective loudness can be increased by selection of electrodes further spaced apart. BP+1 is commonly used in bipolar mode, indicative of one electrode between the active and indifferent pair (Brown, Geers, Herrmann, & Iler Kirk, 2004).

Early studies of bipolar stimulation in cats (Van den Honert & Stypulkowski, 1984) suggested that it allowed activation of auditory fiber groups with good specificity: an important goal in electrical stimulation as it attempts to replace the tonotopic mapping of frequencies, achieved by the travelling wave along the basilar membrane of the cochlea in normal hearing individuals. Bipolar stimulation is also non-simultaneous, which reduces deleterious effects from inter-channel interactions (Brown, et al., 2004).
Common ground stimulation (CG)

The common ground mode of stimulation is often offered as a safer option for use in young children, it was considered they could reliably report discomfort from loud stimulations in the incidence of electrodes shorting together. Common ground stimulation is achieved by designating one electrode active and all other 21 electrodes indifferent, and thus the return path of current flow. This configuration prevents loudness discomfort as no stimulation will occur when a single electrode shorts (Brown, et al., 2004).

Monopolar stimulation (MP)

The birth of the Nucleus 24 series in 1997, introduced additional extracochlear ball and plate electrodes to the existing 22 intracochlear electrodes. The extracochlear electrodes are configured as ground electrodes while the other 22 intracochlear electrodes can all act as active electrodes. The current flows from an intracochlear electrode to an extracochlear electrode, providing a wider field of excitation due to the extended current return path (Ryan, Miller, Wang, & Woolf, 1990; van den Honert & Stypulkowski, 1987).

Currently, monopolar stimulation has surpassed bipolar in popularity due to several advantages. Using a specially designed implant which could stimulate via both modes, it was demonstrated that with monopolar stimulation, one is able to achieve the same threshold and comfort levels in the CI Map as with bipolar stimulation, using lower current levels (Zwolan, Kileny, Ashbaugh, & Telian, 1996). Consequently, this reduces overall power consumption of the processor. Many have also debated whether the broad field of excitation could provide as many localized specificity as bipolar mode. Zwolan et al. (1996) indicated no significant difference in five measures of speech recognition scores, as well as the participants reporting favourably towards monopolar mode in terms of comfort. However, this initial study was limited by a small population of 6 participants. Others have found improvements in speech recognition scores with stimulation from wider electrode configurations, although the region of stimulation along the tonotopically mapped cochlea appear to play a crucial role (Pfingst, Franck, Xu, Bauer, & Zwolan, 2001; Pfingst, Zwolan, & Holloway, 1997). These findings suggest that place specificity is not an impairing factor for monopolar stimulation, as other considerations of electrical stimulation are also implicated.
1.1.3 Speech processing strategies

The speech processor plays an important role in applying algorithms and techniques to convey frequency, timing, and intensity information into the radio frequency signal transmitted through the skin, delivered to the electrodes. Processing a single stimulus via many channels may provide poorer information and degrade the overall performance (Brown, et al., 2004). Thus, varying processing strategies have been developed with the aim of improving efficiency of stimulus delivery especially for speech perception.

1.1.3.1 Feature extraction

Prior to 1994, a type of processing strategy known as feature extraction was the only one available to all CI users implanted with Nucleus devices (Cochlear™, 2006). This early strategy involved extraction of the fundamental frequency (F0) and amplitude, and frequency information of the first two formants of speech (F1 and F2). It was thought that these key patterns of speech could appropriately encode the signal. But the effectiveness deteriorated in the presence of background noise.

1.1.3.2 Filterbank technology

To achieve higher resolution in temporal and spectral encoding, filterbank processing strategies were developed in the 1990s. Filterbanks are arrays of band-pass filters which encode for presentation of the signal to the 22 separate electrode channels of the CI. The array of 22 filterbank outputs operates continuously in \( n \) number of parallel signals. The processing strategy will then sample and select the filter outputs, allowing delivery to the internal implant via the correct channel at the appropriate time and sequence; as the implant can only stimulate channel one at a time. Figure 4 below shows the sequence of processing with filterbank technology.

Filterbanks are further influenced by the mode of stimulation and available electrodes which can be programmed for stimulation. For example, the number of channels in use with bipolar stimulation is affected the distance between adjacent electrodes. Partial electrode array insertions or inactive electrodes also alter the pattern of stimulation due to channel availability.
Speech processing strategies currently used are all based on the filterbank techniques; strategies differ in the number of band-pass filters used and subsequently sampling rate, and intensity of stimulation which is applied to each channel. Three filterbank strategies have been adopted by the Nucleus devices and are discussed further below.

![Diagram](image)

Figure 4: Sequence involved in filterbank processing. The incoming signal is sampled (1) and constantly passed through the band-pass filters (2). Twenty filters (3) provide a sample of the output across the frequencies (4). Of the output sample, a number of maxima with highest energy are selected to be delivered to the implant electrodes (5). Stimuli is delivered as biphasic pulses through these electrodes (6) (Cochlear™, 2006, p. 14).

**Spectral Peak (SPEAK)**

The SPEAK or Spectral Peak strategy was introduced by Cochlear Corporation in 1995 for the CI 22 M implant (Cochlear™, 2006). It was designed with the aim of shifting emphasis onto the spectral component of the signal. This is accomplished by dividing and analysing the acoustic spectrum of the incoming signal and identifying 6-10 peaks from the spectral bands with the highest energy levels known as ‘maxima’ (m). The signal delivers stimulation to these selected bands in a particular sequence. To conserve the maximum rate of stimulation across the electrodes, this strategy employs these 6-10 bands as the only bands available for stimulus presentation at any one time. Thus with 6 channels maxima, the filterbank samples and delivers the stimulation at a rate of 250 Hz (Cochlear™, 2006).
Continuous Interleaved Sampling (CIS)

Continuous Interleaved Sampling strategy was originally developed for the Ineraid implant which had 6 intracochlear electrodes which used 6-band filterbank strategies. With the small number of electrodes, it presented with poor spectral resolution (Brown, et al., 2004), but aimed to provide higher stimulation rates to emphasise the temporal cues. The Nucleus devices allow stimulation through 6-8 channels, with a high rate pulsatile stimulus of 900-2400 Hz (Cochlear™, 2006). The disadvantage of the CIS strategy remains that not all 22 electrodes and channels are used. Several studies have found mixed results assessing speech perception with the strategy. An early study found immediate open-set speech perception improvements after one week with a CIS strategy compared to an old compression analogue processor (Wilson, Finley, Lawson, Wolford, & Zerbi, 1993). Another group reported contradictory evidence that the high stimulation rates from CIS processors do not significantly result in improved speech perception in Clarion and Nucleus CI users programmed with CIS strategies (Friesen, Shannon, & Cruz, 2005). They suspected that high temporal resolution does not infer improved extraction of speech cues from phoneme, word and sentences. Wilson et al. (1993) also noted that trade-offs between aspects of the filtering, duration of the pulses, and pulse rate had to be adjusted to obtain the optimal setting for each individual user.

Advanced Combination Encoder (ACE)

The most recent filterbank strategy was introduced with Nucleus 24M devices, which provided the clinical flexibility of selecting the specific number of spectral peaks (n) and band-pass filters (m) sampled (Brown, et al., 2004). ACE or Advanced Combination Encoder attempts to combine the best qualities from previous strategies; it has more channels than SPEAK, and a higher total stimulation rate than CIS. Between 8-12 spectral speaks are selected from the acoustic signal and divided into 22 band-pass filters. It supports up to a maximum stimulation rate of 14400 Hz, although majority of CI users comfortably utilise rates between 900-1200 Hz (Cochlear™, 2006).

Studies have shown improved open-set perception of words in quiet and noise for implanted children after transferring from SPEAK to ACE strategies (Psarros et al., 2002). However, it should be noted that learning and experience are difficult to control for in such studies.
Conversely, it has been shown that increases in the stimulation rate, as proposed by the ACE strategy, introduced no significant improvements in CNC word tests and CUNY sentences (Vandali, Whitford, Plant, Clark, & Graeme, 2000). It was suggested that individual preferences varied widely and should be an important consideration for altering CI parameters with any processing strategies. The reduced significance of temporal aspects of processing strategies further implicates the importance of spectral encoding with channel technology in CI users.

1.1.4 Electrograms

The 2-dimensional Electrogram is a graphical display of the output after the stimulus has passed through all the external and internal circuitry. These are accomplished by capturing the transmitted radio frequency signal from the speech processor and decoding through a dual processor interface (Wai, Bögli, & Dillier, 2003). Analysis is made via a software called sCILab, with varying interface cards for the appropriate processor or device. As depicted below in Figure 5, the 2D Electrogram displays the distribution of spectral information across the 22 intracochlear electrodes on the y axis. The x-axis exhibits a time measure of the signal; and thus spectral information as a function of temporal output. Furthermore, the electrogram provides intensity or acoustic level of the signal by a colour-coded scale (Wai, et al., 2003). Electrograms have been used to study processing strategies, parameters, and the electrode activation patterns of complex stimuli such as speech.

Figure 5: Example of 2D Electrogram made in sCILab of the German word “beissen” processed through an ESPrit 3G processor with SPEAK strategy at 250 Hz. (Wai, et al., 2003, p. 390).
1.1.5 Electrical stimulation of the auditory nerve

Even with advancements in various processing features, the most useful strategy for a CI user is training their auditory system to identify and understand the features of the incoming artificial signal. Successful CI users are fine examples of how the auditory system has the ability to depict and decode an electrical signal; processing its characteristics both temporally and spectrally. Currently, CI designs still face challenges in improving the imitation of physiological processes which occur in the normal hearing pathways.

In the normal hearing individual, spectral encoding of the signal begins with the tonotopically-mapped basilar membrane. Tonotopy has been established throughout the auditory pathway, describing the spatial organisation of neuronal populations grouped accordingly to the frequency spectrum (von Békésy, 1960). Frequency discrimination primarily occurs as place-specific hair cells depolarise to specific frequencies and initiates the excitation towards the nerve fibre, which maintains the tonotopy. The stimulation from the CI electrodes follows this ‘place principle’ in a fairly similar matter (Pickles, 1988). Dependent on the type of device, current multi-channel implants have between 6-22 intracochlear electrodes positioned from basal to apex of the Scala tympani; approximately situated near the spiral ganglion cells of the modiolus (Klinke & Hartmann, 1997). Spectral encoding is dependent on the number of channels available in processing strategies as described earlier. There are some suggestions that stimulation below 1 kHz may be disadvantaged due to the positioning of the inserted electrodes (Clark et al., 1987). However, others have suggested that this may not be significant as the electrodes are situated within the highly conductive perilymph (Cohen, Saunders, Knight, & Cowan, 2006). Issues arise in cases where the site of activation from more than one channel overlaps and causes inter-channel interactions which may confuse the ascending signal. It has been estimated that there are only approximately 4-8 sites where the neuronal population can be activated without overlap (Wilson & Dorman, 2008). Thus the focus of processing designs have been exercised over improving the efficiency of the channels involved instead of increasing number of channel for activation.

Temporal information is translated via the phase locking principle in normal hearing individuals (Pickles, 1988); in which excitation or firing of the auditory fibres concurs with the same phase from the stimulus. Speech processors sample the incoming signal, and extract
the change in sound over time, known as an *envelope*. The sampled temporal information is processed and delivered as phase-locked pulses through the electrodes. Thus CI stimulation provides more temporally accurate encoding than physiological activation of the auditory nerve in normal hearing. However, it is unclear if increased peripheral synchrony is adopted and interpreted well by the central auditory processes. Klinke and Hartmann (1997) even postulated that the introduction of noise into some of the processed stimulation may portray a more realistic speech signal. Various studies have found mixed findings on effects increased rates of stimulation, on precision of the temporal information delivered. Contrary to findings from Vandali et al. (2000), improved speech perception in users have been reported in Nucleus CI 24 users with a stimulation rate of 1800 Hz (Holden, Skinner, Holden, & Demorest, 2002). However, an adaptation effect of a refractory period in between activation in the nerve fibres must be considered prior to making clinical decisions. The amount of charge or amplitude of the biphasic pulses indicates the encoding of loudness into the signal. Therefore, the fine-tuning of CI maps will set the dynamic range of individual CI users between their aided threshold (T) levels and comfortably loud (C) levels (Cochlear™, 2006).

Temporal and spectral encoding should be considered simultaneously with more analyses of specific aspects of speech in processing strategies. It has been suggested that spectral and temporal resolutions can be traded between one another to some degree (Nie, Barco, & Zeng, 2006). Nie, Barco and Zeng (2006) found that increased emphasis on temporal information resulted in higher scores for consonant recognition tests, while vowel perception improved with more spectral emphasis in programming. Clinically, these are important considerations when applying to individual needs of the CI user. Practical factors such as depth of insertion, electrode-nerve interface, integrity of the auditory fibres also have a huge implication in the processing of the signal as well (Wilson & Dorman, 2008). More research is required in this field to further understand how processing strategies combine with physiological factors to produce the variance in CI performance. These are important considerations as the pattern of activity evoked at the level of the auditory nerve has an impact on the activation in higher auditory centres, and their subsequent interpretations and reactions to the signal.
1.2 Speech perception in CI rehabilitation

1.2.1 Language acquisition and development of speech perception

Children with severe to profound congenital hearing loss are faced with the dual task of not only interpreting the novel electrical stimulation, but also utilising the cues to develop spoken language. Svirsky and colleagues (2000) have established that language acquisition and development of an oral linguistic system can be largely attributed to implantation at the appropriate time in children (Svirsky, Robbins, Kirk, Pisoni, & Miyamoto, 2000). Language abilities were assessed by the expressive language section of the Reynell Developmental Language scales (Reynell & Huntley, 1985) in 70 profoundly deaf children before and after their implant. Results demonstrated that although expressive language is delayed in CI children compared to normal hearing children, their abilities are refrained from further deterioration with the implant. Furthermore, earlier implantations resulted in proportionally shorter delays in language development (Svirsky, et al., 2000).

The same theme was replicated from a longitudinal study with children grouped by age of implantation prior to 4 years of age (Colletti, 2009). It was suggested that children fitted with implants as early as 3 years may already have increased difficulty in catching up with age-matched normal hearing peers; an effect which becomes worse as age of implantation increases. These findings are consistent across the literature; even with long term usage and experience duration with CI, those children with delayed implantation may never achieve speech perception outcomes comparable to those implanted earlier (Harrison, Gordon, & Mount, 2005; Nicholas & Geers, 2007). Nicholas and Geers (2007) assessed spoken language skills of children who were implanted before 3 years using the preschool language scale. Their findings were also consistent: to achieve age-appropriate spoken language by 4.5 years prior to starting school, children need to be implanted within the first 12-18 months regardless of residual hearing (Nicholas & Geers, 2007). Further longitudinal research is needed to investigate if these effects of early intervention continue to progress and benefit the young CI user throughout their schooling years.

The evidence surrounding early identification and early implantation, has initiated research surrounding specific factors which heavily impact the development of language in CI
children. Gordon and colleagues identified factors in children with no open-set speech perception after two years of experience with their implant (K Gordon, Daya, Harrison, & Papsin, 2000). Concordant with previous studies, the significant pre-implant factors were age of implantation and duration of deafness. Post-implantation issues identified with significance included technical issues with device programming, integrity of the implant, and rehabilitation management (K Gordon, et al., 2000). The appropriate therapy for an adequate period post-implant and school placements were indicated to be an important factor for their data set. Unfortunately these analyses were only from five children, and a larger sample may have revealed the strength of these issues further.

As identified by Gordon et al. (2000), appropriate therapy post-implant is a crucial component to any CI programme. Auditory verbal therapy (AVT) is a widely adopted approach to train the user to rely solely on their sense of audition; it is also important for the learning processes involved to make sense of the novel stimulus. Auditory training generally encourages the discrimination between similar phonemes and syllables. More research into language-scaled effects of auditory training is required. Currently, it is difficult to generalize that improvements of small scale phonemic and word discrimination, will directly relate to improved perception in the context of sentences and general stimuli across verbal language (Tremblay, 2007).

1.2.2 Measures of speech perception in CI users

The variety of current uses for a cochlear implant results in a wide range of performance abilities in CI users. Speech perception is commonly used to gauge the performance of the user before and after implantation, and track changes through their rehabilitation. One of the main challenges in the literature is that CI programmes assess speech perception through a wide range of tests. From infancy to adulthood, no single test is adequate in the accurate judgement for all CI users. Gifford, Shallop and Peterson (2007) assessed speech perception in 156 postlingually deafened CI users with a battery of open, closed-set, monosyllabic words and sentences tests, both in quiet and in background noise (Gifford, Shallop, & Peterson, 2008). They made strong suggestions that as CI programmes age, there is a need for speech testing materials to be more difficult as ceiling effects from some clinical populations may influence the usefulness of speech perception tests per individual CI user.
As post-operative performances in CI users gradually become more reliable and successful, a minimum speech test battery (MSTB) was identified by specialised clinicians, and cochlear implant manufacturers (Luxford, 2001). The battery consists of both word and sentence tests with multiple lists. It was important for the materials to be truly different as randomization may result in repetition, which was thought to falsely increase the scores over time. Suggestions were made for CI programmes to use recorded tests in favour of monitored live voice presentations; and to calibrate all testing by scaling test materials on the CD to match a single calibration tone and calibration noise (Luxford, 2001). The New Zealand Adult NCIP currently administers two tests from the MSTB, the Hearing-In-Noise-Test (HINT) with sentences, and the Consonant-Nucleus-Consonant (CNC) word test.

The ceiling effect is a common problem reported in the literature when investigating clinical speech perception measures. HINT sentences can be tested in quiet, and with a fixed level of noise presented at either +10 dB or +5 dB signal-to-noise ratio. This increasing level of difficulty with more background noise was suggested to avoid ceiling effects as CI users improve (Nilsson, Soli, & Sullivan, 1994). HINT in quiet has been suspected to commonly reach the ceiling effect; with reports such as 28% of the participants scoring 100% in the quiet condition (Gifford, et al., 2008), and 49% of the participants scoring above 80% in another study (Firszt et al., 2004). Although reducing the SNR allows more room for improvements from the CI user, it also simultaneously renders the battery to be devoid of an open-set sentence test in quiet (Gifford, et al., 2008).

Conversely, the potential of the ceiling effect is not commonly reported for CNC word recognition tests (Firszt, et al., 2004; Gifford, et al., 2008). CNC words are phonemically balanced within and across each list as modelled by the English language. Furthermore, they are can also be delivered in the context of noise to increase the difficulty of the materials; further avoiding ceiling effects in high performers.
1.3 Electrophysiological measures of the auditory system

In 1929, Berger first recorded ongoing brain activity in humans, and termed this measure of electrical potentials as electroencephalography (EEG). Throughout the following decade, recordings influenced by auditory stimulation were reported by Davis and colleagues (P. Davis, 1939; P. Davis, Davis, Loomis, Harvey, & Hobart, 1939; Hall, 1992). It soon became clear that auditory events or the presence of a stimulus, may elicit distinctive and repeatable recordings in comparison to the background electrical noise. These are termed Auditory Evoked Potentials (AEPs) or Auditory Event-related Potentials (AERPs), when the stimulus used is of an auditory nature. Several subtypes of AEP measures have been developed since; to clinically assess the sites of lesion along the auditory pathway when hearing or physiological pathologies presents itself.

The theory behind far-field recordings is analogous across each type of AEP measure. When electrodes are placed on the scalp, they are capable of recording minute changes in voltage due to cellular signalling. The act of action potentials firing across neuronal populations leads to depolarisation of individual cells. Depolarisation results in two outcomes: a voltage difference is created due to flow of ions across the neuronal cell membrane, and it facilitates an electrical field surrounding the cell with opposing polarity at either ends known as a dipole. The dipole-to-dipole interaction entails a positive to negative direction of current flow from current source to current sink, which orientates the electrical impulse along the nerve fibre (Eggermont, 2007). Both the orientation and size of voltage difference is measured as an action potential. Collectively, action potentials from neuronal groups create a compound action potential (CAP) flowing across different neuronal structures. The impedance across different structures, such as brain matter, interstitial fluids, meninges, and the skull bone are also likely to impact the current flow (Grandori, Hoke, & Romani, 1990). Compound action potentials can be detected as either a near or far field response by close or distant electrodes respectively.
Four components are considered to contribute to the overall generation of the evoked potential (Hall, 1992):

1. Number of cells activated by the ‘event’ of the stimuli

2. Synchronization of the depolarization amongst parallel neuron populations along the pathway

3. Geometric organization of cellular membranes, and consequently their dipole orientation

4. Impedance variance between recording electrode and site of generation as layers of cellular and non-cellular tissues separates them.

Furthermore, the end response is affected by structural physiology as depicted in Figure 6 below, where only structures that support a spatial alignment of neurons orientated in the same direction as the current flow contribute to the evoked potentials measured (Eggermont, 2007). The gyri and sulci of cortical folds will also cause distribution of current source and sinks in varying orientations, generating highly dynamic electrical fields. These implications depict the complexity of recording an evoked potential waveform; single dipole units are too simple to model structures accurately. Therefore current day electrophysiology utilises 3 orthogonal electrode pairs to describe a 3-dimensional Lissajous trajectory (3-CLT), defined by voltage space along the pathway. The 3 dipole pairs summate to provide a hypothetical summary of the spatially coherent displacements of current sources and sinks due to activation (Grandori, et al., 1990).

The movement of the dipole vectors occur with the continuation of an electrical impulse, and their strength and location can be implored as a function of time to provide temporal information.

Figure 6: Direction of dipoles across a neural fold in the cortex. Structural orientation of the fold and layers of resistance endured by the evoked potential recording at the scalp (Grandori, et al., 1990).
Auditory evoked potentials are of great interest clinically as they expose sites of lesion along the auditory pathway which hinders the auditory signal. From the cochlear hair cells to neural cortices, AEP measures have been uncovered to support the diagnosis of lesions and provide an understanding behind mechanisms of the auditory system.

1.3.1 Auditory Brainstem Responses

Auditory brainstem responses (ABR) is a measure which reflects the synchronised firing of the signal through the auditory nerve to portions of the auditory brainstem, found within the first 10-15 milliseconds. Six distinctive peaks characterise the waveform; peaks I-II representative of activity from the auditory nerve, and peaks III-V represent communication between the cochlear nucleus complex, upper lateral lemniscus, and inferior colliculus. It must be noted that numerous structures and associated projections and communications all contribute to the ABR recording (Hall, 1992; Picton, Hillyard, Krausz, & Galambos, 1974). Currently, the ABR plays a crucial role in threshold-seeking audiometry. The test can be used to obtain frequency-specific responses and provides the opportunity to test participants under sedation; beneficial in young children and difficult-to-test populations.

1.3.2 Middle Latency Responses

The middle latency response (MLR), is a projection further along the auditory pathway found approximately 10-60 milliseconds post-stimulus onset. Unlike the ABR, this response is affected by the sleep state. The waves of the MLR arise from deep generations within subcortical regions to provide the 6 known waveforms: No, Po, Na, Pa, Nb and Pb (See Figure 7). Out of the six known waveforms only the Na and Pa components are consistently recorded between individuals. Evidence from intracranial recordings and lesions studies suggests ambiguity surrounding the Pa with P1 of the late latency responses (Hashimoto, 1982). Clinically, the MLR has been used for threshold-seeking as well as further research into central auditory processing. There is great interest in the neural generators of the MLR and its wave component, as they are proposed to provide an insight into primary and/or secondary auditory cortex function, through its associations with the midbrain, thalamus and thalamocortical projections (Pratt, 2007).

For cochlear implant users, a further set of electrophysiological techniques are available. Stimulation can be delivered electrically from an external generator; bypassing the speech
processor and thus eliminating room and environment acoustics. These electrical measures provide a parallel for all AEPs in assessment along the auditory pathway and therefore named acronyms such as eABR or eAMLR.

![Figure 7: Adapted from Picton, Hilyard, Kraus and Galambos (1974); Progression of traces of Auditory Evoked Potentials from auditory brainstem responses to obligatory cortical auditory evoked potentials (Picton, et al., 1974, p. 181).](image)

### 1.3.3 Cortical Auditory Evoked Potentials

Cortical Auditory Evoked Potentials (CAEP), Auditory Late Responses (ALR) or Late Latency Responses (LLR), was reported as early as the discovery of AEPs by Davis and colleagues in 1939 (P. Davis, 1939). Several classifications have been used to further categorise the responses; based on latency and the stimulus. Some have described it as slow and late latency responses, separating the P1-N1-P2 complex with the P300 potentials. More practically, these AEPs derived from the cortices have been divided into *sensory* or ‘*obligatory*’ and *processing-contingent* or ‘*discriminative*’ potentials.

Obligatory CAEPs are described by the P1-N1-P2 complex, and considered obligatory due to exogenous factors of the stimulus, such as intensity and presentation rate. Conversely, the discriminative CAEPs are thought to be governed by the endogenous psychological significance of the stimulus. This is inclusive of the thought of the stimuli itself and attention state. However, this classification system has been controversial as the psychological components may invade sensory potentials and the physical characteristics may attribute to the late latency responses as well (Stapells, 2002).
1.3.3.1 Discriminative CAEPs (MMN and P300)

Discriminative CAEPs are characterised by the P3 or P300 component and the mismatch negativity (MMN) which is produced by an ‘odd ball’ paradigm. The paradigm is elicited when a novel sound is detected against the memory of a stream of similar sounds. The detection of the ‘oddball’ is based on the complex neural encoding system where comparison of the memory and novel stimuli characteristics can be made. The P300 (also known as P3) response refers to the positive deflection at approximately 300ms post-stimulus onset. The P300 response indexes further processing of consciously discriminated sounds, and thus has been heavily studied in attention and memory. MMN has been widely explored for its insight into central auditory processing. Research has further branched into stimuli discrimination, memory, neurodegenerative diseases and attention (B. Martin, Tremblay, & Stapells, 2007). Input from other sensory modalities and complex cognitive processes are also expected to interact with this measure (Alho, Kujala, Paavilainen, Summala, & Näätänen, 1993; Alho, Woods, Algazi, & Näätänen, 1992; Fu, Fan, & Chen, 2003; Kekoni et al., 1997). However, the generation of both P300 and the MMN response are highly dictated by stimulus and factors such as attention. Pekkonen et al. (1995) found that although MMN have fairly adequate intra-participant test-retest reliability, obligatory N1 responses were more reliable (Pekkonen, Rinne, & Näätänen, 1995). Current demands of newborn hearing protocols and clinical practice has thus been favoured towards obligatory CAEPs, especially in children.
1.4 Obligatory cortical auditory evoked potentials

Research in the area of obligatory cortical responses was overshadowed by the clinical usefulness of auditory brainstem responses for much of the 1970s. Popularity grew for the late latency response over the last two decades; it became evident that there is great utility and implication of onset of cortical processing of the stimulus. The obligatory CAEP is characterised by two positive deflections and a negative deflection. The series of waveform is known as the P1-N1-P2 complex, defined by the polarity and order of occurrence of the waveforms (Picton, et al., 1974). Several other later responses such as N2b-P3b and N400 have been characterised, but the P1-N1-P2 complex remains the most widely studied. The P1-N1-P2 complex provides a measure of detection of supra-threshold auditory skills, such as speech processing (B. Martin, et al., 2007). However, debate still lingers around its potential to demonstrate discrimination of auditory input. Studies on discrimination has gradually focused on to use of varying stimuli and especially speech in eliciting another late AEP known as the acoustic change complex (ACC) (B. Martin & Boothroyd, 1999; B. Martin, Tremblay, & Korczak, 2008). Nonetheless, the P1-N1-P2 complex remains the most clinically useful objective CAEP currently.

1.4.1 P1-N1-P2 complex

The P1 or P100 response is commonly recorded around 50-80 milliseconds (ms) post-stimulus, P2 around 200 ms, and N1 between 100 and 150 ms (H. Davis & Zerlin, 1966; Hall, 1992). Occasionally, a second negative voltage, N2, appears around the vicinity of 180 and 250 ms post-stimulus. The P and N denote positivity or negativity of the waveform, whilst the number suggests the expected latency. The measures of latency, amplitude and waveform morphology can vary widely with different stimulus parameters, recordings sites and age.

The P1-N1-P2 complex offers several advantages over other evoked potential measures. The complex can be reliably reproduced and demonstrates responses to clicks, tone bursts, speech tokens and complex harmonic tones (Näätänen & Picton, 1987; Pekkonen, et al., 1995). The stimulus is presented in a series of repeats, time-locked to the response recorded. Signal averaging of the time window, also known as an epoch, occurs over a number of repeats and generates the response relative to the background EEG. The complex can also be used for estimating behavioural hearing thresholds (Hyde, 1997; Stapells, 2002). However, the ABR
has been more favoured for threshold-seeking, as it is unaffected by sleep and sedation. The obligatory CAEPs are still often combined with ABR results for complicated cases as it depicts the neural processes at the auditory cortex and provides information regarding the integrity of the auditory pathways (Hyde, 1997). There is also reduced electrophysiological noise as the neural structures examined are closer to the scalp electrodes (Stapells, 2002). Obligatory CAEPs have also been used to explore aspects of central auditory processing disorders (CAPD) and auditory neuropathy spectrum disorder (ANSD) (Jirsa & Clontz, 1990; Purdy & Gardner-Berry, 2009; Rance, Cone-Wesson, Wunderlich, & Dowell, 2002). It is thus important to understand the neural constituents of the P1-N1-P2 complex as they give rise to the functional representations of the response.

### 1.4.2 Neural generators of the P1-N1-P2 complex

Naatanen and Piction (1987) suggested that the recorded evoked potential is a summation of total neural activity across all sites which generate neuronal excitation (Näätänen & Picton, 1987). Thus it is of importance to relate to the basic structures when analysing AEP waveforms, which convey series of overlapping dipole-to-dipole interactions. At this level of neuroanatomy, two types of potentials have been identified for the generation and conduction of AEPs: the postsynaptic potentials (PSP) and compound action potentials (CAP). PSP are present at synaptic junctions formed through contact between axons, dendrites, and soma. The flux of neurotransmitters across synaptic clefts and their passage through various channels in this junction generate a slow localized membrane voltage difference that can be detected. Conversely, a fast voltage change propagating along an axon and nerve fibre tracts are electrically represented by action potentials or the CAP (Eggermont, 2007).

Furthermore, a variety of neurons exists with anatomical differences. These serve a purpose in neuronal orientation and distribution across the cortices. For example, the pyramidal cells and their dendritic fields are spread widely into various layers of the cortex, which provides differentiation between negative and positive potentials detected at the scalp. Supported by the source-sink current model, excitation that is orientated to the middle layer via the dendritic fields can result in a positive AEP peak, whilst more superficial activation results in negative peaks at the scalp (Eggermont, 2007).
As the late latency responses lie within the bordering region of the MLR, dissociation of neural generators of the P1 response from the Pb component of the MLR has been difficult. P1 has been implicated as a product of active neuronal signalling within the primary auditory cortex, and Heschl’s gyrus in particular (Salamy, 1984). This was based on intra-cortical recordings via a stereo-electro-encephalographic exploration in temporal lobes of humans conducted by Liegeois-Chauvel and colleagues (Liégeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994). They concluded that the positivity of the response can be attributed to the conduction through ‘deep sink’ layers II and IV of the primary auditory cortex and the subsequent superficial current return gave rise to the generation from the lateral portion of Heschl’s gyrus (Liégeois-Chauvel, et al., 1994). The associative auditory cortex and the thalamocortical projections were also suspected to dominate P1 response. Using neuromagnetic source localisation, Godey et al found that bilateral contiguous sources, of radial and tangential orientation can be responsible for the generation of P1 response (Godey, Schwartz, de Graaf, Chauvel, & Liégeois-Chauvel, 2001). Other studies have continued to provide evidence of involvement amongst other cortical regions such as the planum temporale, hippocampus, and lateral temporal cortex as neural sources of P1 (B. Martin, et al., 2007).

The P1 response is also the primary marker for maturation of the auditory system. In children, the P1 waveform is significantly larger in amplitude and longer in latency; occurring as the dominant waveform of the entire CAEP response. Kraus and McGee have shown through MLR studies that the acoustic signal can reach the auditory cortices within the first 20-30ms in normal hearing individuals (Kraus & McGee, 1993). Therefore, children’s P1 response with longer latencies should include aspects of the cortical processing from the feedback and return pathways of communication between the primary auditory cortex and its association areas.

Stapells (2001) regards the formation of N1 as the arrival and detection of the auditory input (Stapells, 2002). Although the response is non-discriminatory, it represents arrival of “potentially discriminable” content (B. Martin & Boothroyd, 1999). Furthermore, MEG
evidence showing frequency-dependent 3D dipole orientations suggests that N1 generators may be tonotopically organised (Verkindt, Bertrand, Perrin, Echallier, & Pernier, 1995). N1 has been postulated to originate in various sites within both primary and secondary auditory cortices, and non-specific modality areas (B. Martin, et al., 2007; Stapells, 2002; Vaughan & Ritter, 1970). The N1 wave itself is comprised of three components: the frontocentral negativity (N1b), the biphasic T complex, and a negativity wave with non-sound specific controversial origins (Stapells, 2002).

The N1b is suggested to arise from bilateral temporal lobe projections near the auditory cortex in the superior temporal lobe, specifically the planum temporale, via vertical dipole orientations (Vaughan & Ritter, 1970). This initial component of the N1 wave has been implicated in the attention orientated towards the onset of sound stimulus. Näätänen and Picton (1987) has proposed that this component is important for production of short-term sensory-memories (Näätänen & Picton, 1987). The T complex was investigated over recordings from the temporal lobe and vertex (Wolpaw & Penry, 1975). The presence of biphasic waves was observed: a positivity at 105-100ms and negativity at 150-160ms were recorded from the secondary auditory cortex in the superior temporal gyrus from dipoles of radial orientation (Vaughan & Ritter, 1970; Wolpaw & Penry, 1975). This large response has been suggested to reflect activation from the primary auditory cortex and interactions with the association cortices. The final component of the N1 waveform has been queried to interact with non-auditory specific centres such as the motor and pre-motor areas, which are affected by alertness or attention state. Näätänen and Picton (1987) have proposed that this third controversial N1 component may represent complex cerebral mechanisms under the control of the reticular formation and thalamus, involving arousal and perception of the individual.

\( P2 \)

The second positive wave P2 has been implicated with origins from structures which were non-modality specific (Ponton, Eggermont, Kwong, & Don, 2000). Aside from the primary and secondary cortices, the major centre of P2 generation resides with close proximity to Heschl’s gyrus (Lütkenhöner & Steinsträter, 1998). The mesencephalic reticular activating system (RAS) has also been shown to be a source of generation (Rif, Hari, Hämäläinen, & Sams, 1991). However, the individual neural generators prove to be difficult to isolate when
such complex interactions are at play. This is supported by the influence of attention on P2 measures of amplitude (Alho, Tottola, Reinikainen, Sams, & Naatanen, 1987; Rif, et al., 1991). Functionally, the diverse generator sites for P2 advocates its role in higher-order analysis and examination of the stimulus carried by earlier structures of the auditory pathway (Ponton, et al., 2000).

### 1.4.3 Effects of variables on CAEP measures

#### 1.4.3.1 Measures: Latency, Amplitude and Area-Under-the-Curve in waveform analysis

The evoked potentials literature has always been dominated by the two fundamental measures of latency and amplitude. Latency, measured in milliseconds, describes the time course from the stimulus onset to the capture of peaks or valleys of the evoked potential (Hall, 1992). It therefore describes the neural conduction for the acoustic signal to encode and spread from the peripheral to the central auditory system, encompassing time and conduction velocity. The amplitude or magnitude is a measure of the potential difference in microvolts (µV); hence, a reflection of the strength of the overall excitation. Absolute amplitude measures are affected by the baseline correction of the evoked waveform; several techniques have been used to index this measure. Amplitudes are commonly marked as the value of the peak with the largest magnitude in the waveform (Hall, 1992). Other studies have found the amplitude difference or the ‘peak-to-peak’ of N1-P2 to be useful in adults.

The measure of ‘area-under-the-curve’ (AUC), or the area beneath the waveform, has been utilised across different measures of evoked potentials. In research with middle latency responses, McGee et al first drew attention to its usefulness due to the need to expand MLR filters to avoid waveform distortions. Suzuki et al reported increases in intra-participant and inter-participant variability in latency and amplitudes of the MLR with filter expansion (Suzuki, Hirabayashi, & Kobayashi, 1984). AUC measures in middle latency responses are also supported by evidence that MLR latency and amplitudes are affected by participant variables such as age, sleep state, and stimulus parameters of rate, level and filter (McGee, Kraus, & Manfredi, 1988; Pynchon, Tucker, Ruth, Barrett, & Herr, 1998). McGee argued that amplitude only reflected the activity at one point in time, while the latency described the time
course for which the excitation was sustained. Therefore, the area measure of $P_a$ in MLRs is a representation of total neural synchrony and activity (McGee, et al., 1988). Furthermore, McGee et al suspected that since the area reflected the covariance between latency width and amplitude, it should remain stable under situations where the traditional measures vary inversely. This was confirmed in their findings where the Pa area remained stable, as amplitude and latency varied with changes to filter and stimuli envelopes. Interesting to note, however, a significant change was found with the area measure alongside amplitude and latency with a low frequency tone pip at 500 Hz. This suggested potential usefulness for MLR area measures in low frequency aspects of auditory processing (McGee, et al., 1988).

Pynchon and colleagues adopted a different area calculation technique by taking the midpoint of the grand average of latencies across the study sample, and aligning it with point of Pa peak (Pynchon, et al., 1998). Area under the waveform was then integrated across the grand average width of latency. Their study did not report any significant effects from this measure compared with the traditional MLR latency and amplitude measures, as a function of age (Pynchon, et al., 1998).

![Diagram](image)

**Figure 8**: Area calculations of Pa of MLR waveforms using knowledge of the width and amplitude of Pa (McGee, et al., 1988, p. 122).

Area measures in cortical responses have also been popular as an indication for detection of the mismatch negativity. Cortical responses generally fluctuate more than lower-order AEPs. Although N1 and P300 remain sufficiently reliable on test-retest reliability, MMN is heavily influenced by signal-to-noise ratio. McGee, Kraus and Nicol (1997) reported identifying presence of the MMN using various statistical methods based on signal detection theory (McGee, Kraus, & Nicol, 1997). They reported that a combination of using area and onset latency to calculations were more advantageous than visual identification of parameters, in improving the judgement of MMN validity (McGee, et al., 1997). Area calculations are currently widely used for MMN, and evoked potentials systems such as Neuroscan systems have developed integration algorithms in the software for waveform analysis. Sharma and
colleagues conducted a similar study using such software, and concluded that MMN identification was most accurately achieved with an area criterion of $>110 \mu V.ms$ (M. Sharma et al., 2004). They further suggested for area criteria to be clinically useful, normative data needs to be obtained for specific populations (M. Sharma et al., 2004).

The clinical usefulness of CAEP measures is often jeopardised; no gold standard currently exist as settings for recording CAEPs in adults and children. These settings have been difficult to ascertain as they are influenced by both stimulus and participant variables, found to affect latency and amplitudes widely throughout the literature.

**1.4.3.2 Stimulus frequency and intensity level**

The effect of changing stimulus intensity or level on CAEP measures has been thoroughly studied since the 1960s. It is established that effect on amplitude from increasing stimulus intensity is proportional (H. Davis & Zerlin, 1966; Picton, Woods, & Proulx, 1978), with the exception of a saturation effect seen at higher intensities above 50 or 60dB HL (Picton, et al., 1978). Picton, Woods and Proulx postulated that it may either be contributions from saturation of the generator neurons or the overlap of generator populations achieved by high level stimulation (Picton, et al., 1978). The effect on latency was found to decrease with increasing intensity of the stimulus (Rapin, Schimmel, Tourk, Krasnegor, & Pollak, 1966).

Jacobson and colleagues explored the effects of a 250, 1000, and 4000 Hz tone stimulus on multichannel recordings of N1 (Jacobson, Lombardi, Gibbens, Ahmad, & Newman, 1992). In agreement with evidence from single-channel studies (McCandless & Best, 1966; Rapin, et al., 1966), they found that N1 had a longer latency and larger amplitude for lower frequency stimuli. Jacobson et al. proposed that both structural contributions from both the peripheral and central auditory system may combine for such an effect. Consistent evidence has been found for the effect of larger amplitude and more distinctive waveform (H. Davis & Zerlin, 1966; Jacobson, et al., 1992); and dipole-source modelling studies have alluded to a more superficial source for cortical reception of low frequency stimuli than those of higher frequencies in the deeper layers of the cortex (Pantev et al., 1988). Jacobson theorized that deeper generators of the higher frequency responses had to overcome more impedance from neural structures, and their electrical currents which induce a ‘smearing’ effect and reduction of the recorded response at the scalp. This is supported by more recent functional MRI
studies (Bilecen, Scheffler, Schmid, Tschopp, & Seelig, 1998) where higher frequency excitation in the auditory cortex was found to be more medially and frontally distributed than low frequency excitation. Furthermore, broader area of activation was demonstrated in the lower frequency tone of 500 Hz used (Bilecen, et al., 1998).

More peripherally, Jacobson also discussed the potential for a larger summation of energy for low frequency stimuli due to recruitment of neurons that are excited by high frequency stimuli. Studies comparing effects of frequency and intensity levels of stimuli interaction on amplitude, suggested that the presentation of low frequency stimuli at suprathreshold levels, causes a broader recruitment of high frequency-specific neurons peripherally due to mechanics of the travelling wave from basal to apical regions of the basilar membrane , (Antinoro, Skinner, & Jones, 1969). The draft from a larger pool of source generators from the periphery, ascending to the central auditory system, may be reflected in the scalp recordings. However, Antinoro et al. (1969) also reported that at high stimulus intensities, the effect of larger amplitude in low frequency stimuli was dismissed above 2000 Hz. Evidence of the same frequency on amplitude relationship has been discussed in low intensity input (low sensation levels measure in dBSL), which raises contradictions with the theory of peripheral contributions somewhat (Antinoro, et al., 1969; Rapin, et al., 1966).

The effects on latency from varied frequency of tones are not in agreement. While Jacobson et al. and other groups have reported later latency for lower frequency stimuli (Jacobson, et al., 1992; Woods, Alain, Covarrubias, & Zaidel, 1993), Zerlin and Naunton (1974) found the reverse to be true; higher frequency stimuli evoked longer latencies (Zerlin & Naunton, 1974). Zerlin and Naunton further considered that this was unexpected for the theory where restricted contribution from the high frequency portion of the basilar membrane was in effect. Jacobson et al reasoned that conduction velocities between various tonotopical components of the primary auditory cortex may also play an important role (Jacobson, et al., 1992).

1.4.3.3 Rate and Inter-stimulus Interval (ISI)

The rate of repetition is related to the inter-stimulus interval (ISI). The ISI is defined as the period of time between the offset of the stimulus and the onset of the next stimulus in the stimuli train; thus the rate of presentation is inversely proportional to the ISI. Picton, Woods and Proulx (1978) reported that the N1 waveform was most affected, whereby increased rate
of presentation (reduced ISI) elicited smaller amplitudes (Picton, et al., 1978). This has been an apparent effect noted widely as a phenomenon known as ‘habituation’ (Kandel, 1991). Habituation is a common concept in neuroscience and can be described as the learned cessation of response due to a repeating stimulus (Kandel, 1991). This results in a loss of neural summation and thus reduced amplitude. Habituation has been considered to be affected by refractoriness at the single neuron level. Refractoriness describes a period post-depolarisation of which new excitation and action potentials cannot be elicited. Picton and colleagues (Picton, et al., 1974) believed that refractoriness and habituation differed; the refractory state should resolve eventually while further reduction in amplitude continued in habituation. Budd and colleagues (1998) attempted to separate effects of refractoriness and habituation in the N1 response; and found that N1 decline in amplitude was strongly contributed by the refractory process (Budd, Barry, Gordon, Rennie, & Michie, 1998). However, little effect has been reported on latency from stimulus rate changes (H. Davis, Mast, Yoshie, & Zerlin, 1966).

1.4.3.4 Stimulus duration

Using a toneburst stimulus, Onishi and Davis discovered that an increase was observed in CAEP amplitude when the duration of the toneburst was up to 30-50ms (Onishi & Davis, 1968). Should the duration last longer than 5 seconds, an ‘adaptation’ effect on the amplitude was described by Picton, et al. (1978) (Picton, et al., 1978); an increasing attenuation of the N1 amplitude was observed as the duration increased across 3 to 9.24 seconds (Picton, et al., 1978).

1.4.3.5 Participant variables

The state of the participant has an impact on latency and amplitude measures of CAEPs. The state of arousal has been implicated; early studies from the 1970s have revealed that the effect in amplitude on the evoked responses occurs from N1 onwards. Attention was found to produce an increase in negativity of the N1 and P2 components; in other words, the amplitude increased for N1 and decreased for P2 (Campbell, Bell, & C, 1992). States of alertness and sleep are also influential. De Lught and colleagues found that as one enters sleep through stages of awake, 1, 2, 3 and 4 (REM), a negative slow wave that overlapped the P1-N1-P2 complex may gradually attenuate this complex which is larger in amplitude during alert and
awake states (de Lught, Loewy, & Campbell, 1996). N1 thus became increasingly attenuated, and P1 and P2 increasingly augmented during sleep onset. No reported correlations with latency effects were noted (de Lught, et al., 1996).

1.4.4 Speech-evoked CAEPs

Cortical response elicited by a range of stimuli including frequency-specific tones, clicks, music, and speech stimuli have been widely studied (Näätänen & Picton, 1987). Kurzberg (1989) provided evidence that contrasting speech stimuli were capable of eliciting different CAEP morphology which may infer different underlying neural representations of speech sounds (Kurtzberg, 1989). Most research support the idea that speech stimuli activate dissimilar areas of the cortex. For example, the N1 generators of a 1000 Hz tone were reported to be more posterior, inferior, and medial to vowels (Eulitz, Diesch, Pantev, Hampson, & Elbert, 1995). Although the inference favoured the use of discriminatory CAEP measures, it was suggested that the combination of ABR and CAEP measures would be optimally advantageous in assessment of central auditory processing and the effects of amplification. Thus popularity for CAEP studies exploring speech stimuli characteristics grew, and a difference in CAEPs between speech and non-speech stimuli has since been established (Ceponiene, Torki, Alku, Koyama, & Townsend, 2008).

Varying types of speech stimuli have been studied, including consonants, vowels, consonant-vowels, syllables and articulatory characteristics, and even across languages. Larger amplitudes and longer latencies in N1 and P2 from a Finnish vowel of /a/ than its frequency-matched tone response was reported (Tiitinen, Sivonen, Alku, Virtanen, & Näätänen, 1999). It was also suggested that spectral dissimilarities of the vowels were related to further distance apart in a tonotopical map of their cortical generators (Obleser, Elbert, Lahiri, & Eulitz, 2003). Consonant-vowel stimuli have commonly been used as they elicit overlapping responses, explaining the transition from onset of the consonant to onset of the vowel; which is now named the acoustic change complex (Ostroff, Martin, & Boothroyd, 1998).

In longer duration speech stimuli, the voice-onset-time has been implicated in affecting the cortical response recorded. Lisker and Abramson (1964) first defined voice-onset-time (VOT) as the time between a stop consonant release and the onset of voicing of the speech
stimuli. The central auditory system has been previously suggested to be highly sensitive to stimulus onset transients in tones and pulse noise (Phillips, Hall, & Boehnke, 2002). Phillips et al. (2002) further suggested that onset responses are detected by the ability of neural mechanisms to identify properties of the stimulus. Thus the temporal properties has been identified as an important acoustic cue for speech perception and hence studied via cortical responses. Varying durations of VOT have elicited different morphology between children and adults, suggested to infer maturational changes in the neural encoding of speech cues temporally (King et al., 2008). Latency differences in adult CAEPs when comparing between voiced and voiceless stimuli were also found (Sharma, Marsh, & Dorman, 2000). However, it was postulated that synthetic speech tokens, as used by Sharma et al. (2000), may produce different responses compared to the natural speech tokens (Tremblay, Friesen, Martin, & Wright, 2003).

Natural speech tokens have been suggested to imitate the human speech characteristics, and thus favoured in evoked potentials assessments. Tremblay and colleagues established that they could record CAEPs from Cz with good test-retest reliability in normal hearing adults, from four naturally produced speech tokens /bi/, /pi/, /jɨ/ and /si/ (Tremblay, et al., 2003). With reliability established, research has been investigating temporal aspects of speech encoding of CAEPs. Effects of stimulus duration has been found to also mirror in the natural speech stimuli-evoked CAEPs; responses evoked from stimuli with longer duration, have smaller amplitude and later latency (Agung, et al., 2006; Beukes, et al., 2009). Beukes, Munro and Purdy (2009) investigated interactions between ISI and stimulus duration with tones and speech phonemes of /m/ and /sh/ (Beukes, et al., 2009). They reported that with a short ISI of 1125 ms, CAEP amplitudes reduced with longer stimulus duration of 500 ms. However, increased ISI did induce the difference in amplitude only for the speech stimuli. The authors supported the idea of duration-sensitive neurons in neural processing of speech, and further speculated that the complexity of speech processing may require a higher level of sensitivity (Beukes, et al., 2009). Without this degree of neuronal sensitivity, spectral cues may become degraded in compensation of temporal reception and processing.

The most optimal ISI and stimulus duration for eliciting CAEPs from a speech token in infants 3-7 months was established by a similar study (Golding, Purdy, Sharma, & Dillon, 2006). Golding and colleagues reported that using a fixed ISI of 750 ms, duration of longer
than 35 ms in /m/ and /t/ did not produce significant latency or amplitude changes to the infant P1. Conversely, when the duration was fixed at 79 ms, varying the ISI did not have an effect on latency, and the increase in P1 amplitude was only linear in /t/, not /m/. Golding et al. hence recommended that use of an ISI of 1125 ms and duration of no longer than 35 ms was optimal for acquisition in infants (Golding, et al., 2006).

Many recent studies have focused on the spectral distinction of natural speech stimuli to elicit different CAEPs: suggestive of neural ability to encode spectral differences from speech stimuli. The seven Ling sounds /m/, /u/, /a/, /i/, /sh/, /s/ and /c/, were used by Agung and colleagues (2006) to explore the neural encoding of sounds spanning the speech spectrum in normal hearing adults. The most distinguished differences between responses resulted between high and low frequency tokens, but the differences between each of the Ling sounds were not sufficient to use as a measure of discrimination.

More studies have examined the use of less spectrally-specific combinations of natural speech tokens as stimuli in normal hearing adults. Although it was not their aim, Beukes, Munro and Purdy (2009) reported data from the speech stimuli of /m/ and /sh/ phonemes could be investigated spectrally (Beukes, et al., 2009). The /m/ and /sh/ phonemes represented spectral energy from low and high frequencies respectively. Both N1 and P2 were reported with longer latencies for the low frequency /m/ stimuli. However, the amplitude was larger for N1, but smaller for P2 in the responses from the low frequency phonemes. This exact pattern of interactions were also reported in a recent study, using the /mae/ and /tae/ phonemes (Munro & Purdy, 2011). Until recently, contradictory findings were uncovered in responses to tones (500, 2000 Hz) and phonemes (/t/, /k/, /d/ and /g/) in normal hearing adults (Purdy et al., 2011). Longer N1 and P2 latencies were observed for the higher frequency /t/ and /k/ by approximately 10 ms compared to the /g/ and tonal stimuli. It should be noted that phonemes used in this study varied widely in duration. Although, Purdy and colleagues (2011) suggested that differences are unlikely to be caused by stimuli duration as many previous studies using speech stimuli have all obtained similar amplitudes.

Discrimination of speech stimuli have also been investigated in young infants (Golding, et al., 2006; Purdy, et al., 2011). Purdy et al. (2011) also tested with an additional /m/ phoneme in their infant study to represent lower frequency energy evocation; hence more spectral contrast from the others. Together the /m/, /g/ and /t/ represent the span of low, mid, and high
frequency stimuli across the speech spectrum (Purdy, et al., 2011). The largest P1 amplitude difference was reported between the speech and tonal stimuli, and between amplitudes elicited from /t/ and /g/. The infants in this group also reliably elicited an N_{late}, occurring around 346-421ms for the speech phonemes (Purdy, et al., 2011). The /t/ in the infants evoked a shorter latency and larger amplitude. The authors suspect that the differences may be attributed to the nature of the stop consonant /t/. MEG evidence has suggested that larger and shorter responses were found for stop consonants (Gage, Poeppel, Roberts, & Hickok, 1998). Furthermore, the /m/ phoneme used in this study consists of a more gradual rise in energy temporally, compared to the fast /t/ (Purdy, et al., 2011).

1.4.5 Neuromaturation of obligatory CAEPs

Changes to the CAEP morphology, latency, and amplitudes from birth to adulthood have been widely established in the literature (Kushnerenko et al., 2002; Ponton, Eggermont, Khosla, Kwong, & Don, 2002; Ponton, et al., 2000; Wunderlich & Cone-Wesson, 2006). Well prior to birth at 32-38 weeks, AEPs from tone bursts were able to be recorded trans-abdominally from the foetus (Sakabe, Arayama, & Suzuki, 1969). Weitzman and Graziani recorded cortical auditory evoked potentials in preterm babies as early as 24 weeks gestation, and found a predominantly negative response as opposed to the P1, due to the immature auditory system (Weitzman & Graziani, 1968). Broadly speaking, in normal hearing children, a P1 response of large amplitude dominates the obligatory CAEP as opposed to the N1-P2 in adults.

More complex differentiation of the CAEP from childhood into adolescence has been explored. Kushnerenko and colleagues (2002) discovered in infants between 0-12 years, the large positivity was either single-peaked (P150) or double-peaked (P150 and P250). These peaks originate at full term around a latency of 200-300ms. They concluded that the positivity increased in amplitude between 0-3 months, reclining and stabilizing around 6 months. From 6 months onwards, a division of the positivity is created by strengthening of the negativity N250, until the formation of P150- N250-P350 can be recorded from a central electrode around 12 months. These findings are supported by Kurtzberg et al. (1984) with speech stimuli, and also others who have termed the positivity P2 (Wunderlich & Cone-Wesson, 2006), or N1b (Sharma, Kraus, McGee, & Nicol, 1997). It has been proposed to reflect the
rapid development of generators of the infantile auditory system (Kurtzberg, Hillpert, Kreuzer, & Vaughan, 1984; Kushnerenko, et al., 2002). Histological evidence from human autopsy samples have suggested this is vital for the formation of synaptic connections, increased myelination, and density of pyramidal cells connecting to the cortical layer III (Huttenlocher & Dabholkar, 1997).

Evidence for rapid and large-scale maturational changes in infants has been widely established. In a large scale study of 264 children between 28-29 weeks conceptional age (CA) to 16 years of age, 2 transitional periods of the CAEP morphology were reported: one during 36-41 weeks CA, and the second at approximately 5 years (Pasman, Rotteveel, Maassen, & Visco, 1999). During these transitional stages, the cortical responses appear to become reduced in amplitude. For the second transitional period around 4-6 years, a smaller P150 or P2 response was produced. Inconsistent findings have surrounded CAEPs of young toddlers at a pre-school age in the literature (Čeponiene, Lepisto, Alku, Aro, & Näätänen, 2003; Pasman, et al., 1999). Older children around 3 years were reported to have longer P1 and N2 latency than those children of school age (Čeponiene, et al., 2003). P1 waveform have also been reported with large amplitudes comparable to data from 12 month old children from Kushnerenko et al. (2002). (Čeponiene, et al., 2003; Kushnerenko, et al., 2002). In other cases, similarities in P1-N2 were consistent between children aged 4 and 9 years (Čeponiene, Rinne, & Näätänen, 2002). More research is required to investigate the maturational changes during these pre-school years. It is evidence that comparing and making inferences amongst maturational effects is that varying stimuli are used with different variables. Scalp topography and procedures often differ and thus latencies are merely approximate. Various nomenclatures have also been adopted for different waveforms.

The maturation of the P1 latency is consistently agreed across many studies in normal hearing school-aged children in the age range between 5 years to adolescence around 14 years (Gomes et al., 2001; Ponton, et al., 2002; Sharma, Dorman, et al., 2002b). In school-aged children, The prominent P1 wave was observed to reduce in latency to below 100 ms and reduce in amplitude across the ages of 6-7 to 12 years (Sharma, et al., 1997). Conversely, the negativity previously described as N250, was found to occur at approximately 200 ms and increased in amplitude from 6-12 years (Sharma, et al., 1997). With this growth of evidence, P1 latency has become a strong biomarker for auditory maturation, and thus studied in
individuals with maturational delays, such as CI users, and those with auditory processing issues (Sharma, Dorman, et al., 2002b).

Gradual maturation and differentiation of the N1 waveform becomes observable from around 8 years and older (Pasman, et al., 1999; Ponton, et al., 2002). Ponton et al. (2002) provided evidence from dipole source modelling, that differentiation of the broad P1 in children occurs gradually into the P1-N1-P2 complex. A significant decrease in N1 latency was also reported up to the age of 12 years (Gomes, et al., 2001). The mature auditory morphology encompasses a P1 approximately 50 ms post-stimulus onset, N1 around 100 ms, and P2 at 200 ms. Often a small residual N2 or N250 can be observed. Speech-evoked cortical responses have been suggested to be more reliable in the mature P1-N1-P2 form at the age of 12 years from the Cz recording site (Gilley, Sharma, Dorman, & Martin, 2005).

Figure 9: Progression of CAEP maturation from 28 weeks conceptional age to 14 years (Pasman, et al., 1999, p. 81).
1.4.6 Auditory plasticity and effects of deprivation on CAEPs

The literature discussed thus far has been works surrounding individuals with normal hearing. However, studies into the hearing impaired auditory system from both human and animal models have founded our understanding of normal auditory function. Functional magnetic resonance imaging (fMRI), magnetoencephalography (MEG), and AEPs have been used widely to study deprived auditory systems, and subsequently the effects of maturation and delayed stimulation from hearing aids and cochlear implants (Ponton et al., 2001; Purdy, Kelly, & Thorne, 2001). The idea of physiological plasticity simply describes the ability of the neural system to constantly undergo changes due to new experiences of sensory input (Greenough, 1975). The underlying neural mechanisms of plasticity involve increasing the number of synapses involved, synchrony of neuronal excitation, greater synaptic signalling from the peripheral to central nervous system, and the functional differentiation and specialisation of neurons for overall improved efficiency.

1.4.6.1 Effects of auditory training

One way in which auditory plasticity has been examined, is through exploring effects of auditory training on the neural network. Kraus and colleagues (1995) recorded an increase in amplitude of the discriminative MMN from variants of the /da/ syllable after six hours of training over one week in normal hearing adults (Kraus et al., 1995). Similar results were further replicated in a VOT study in which controls without training showed no significant changes in their MMN amplitudes (Tremblay, Kraus, Carrell, & McGee, 1997). MEG studies also strengthened the idea of this training effect (Menning, Roberts, & Pantev, 2000).

Although these clues about discrimination have been strongly coherent, it has been proposed that neural processing of detection and discrimination are inter-related, and discrimination must also be preceded by improvement of detection (Tremblay, 2007). Sharma & Dorman (1999) found increases in VOT to correlate with gradual increases in N1 latency (Sharma & Dorman, 1999). Tremblay et al. (2001) in another study observed that P2 amplitude was a better predictor of improvement of a long and duration VOT stimuli post-training (Tremblay, Kraus, McGee, Ponton, & Otis, 2001). Various other stimulus types such as tones, and training paradigms such as vowel segregation, frequency discrimination, and training duration have shown similar changes in obligatory and discriminative CAEP measures.
indicates a basis for many complex processing tasks at play; suggestive that training implicates neural sensitivity to memory, attention, decision making, and stimulus exposure. The P2 waveform due to its multitudes of source generators provides an interesting basis for studying neural mechanisms of plasticity associated with training. The fine details of neural plasticity behind training are currently not well understood. However, it is clear that training has considerable impact on CAEP responses.

1.4.6.2 A sensitive period for implantation

The fitting of hearing aids and cochlear implants to a deprived auditory system is comparable to undergoing training-induced plasticity. The downstream effects of amplification from hearing aids can be analysed at the level of the cortex through CAEPs. Cochlear implants which directly stimulate the auditory nerve provide even greater synchrony of the signal input toward the cortices. Thus the effects and variations of auditory deprivation, such as duration, lateralisation and degree of maturation, have been widely studied using CAEPs in cochlear implanterees.

Firstly, if considerations of maturational effects on CAEPs are placed aside, individuals who have acquired a hearing impairment during adulthood, are found to elicit CAEPs consistent with normal adult morphology when stimulated via a cochlear implant (Ponton, Don, Waring, Eggermont, & Masuda, 1993). Research suggests that normal measures can still be obtained after long periods of deprivation, as their neural structures reflect the completion of auditory maturation by the age of 14-16 years (Ponton, et al., 1993). However, in the presence of mature auditory structures, stimulation can produce a lateralisation effect in the presence of auditory deprivation. This is often the case for New Zealand CI users who are only funded unilaterally by the government. Evidence has suggested that unilateral deafness causes the shift from asymmetrical dominance of the contralateral pathway, to a synchronous and symmetrical activation, caused by increased interhemispheric excitation. Ponton et al. (2001) proposed that adults with late-onset unilateral deafness of more than 2 years, undergo plastic changes to their cortical generators which are reflected in the similarities between N1 amplitudes of contralateral and ipsilateral hemispheric activation. From regression analyses, peak-to-peak measures of P1-N1 and N1-P2 amplitudes showed that contralateral measures were stronger predictors of ipsilateral measures; when comparing between those deprived for more than 2 years, and deafness of less than 2 years (Ponton, et al., 2001). The authors
support the view that deprived but mature auditory systems still maintain the capacity to seek and respond to novel-experiences and sources of stimulation.

The under-developed auditory system however, faces an entirely different need for neural plasticity as the experience is immature. The P1 waveform is currently the most documented biomarker used to study cochlear implants in children who are prelingually deafened. As previously stated, P1 latency decreases rapidly from approximately 300 ms to under 100 ms over the first 3 years of life (Ponton, et al., 2000). This finding was replicated by Sharma, Dorman and Spahr (2002b), who compared P1 latencies between 136 normal hearing children and 121 congenitally deafened children with cochlear implants (Sharma, Dorman, et al., 2002b). P1 latencies of normal hearing children revealed an exponential decrease with increasing age; and were used to derive 95% confidence intervals for comparison with P1 latencies in children with CI (Figure 10). Their findings suggested a clinically significant sensitive period for implantation. P1 latency in children who were implanted younger than 3.5 years, were framed within the confidence intervals. Deviance from these norms were observed in children implanted after 7 years and to a lesser extent, those implanted between 3.6-6.5 years (Sharma, Dorman, et al., 2002b).

In a series of similar studies (Sharma, Dorman, & Spahr, 2002a; Sharma, Dorman, et al., 2002b; Sharma, Dorman, Spahr, & Todd, 2002), it was observed that provision of stimulation to the deprived and immature auditory system saw rapid changes in the P1 latency and CAEP morphology during the first 6-8 months post-implant. It was also noted that these changes were much more accentuated than those due to neuromaturation in their normal hearing peers. By 6-8 months post-implantation, children implanted prior to 3.5 years of age had P1 latencies which were age appropriate. Children implanted later than 3.5 years displayed abnormal morphology and later latency, even with years of CI experience (Sharma, Gilley, Dorman, & Baldwin, 2007). These findings are consistent with an earlier study by Ponton et al. (1996), demonstrating that lack of auditory stimulation hinders development of the pathways; for which that maturation into the adult P1-N1-P2 complex is delayed by 5 years in CI users (Ponton, Don, Eggermont, Waring, & Masuda, 1996). They suggested that three factors are implicated in this delay: the unilateral stimulation from the CI; the intermittent nature of stimulation as there are times when it would be switched-off; and the extent of the deprivation (Ponton, et al., 1996). It was thus postulated that perhaps the arrestment of
excitation into varying layers of the auditory cortex can occur at different periods of cortical maturation, due to the time of deafness onset.

Figure 10: 95% confidence intervals of P1 latency maturation in normal hearing people from 0.1-20 years. The intervals are overlaid with P1 latencies from children CI users categorized by their age of implantation (Sharma, Dorman, et al., 2002b, p. 535).

1.4.6.3 Mechanisms of auditory plasticity

The idea of a sensitive period for which the auditory system is most optimally plastic, has important clinical relevance in support of early amplification and cochlear implantation. Thus it is important to understand underlying mechanisms to provide better clinical advice.

Evidence from electrophysiological and imaging studies has revealed several mechanisms of auditory plasticity which are supportive of this sensitive period. Currently a few models have been suggested to explain the changes and rate of change reflected in the P1 waveform.

Cross-modal recruitment describes the adaptation of the deprived auditory cortex in recruitment of input from other sensory modalities, such as visual or somatosensory areas of the cortices. PET scans of glucose metabolism were examined in prelingually deafened patients. It was observed that the duration of deafness was reflected by the increase in activation of other cortical areas such as visual, insula, and parietal areas (D. Lee et al., 2001; H. Lee et al., 2005). In a fMRI study, it was also found that attention to the visual stimuli of American Sign language in deaf individuals elicited greater activation within constituents of the auditory cortex, compared to normal hearing individuals (Fine, Finney, Boynton, 

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Dobkins, 2005). These findings are suggestive of some form of re-organisation in higher order processing which has instructed the receptiveness to other sensory areas. However, Sharma, Dorman and Spahr believed that this was probably not the principal mechanism of auditory plasticity in children CI users.

Another theory used to explain the effects of a sensitive period suggests that auditory deprivation causes cortical de-coupling: the event of disconnection of communication between primary and associative auditory areas. Kral and colleagues lead histological studies based on cortical samples from the congenitally deafened cat model, which was stimulated electrically by a cochlear implant (Kral, Tillein, Heid, Hartmann, & Klinke, 2004). After the feline-equivalent of a sensitive period, electrical stimulation showed delayed or absent activation of deep layers V and VI (supragranular layers and infragranular layers). These delays significantly hinder higher-order feedback projections to the infragranular layers of the primary auditory cortex, which then relays onto subcortical auditory layers (Kral, et al., 2004). Hence the deterioration of feedback from these infragranular layers causes a ‘de-coupling’ of communication between secondary and primary auditory cortices, reducing the inhibition via top-down processing mechanisms which is essential in normal auditory function. This theory further supports cross-modality recruitment, as the action of de-coupling areas enables the association cortices to be more receptive to other sensory inputs (Sharma, Nash, & Dorman, 2009). Furthermore, Kral and colleagues (2004) suggests that the analysis of incoming acoustic signals reduces in efficiency upon re-direction of function in these secondary processing areas; which are implicated for language development and auditory processing.

1.4.6.4 CAEP evidence of cortical re-organisation

Cross-modal recruitment and cortical de-coupling thus combine to result in re-organisation of auditory processing in cortical areas. These mechanisms of reactivated plasticity after long periods of deprivation can be interpolated clinically into CAEP morphologies. Abnormal morphologies have been reported throughout the literature, describing cortical re-organisation reflected after re-stimulation of the auditory system (K Gordon et al., 2010; Ponton & Eggermont, 2001; Sharma, et al., 2009). Sharma, Nash and Dorman (2009) have recently presented clinical examples of varying morphologies seen in children who have experienced periods of auditory deprivation. They suggest that a ‘deprivation negativity’ seen as a sharp
negative wave preceding a delayed P1, occurs as a hallmark of an unstimulated auditory system, although it may still be plastic. Should a hearing impaired individual receive acoustic stimulation, a delayed P1 with longer than normal latency is expected, without the presence of the deprivation negativity. Lastly, Sharma and colleagues (2009) characterised a polyphasic morphology to correlate with abnormal development and/or reorganisation of the auditory pathways. They argue that these may be observed in older children who are aided or implanted, and can also be found by recording from the non-implanted side of unilateral CI recipients (Sharma, et al., 2009).

Figure 11: Examples of varying CAEP morphology due to effects of deprivation. In descending order: (1) Children with normal hearing and auditory development. (2) Children with poorly stimulated to unstimulated auditory system. (3) Children who may not have experienced a high degree of deprivation either due to delayed amplification or implantation, or their impairment was not severe-profound to begin with. (4) Polyphasic morphology from cortical re-organisation, found in those implanted after the sensitive period (Sharma, et al., 2009, p. 274).

Recently, some agreement in characterisation of abnormal morphologies due to deprivation and cortical reorganisation has been considered. CAEPs were recorded from acute CI stimulation in 72 children who were bilaterally implanted simultaneously, and three types of CAEP morphologies were reported. Approximately half of the children had delayed P1 morphology preceded by a negative peak similar to the deprivation-related negativity reported by Sharma and colleagues (2009). The rest of the children exhibited responses of P1 morphology typical of normal hearing, and a multi-peak response also similar to the polyphasic response described previously. Furthermore, they examined correlation of these morphologies with numerous factors including hearing aetiologies and CI details, in which a
correlation with the genetic mutation of the GJB-2 gene was found with 79% of the deprivation-negativity responses. Gordon and colleagues postulated that this negativity may even consist of immature MLR components, and is suggestive of abnormal development and poor maturation even in the presence of CI stimulation. Multi-peak responses were also considered to reflect some evidence of re-organisation, and suggested to be an intermediate stage between the normal positive P1 response and the deprivation negativity response (K Gordon, et al., 2010). Nonetheless, it is clear that source localisation of generators involved in reorganisation should be studied, as well as the numerous factors that affect these sources.

There is also growing evidence that for some CI users, their CAEPs may never become comparative to normal hearing peers of the same age, even with cortical reorganisation after deprivation-induced delays. In some children with CI, it has been proposed that there may be delay-dependent refusal for the emergence of their N1 waveform; which is consistent with sustained immaturity found in congenitally deafened white kittens, fitted with CI after a long duration of profound deafness (Ponton & Eggermont, 2001). In an older study, Ponton and colleagues described cases of unique CAEP morphology in young adults who experienced a prolonged duration of deprivation prior to delayed implantation in childhood (Ponton, Moore, & Eggermont, 1999). They suspected that the onset of deafness and duration of deafness may be a strong predictor of whether the rate of maturation for P1 latency in these individuals will become comparable to normal hearing maturation. They presented longitudinal CAEP traces of two children: one congenitally profound and the other affected by meningitis at the age of 3.5 years, whom were both implanted at 6 years. Initially, rapid reduction in P1 latency was observed and appeared appropriate as previously reported in novel stimulation on the deprived auditory system. However, their P1 amplitudes were maintained and the adult-like P1-N1-P2 complex was unlikely to be achieved even after adjustment for their hearing age. In the meningitis case, some stimulation and maturation of the system may have commenced in the 3.5 years prior to deafness onset. In this very case, it was also noted that although the N1 waveform was appropriately absent, an age-appropriate P2 response was observed. In further support for a sensitive period of optimal plasticity, the authors concluded that CI users who experienced prolonged delay, may possess P1 latencies which have the capacity to asymptote prior to the realms of normal P1 latency.
Sharma, Dorman & Spahr also emphasised that age is not the only predictor of P1 latency post-implantation, as several children who were implanted late still exhibited P1 latencies within age-appropriate norms (Sharma, Dorman, et al., 2002b). Many long-term factors continue to affect overall neural encoding of the signal, which is reflected in their CAEPs. Although still not well understood, current considerations include the effects and quality of hearing aid usage prior to implantation, residual hearing post-implantation, initial aided thresholds, hearing aetiologies and the effects of auditory verbal therapy (AVT) or other rehabilitative options available to the CI user.
1.5 Cortical auditory evoked potentials in CI users

CAEPs provide a view into neuronal re-organisation and adaptation when cochlear implants begin stimulation upon a previously deprived auditory system. They can further unveil the impact of artificial stimulation on maturation and language acquisition. Clinically, they have the potential for predicting performance outcomes, verification of mapping adjustments, and even for the assessment CI candidacy (Purdy & Gardner-Berry, 2009). Unfortunately, typical responses from CI users are difficult to ascertain due to the variables such as their deafness history, and parameters for which CI stimulation and CAEP acquisitions are conducted in. The research in this field is often hard to compare and generalize; thus it is important to understand the basic differences of response in CI users and normal hearing individuals.

Under electrical stimulation, Firszt, Chambers and Kraus (2002) observed significant increases in N1-P2 amplitudes with increasing levels of stimulus current from biphasic pulse trains (Firszt, Chambers, Kraus, & Reeder, 2002). Latency was not significantly altered when stimulation was delivered from apical to basal electrode sites. However, they reported that the average latency of N1-P2 responses were earlier than expected compared to existing literature on normal hearing individuals (Firszt, Chambers, Kraus, et al., 2002). This finding was not consistent with the earlier study from Micco et al (1995), where the latencies were not significantly different between their Nucleus 22 CI participants and normal hearing controls (Micco et al., 1995). However, Micco et al also delivered their stimuli via a soundfield loudspeaker, and their participants had Nucleus devices instead of the Clarion devices tested by Firszt and colleagues (2002). Kelly (2001) also did not find latency differences were significant between the CI and normal hearing groups from tonal stimuli of 0.5, 1, and 4 kHz presented in soundfield (Kelly, 2001; Kelly, et al., 2005). However, as the frequency of the tones increase, N1 amplitude was observed to be smaller in CI users (Kelly, et al., 2005).

Effects of using speech stimuli for CAEPs have been highlighted previously. Groenen et al. (2001) compared CAEP measures between CI users and normal hearing controls stimulated by tones and three speech contrasts, differing in place of articulation, voicing, and vowel transition. Delayed latency and smaller amplitudes were reported in the CI group compared to the normal hearing group. However, they reported that there was a vast variability in the latency and amplitude measures within the CI group, which may have contributed to an
overall averaging effect in the grand average waveform, which displayed no distinct peaks for the consonant speech stimuli (Groenen, Beynon, Snik, & Broek, 2001).

1.5.1 Behavioural speech perception and CAEPs in CI users

There has been growing interests in uncovering relationships between CAEPs and behavioural speech perception in CI users, as they infer great clinical utility. However, mixed findings have been reported throughout the field, with varying types of stimuli and speech perception tests administered. Firszt, Chambers and Kraus (2002), compared CAEPs in adult CI users with a large battery of behavioural speech perception tests inclusive of CNC monosyllabic words, HINT, CUNY sentences, and the revised SPIN test, in the twin study from the publication discussed previously. No significant relationship between any of the speech tests were found with electrically-evoked cortical responses. Kelly et al. (2005) reported shorter P2 latencies recorded from Cz, from adult CI users who had better speech perception scores from CNC phoneme tests. This effect was only observed with a 250 Hz stimuli, but not for 1000 Hz and 4000 Hz (Kelly, et al., 2005). However, both studies reported that longer duration of deafness prior to implant were found in the individuals with poor speech perception, and thus is difficult to remove from the effect that deprivation has on CAEPs when comparing with speech abilities. Behavioural speech perception has also been compared to measures with eALRs (Makhdoum, Groenen, Snik, & Broek, 1998; Maurer, Collet, Pelster, Truy, & Gallégo, 2002) Makhdoum, Groenen, Snik and Broek (1998) also reported consistent results of a marginal negative correlation between P2 latency and scores from a spondee identification test in eALRs (Makhdoum, et al., 1998).

Behavioural speech measures have also been examined in paediatric CI users. In young infants, it has been reported that increased canonical vocalisations, such as vowels, consonants, consonant-vowel syllables, vowel-consonant syllables, coincided with rapid P1 latency decreases in the first three months post-implantation (A. Sharma et al., 2004). The strength of this finding was depreciated by only having two participants aged around 13-14 months. Furthermore, the data was sampled from recordings of interactions with caregivers, which may not be the fairest representation of each infant’s speech abilities. Comparisons are difficult to make across the literature as there are a range of speech tests for children according to their age-equivalent capabilities. Additionally, the capabilities of a child with a
Cochlear implant are generally incomparable with an age-matched normal hearing peer, due to the factors affecting language acquisition discussed earlier. There are also no speech perception measures which will accommodate abilities across the range of ages which many studies encompass.

Furthermore, the relationship between behavioural speech measures and CAEPs in infants and young children cannot be considered without the compounding effects of auditory maturation. It has been long suggested that maturation of early implanted children occur at the same rate as the normal hearing children, although they incur a delay by approximately 5 years (Ponton, et al., 1996). Delay in implantation is also known to be reflected in delayed language development of CI children (Svirsky, et al., 2000). Studies have found indications that the age of implantation and thus lengths of auditory deprivations were strong indicators of CAEP morphology and language development. Poorer speech perception outcomes, gauged by a battery of closed-set (Word identification by picture index, test of auditory comprehension) and open-set tests (Glendonald test of auditory speech perception, Phonetically balanced kindergarten words), were correlated with atypical CAEP morphology even after approximately 6 years post-implantation (K Gordon, Tanaka, & Papsin, 2005).

Gordon et al. (2005) reported three types of CAEP morphology, where the most atypical type consisted of a large negativity preceding a positive wave, was more common in prelingually deafened children implanted after 7-10 years, who also achieved lower speech perception scores. However, exceptions of varying speech perception abilities were also observed in some earlier implanted children.

More recently, young CI users aged 11-18 years were either identified as either ‘good’ or ‘fair’ speech perception abilities by their scores on the monosyllabic open set Phonetically Balanced Kindergarten test (K Gordon, Tanaka, Wong, & Papsin, 2008). They reported a positive peak of large amplitude in both groups which exhibited delayed latencies in comparison to the P1 waveform in normal hearing children, but similar to P2 latencies. Furthermore, the group with fair speech perception also displayed the same atypical morphology described in their earlier study, with large negativity preceding the positive waveform. They suggested that these may represent varying degrees of cortical immaturity, and that speech perception appears to correlate with the atypical morphology described by previous studies on deprived auditory systems (K Gordon, et al., 2008; Sharma, et al., 2009).
Interestingly, should the delayed positive waveform in the CI children be a reflection of the equivalent of P2 in normal hearing individuals, an argument can be made in favour of cortical re-organisation due to speech processing and language development. As the literature from young CI users are implicated with numerous factors which hinder speech development, the N1-P2 waveforms remains a more mature and concrete indicator of speech perception and cortical organisation of language for CI users.

1.5.2 The cochlear implant artefact

Currently, electrophysiology in CI users is riddled with the problem of a stimulus artefact (Gilley et al., 2006; B. Martin, 2007; Mcneill, Sharma, & Purdy, 2009). This is a unique problem when the spread of electrical activity across the scalp is detected by the EEG due to stimulus onset (Shallop, 1993). Other thoughts on the cause of the artefact relate to the structure and positioning of the internal implant, where the positioning of the receiver coil, electrode array within the cochlea may also have an effect (Shallop, 1993).

Over the last decade, the literature has reported increased incidence of the artefact. Many have alluded to the possibility that this may be a result of monopolar stimulation instead of bipolar stimulation. The monopolar mode has only been available with the Nucleus 24 implants, where two extracochlear ground electrodes are distally situated from the active intracochlear electrodes (Gilley, et al., 2006). It has been suggested that this configuration causes the electrical current to travel a greater distance compared to activity between adjacent electrodes in bipolar stimulation; thus, more spread of excitation is recorded at the scalp. Xiaoxia and colleagues (2010) investigated the relationship between the artefact and the two modes of stimulation in a chicken carcass model (Xiaoxia, Kaibao, Karp, Tremblay, & Rubinstein, 2010). The carcass tissue was implanted with 3 electrodes and fitted with a standard Nucleus Freedom external speech processor. A significantly larger CAEP artefact was observed with monopolar stimulation with sound field presentation of the speech stimuli /heed/ (Xiaoxia, et al., 2010). Thus the authors concluded that bipolar mode of stimulation for artefact-prone participants may be clinically advantageous. However, it prevents the CAEP recording to be directly compared with the individual’s favoured settings. Furthermore, monopolar stimulation is currently the most commonly used in the Nucleus 24 and Nucleus 5 devices from Cochlear Ltd, and the manufacturer default setting.
Using the chicken tissue model, Xiaoxia and colleagues also investigated the effects of a 1000 Hz tone and ‘heed’ speech stimuli on the artefact (Xiaoxia, et al., 2010). The shape of the artefact as a response to tonal stimuli remained more constant, following the duration of its stimuli. This is mirrored in the ‘heed’ stimuli where each of its syllables was reflected in the more disrupted recording (Xiaoxia, et al., 2010). These experiments on the chicken carcass model confirm the contribution from the electromagnetic response, and provide an inexpensive avenue for studying the stimulus artefact.

The artefact in the recordings becomes problematic as numerous studies have reported contamination of the entire CAEP or the early components of the CAEP, such as the P1 waveform. The artefact contamination over this latency period mostly implicates the P1-N1-P2 response compared to the ACC or discriminative CAEPs. Aspects of the artefact morphology have been described in the literature (Gilley, et al., 2006; Xiaoxia, et al., 2010). Gilley et al reported presence of a pedestal-like artefact which is time-locked to the duration of the stimulus. The pedestal-like artefacts are also approximately 5-10 times larger in amplitude, effectively masking the neural CAEP via larger amplitude and duration (Gilley, et al., 2006). Thus several techniques have been investigated in attempts to minimize the stimulus artefact in CAEP recordings.

1.5.2.1 Pre-processing strategies of artefact minimisation

The use of a stimulus with alternating polarity has been trialled as the technique is commonly utilised in ABR recordings. The EEG averaging of condensation and rarefaction components can reduce the electromagnetic artefact (Hall, 1992). However, Gilley et al. (2006) have noted that the temporal encoding by CI speech processors cannot fully accommodate the phase of the acoustic stimulus. Others have also tried to distance the recording site from the CI to reduce the spread of electromagnetic excitation across the skin (Kelly, et al., 2005; Sharma, Dorman, Spahr, et al., 2002). CAEP recordings have been successful in obtaining minimal artefact, with some reports of as low as 12% (Sharma, Dorman, Spahr, et al., 2002). The use of recording amplifier filters have also been proposed to eliminate artefacts partially (Gilley, et al., 2006). Unfortunately, filtering of wide bands can become sensitive to biologic artefacts such as eye-blinks and noise from muscle of the neck and shoulder. Gilley et al. (2006) suggested that the use of low-pass filtering at 30 Hz can be useful as often the
stimulus of interest has little energy in this frequency spectrum, but this technique may be partial to reception of noise which may obscure the neural response.

1.5.2.2 Post-processing strategies of artefact minimisation

Four techniques applied to CAEPs post-acquisition have been studied to date: principal component analysis (PCA), independent component analysis (ICA), optimized differential reference (ODR), and the subtraction method. The advantage of post-processing strategies resides in shorter testing duration which is optimal in the paediatric population.

Principal component analysis has been widely adopted, and has even been employed in removing ocular artefacts (Casarotto, Bianchi, Cerutti, & Chiarenza, 2004; Jung et al., 2000; Pantev, Dinnesen, Ross, Wollbrink, & Knief, 2006). PCA operates by attempting to reduce the artefact after separation of components by the amount of variance explained. The components which explains the most variance of data is targeted. Jung and colleagues (2000) noted that this was not particularly useful as it poorly distinguishes between the neural and artificial constituents.

The independent component analysis (ICA) stems from the basics of PCA, and uses strict higher-order statistics to identify the individual contributions of the artefact. The components are generated from a mixing matrix and are highly independent. Each component is considered to stem from independent generators and thus the artificial components can be eliminated (Makeig, Jung, Bell, Ghahremani, & Sejnowski, 1997). Debner, Hine, Bleech and Eyles (2008) successfully used the technique of independent component analysis to eliminate the artefact in a CI user (Debner, Hine, Bleech, & Eyles, 2008). Gilley et al. (2006) supported the use of ICA and reported identification of at least two contributing independent components in all five children with CI tested. However, they reported difficulty in determining a conservative criterion for ICA. Furthermore, the authors noted that the processing of the signal through the CI has an important effect on the analysis of ICA. The programmable settings of the speech processor can vary widely across CI users and thus stimulation patterns which create the artefact can be inconsistent. The internal electrode array orientation and number of electrode active, will also contribute to the electrical nature of the
artefact (Gilley, et al., 2006). These considerations render ICA highly challenging and laborious to facilitate across clinical settings without experienced analysts.

Optimised differential reference (ODR) was studied in the same experiment conducted by Gilley and colleagues, with the ICA technique (Gilley, et al., 2006). ODR attempts to minimise artefact by optimally placing the reference electrode to Cz, away from the source of electromagnetic excitation; where the active intracochelear electrode and its return path to one of the ground electrodes is situated (Gilley, et al., 2006; McGill et al., 1982). It was suggested that the differential recording from this reference site and Cz would produce minimal artefact if it is in close proximity to the isopotential contour (McGill, et al., 1982). The ODR technique found to be successful; but no significant differences between P1 latency and amplitude between CAEPs post-processing from ODR and ICA strategies were detected (Gilley, et al., 2006). Nonetheless, ODR is still less than perfect, as it remains clinically inefficient should the optimal reference site be difficult to locate, or if differences in the isopotential contours varied substantially across population.

Recently, Friesen and Picton have successfully utilised the subtraction method to reduce the artefact from the neural response (Friesen & Picton, 2010). This technique investigates the property of CAEPs when exposed to varying presentation rates. An increase in the amplitude of CAEPs is observed with increasing interstimulus interval (ISI) or decreasing rate of stimulus presentation (B. Martin, 2007). As the stimulus artefact is an artificial effect, its morphology should remain the same with varying ISI. The method thus subtracts CAEPs recorded with a short ISI from those recorded with a longer ISI to eliminate the artefact. Friesen and Picton (2010) confirmed that the N1-P2 amplitudes were increased with increased ISI in normal hearing individuals and CI users. This study employed a 1000 Hz tone and /shi/ syllable with ISIs of 500 and 3000ms, and demonstrated almost full elimination of the artefact in CI users (Friesen & Picton, 2010).

Current CAEP research in CI users is still plagued with the absence of a clinically useful method of artefact minimisation. The subtraction method appears to be more clinically efficient than other post-processing strategies. For the technique to be clinically feasible, it needs to be replicable across clinics, suitable for young children, and perhaps require fewer channels.
1.5.3 CAEPs in verification of hearing instruments

The popularity of cortical responses in CI users has excelled; there are a wide variety of applications including research into the reorganised and plastic auditory system. More importantly, many have alluded to the potential benefits of obligatory CAEPs as an objective verification of hearing aids and cochlear implant performance (Kelly, et al., 2005; Purdy & Gardner-Berry, 2009). Although obligatory CAEPs do not offer insight into complex neural processes such as discrimination of speech, it infers information regarding encoding of speech sounds which are potentially discriminable with experience, neuronal plasticity and auditory training. It provides the verification that the signal has arrived appropriately at the cortex, available for higher order processing; thus an evaluation of the auditory pathway up to the levels below this point. When combined with behavioural measures, CAEPs are an invaluable tool for assessment of any issues with processing through the hearing instrument, or nerve and brainstem pathologies such as cases of auditory neuropathy (Rance, et al., 2002).

However, prior to consideration of auditory deprivation and effects of plasticity in hearing impaired individuals, it must recognised that the hearing instruments will all alter the acoustic signal in some way which may affect the evoked potentials recorded. CAEP investigations in hearing users as a verification of amplification, also faces difficult findings. Studies have reported that increases of 12-16 dB gain across 1000-3000 Hz, did not result in a distinctive improvement in cortical responses between aided and unaided conditions (Billings, Tremblay, Souza, & Binns, 2007; Tremblay, Billings, Friesen, & Souza, 2006). Thus the same issues may be of concern in the verification of CI map settings with CAEPs; the processing algorithms and electrical encoding are important considerations for the deprived auditory system which becomes exposed to a CI.

For cochlear implant users, obligatory CAEPs can aid decisions regarding implant candidacy, and map programming when used cautiously with behavioural data. McNeill, Sharma and Purdy (2009) recently presented two case studies of CAEPs evoked by a /ba/ stimulus in adult CI users 6 years post-implantation (Mcneill, et al., 2009). Speech perception abilities were reported to be quite different between the cases, and the cortical responses were plagued with stimulus artefact contamination. Obligatory CAEPs have gradually become widely accepted for clinical verification of hearing aids and cochlear implants. The test is clinically
attractive as it can be completed with minimal electrodes and low number of sweeps in sound field compared to MMN. The absences of CAEPs indicate non-detection of the signal at the level of the cortices. They also refrain from effects of attention which is optimal for paediatric and difficult-to-test populations. But there are still many gaps in our knowledge regarding various parameters of CI processing which may have an impact on CAEP measures from a speech stimulus. It provides clues regarding the pathway integrity and detection of signal, but not enough understanding currently exists for fine tuning purposes. Longitudinal research revealing specific effects of age of implantation, type of aetiology, quality of auditory training, and CI processing details on the CAEPs would provide an invaluable insight into further developments of this clinical application.
CHAPTER 2 AIMS

This study seeks to investigate the neural processing of speech phonemes in adult and children cochlear implant users, and their relationship with Cortical Auditory Evoked potentials. Specifically, four main aims are investigated:

- To establish if the speech phonemes /m/, /g/ and /t/ generate different CAEP responses in CI users
- To investigate the relationships between CAEPs and speech perception scores in the CI users
- To explore the effects of CI adaptation over 3 months in young CI users and their relationship with CI experience and neuromaturation.
- To characterise the stimulus artefact in CAEP recordings in CI users, and subsequently explore methods of artefact reduction
CHAPTER 3 METHODS

3.1 Participants

All participants in the study were recruited from the New Zealand Northern Cochlear Implant Programme (NCIP), with consent from both the University of Auckland and the Hearing House as the service providers. This research was approved by the University of Auckland Human Participants Ethics Committee (Reference: 2010/215).

3.1.1 Part I: Adult CI users

Twenty four adult cochlear implant users aged between 20-76 years (mean 56.4, SD 18.0) participated in this study. All were regular users and at least 6 months post switch-on, with a stable map. The number of months since switch-on, was termed ‘CI age’, varied from 6 to 175 months (mean 58.7, SD 62.7). From the 24 participants, nine were implanted on the left side and 14 on the right, with one bilaterally implanted. Their most recent aided thresholds were obtained: all between the range of 10-35 dBHL across 250-6000 Hz, except for participant 16 (40-45 dBHL).

A behavioural measure of the duration of profound deafness was quantified by the number of years since they were unable to have a two-way telephone conversation prior to implantation; which ranged from 0.5 to 62 years (mean 11.7, SD 16.6 years). No restrictions on recruitment were made from cause and type of deafness. Hearing impairment were identified as ‘congenital’ if they had some level of hearing loss at birth, and ‘acquired’ if impairment occurred after birth. Congenital hearing loss was reported for ten participants while four others had an acquired loss. Mixed aetiologies existed within the group; of which seven participants reported a genetic component (see Appendix 1).

Cochlear implant devices and settings varied across the group: 21 participants wore Freedom speech processors, two used Nucleus 5 CP810, and only one used an ESPrit 3G. Seventeen participants were programmed with the ACE strategy, and seven used a SPEAK strategy. Participants were either stimulated by a rate of 900 Hz (17 participants) or 250 Hz (7 participants), and each had varying stimulation modes set by audiologists and personal
preferences. The variety if internal implants varied between CI22M, CI24M, CI 24RE and CI512 (See Appendix 2).

3.1.2 Part II: Children CI users

Sixteen children with cochlear implants between 12 months to 12 years (mean 6.2 years, SD 3.5) were involved in part II of the investigation. Children with multiple disabilities, Auditory Neuropathy/Dsynchrony and Autism Spectrum Disorder were excluded. Their CI age varied from 1 to 114 months post switch-on (mean 35.2, SD 32.0 months). Of the 16 children, five were implanted bilaterally. Twelve were tested with their right CI, and four with their left CI.

The children were characterised by 4 types of deafness; congenitally severe-profound, congenitally progressive, acquired severe-profound, and acquired progressive deafness. Congenital and acquired described the presence of any degree of hearing impairment at birth. The nature of their hearing loss was either ‘progressive’ or ‘severe-to-profound’: describing the degree of loss at initial diagnosis. Hearing aid usage and history was noted where the information was available.

Three out of 16 children had the Nucleus 5 CP810 processors and its accompanying internal component (CI512), and the rest were users of the Freedom speech processor, matched to a CI 24RE internal implant, except participant 16 who had a CI24M implant. Twelve out of the sixteen children had their processors set to a 900 Hz rate, while the others varied between 2400, 1800 and 1200 Hz. All users had their processors set to ACE strategy on monopolar stimulation of MP1+2 (see Appendix 2).
3.2 Experimental procedures outline

All participants were contacted after being identified by their audiologists as appropriate for the study. The experimental sessions were booked in either before or after their audiology appointments for ease of travel and to help recruitment.

3.2.1 Part I

Adults were tested in a single session of approximately one hour, where their CAEPs to three phonemes were obtained through two evoked potential systems. This was conducted usually after their audiology appointment, where speech perception scores were also obtained. Four of the twenty four adult CI users did not complete their speech perception tests on the same day as their CAEP recordings due to demands of clinical appointments. However, their speech perception scores were obtained within one month of CAEP responses.

Figure 12: Outline of study progression and difference between parts I and II of the investigation

Part I: Adult CI users

Session One:
- CAEP recordings from the HEARLab and Bio-logic Evoked potentials systems
- Speech perception scores

Part II: Children CI users

Session One:
- CAEP recordings from the Bio-logic Evoked potentials system

Continued CI usage, auditory verbal therapy and schooling

Session Two:
- CAEP recordings from the Bio-logic Evoked potentials system
3.2.2 Part II

CAEPs were similarly obtained in the children using only one evoked potential system. Testing was conducted twice with approximately 3 months (±2 weeks) in between the sessions. During the three months in between, the children carried on as usual and continued to receive auditory verbal therapy or teacher aid at school as they normally would. Each session took approximately one hour.

3.3 Speech perception testing

3.3.1 Tests

The adult CI users were tested using the HINT© (Hearing In Noise Tests) sentences (Nilsson, et al., 1994) and CNC (Consonant Nucleus Consonant) word lists (Lehiste & Peterson, 1959). The tests used were re-recorded versions with Judy Bailey, a female New Zealand native speaker. The sentences were administered in lists of 10, and an average score from two lists were obtained for two conditions: sentences in quiet, and a 10dB signal-to-noise ratio (SNR). The CNC word lists consisted of lists of 50 words, but only half the list was administered from which an average score for words and phonemes were obtained. All testing were conducted in the auditory alone fashion, and a single practice list was provided first to refresh the protocol for each participant. The lists tested were randomized across the group.

3.3.2 Experimental set up and calibration

Speech perception testing was conducted within two sound-proofed clinic rooms at the University of Auckland Clinics. In the first clinic room, the tests were administered through Windows media player software on a desktop computer and a Madsen Auricle Audiometer, delivered through a Bower & Wilkins Vision DS1 loudspeaker. In the second clinic set-up, the tests were played from DVD recordings via a Sony DVP-NS508P DVD player. Sound was delivered through a Wharfedale Modus 1 Speaker, amplified by a TOA PA amplifier (Model A-1031). Images were not displayed from the television as all speech perception testing were auditory alone.

Both sets of equipment were calibrated clinically for the CI programme, which was checked with a sound level meter on a weekly basis. All tests were delivered at 55dB SPL to the participant sitting at 1 meter away at 0° azimuth.
3.4 Cortical Auditory Evoked Potentials

3.4.1 Room configurations

CAEP testing for both part I and II were conducted at the University of Auckland clinics and the Hearing House respectively. In both cases the participant was seated at one meter from the loudspeaker, at 0° azimuth. A television or computer situated behind or below the speaker, played silent movies at their approximate head height to maintain their gaze and attention. The floor was avoided during placement of all wires connected to electrical sources to minimize electrical interference. All redundant connections to electrical sources in the room or nearby vicinity was switched off or removed.

Part I

To improve the acoustics of the partially sound-proofed room, 1200 cm x 1200 cm acoustic foam tiles were fixed directly behind where the participant’s head, in the path of oncoming sound.
Loudspeaker was mounted upon an adjustable stand which was adjusted according to head height of the participant when seated. The computer screen for displaying silent DVDs was slightly positioned to the participant’s right to avoid the speaker stand, and mark was made on the opposing wall as a reminder of head position.

**Part II**

*Figure 14: Room set-up at the Hearing house. Speaker is set at 1 m away on top of the television which plays silent DVDs. Children younger than 3-4 may opt to sit on top of parent’s lap or toddler seat*

All testing was conducted in a sound-proofed audiology clinic room. Children younger than 4 years sat either in another toddler seat or on top of their parent’s lap. A distracter, either parent or audiologist, also assisted with keeping the child seated, calm and quiet during testing. The distracter sat or crouched slightly to the side of the child, avoiding the projected path of the stimuli.

**3.4.2 Stimuli characteristics**

The stimuli used were three speech phonemes /m/, /g/, and /t/. These originated from research with National Acoustics Laboratory (NAL) in Sydney (Frye Electronics Inc, 2010). Based on the International Long-term Average Speech Spectrum (ILTASS), continuous speech spoken by a female was extracted and filtered to reflect levels within a few decibels. The /g/ and /t/ phonemes were high-pass filtered at 250 Hz to minimize low-frequency noise (Frye Electronics Inc, 2010).
As these phonemes are the default stimuli for the HEARLab system, they were generated and amplified via the stimulus controller. For the Bio-logic system, they were uploaded as separate custom sound files. These original phonemes had to be converted from a 44100 Hz to a 48000 Hz sampling rate in Adobe Audition 1.0 software, to be accepted by the Bio-logic software. Due to the sampling rate conversion, the duration of the stimuli was altered slightly between the versions delivered by each system. The original HEARLab phonemes are 30 ms long for /m/ and /t/, and 20 ms for /g/. The conversion reduced the duration of the phonemes to 27 ms for /m/, /t/, and 19 ms for /g/. The shorter /g/ phoneme was designed to help filter out additional low frequencies from the mid frequency spectra (Golding, et al., 2006); which may have affects on amplitudes of the P1 response. The voicing and place of articulation of each speech phoneme used was also different. The /m/ was a voiced nasal, /g/ voiced plosive, and /t/ a voiceless plosive. The voice-onset-time (VOT) also varies between these phonemes. However, this is insignificant as these phonemes are essentially ‘vowel-free’ and contain minimal vowel transition (R. Martin, Villasenor, Dillon, Carter, & Purdy, 2010).

3.4.3 Stimuli transduction

The stimuli were played through the Edifier MP210 active speaker to ensure it was not significantly altering speech characteristics spectrally or temporally. Initially, a white noise of 0.4 seconds in duration, and peak at -3.25 dB, was generated in Adobe Audition 1.0 software, and uploaded through the Bio-logic system as a custom sound file, as there are no parallel options through the HEARLab system. The noise was presented at 80 dBnHL and captured for 30 seconds. Upon analysis, it was assured that no loudness distortion was caused by the speaker.

The phonemes /m/, /g/ and /t/ were then delivered through the Bio-logic after file conversion. These phonemes were set to present at 65 dBSPPL output at a rate of 0.9/s in the stimulus settings, prior to speaker calibration. All stimuli were recorded by a Bruel & Kjær precision Sound Level Meter 2235 with a B&K 4176 microphone at the approximate head level of the participant. The recordings were captured in real-time for 3 minutes and saved as ‘.wav’ files in Adobe Audition, via an output line from the sound level meter. Twenty five copies were randomly sampled from the 3 minutes, and excised into individual ASCII (.txt) files which were exported into Microsoft Excel.
To examine the transduced stimulus temporally, they were averaged and plotted alongside the original electronic copy of the phoneme with the same time factor. The root mean square (RMS) of each individual temporal waveform was also calculated, and the maximum and minimum waveforms were identified (shown in grey in Figure 15). Each acoustic dataset was scaled by a factor of its largest peak for comparability with the electronic data.

To examine the transduced stimulus spectrally, a fast fourier transform (FFT) was calculated from the temporal waveforms, and overlaid with the original phoneme file for visual comparison. Peaks from the concentration of spectral energy for each phoneme are shown in Table 1 below. Note that a lower frequency spectral peak for /t/ was apparent after transduction through the speaker, although the concentration of spectral energy still concurs with the original /t/ phoneme.

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>Spectral peak of acoustic stimuli (Hz)</th>
<th>Spectral peak of electronic stimuli (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>/m/</td>
<td>257.9</td>
<td>257.9</td>
</tr>
<tr>
<td>/g/</td>
<td>1313.9</td>
<td>1313.9</td>
</tr>
<tr>
<td>/t/</td>
<td>3727.5</td>
<td>2625.7</td>
</tr>
</tbody>
</table>

Table 1: Comparison of peak frequencies for acoustic (original) and electronic (transduced) versions of /m/, /g/, and /t/.

Figure 15: Below shows comparisons of the three phonemes before and after transduction through the speaker, both temporally and spectrally. For all temporal comparisons, the maximum and minimum waveforms are in grey to represent the variability.
Temporal waveforms
Spectral Waveforms
3.5 CAEP systems configuration and testing procedures

3.5.1 Calibration

The speech stimuli were calibrated for the Bio-logic® system at both locations in part I and II of the investigations. The Bruel & Kjær sound level meter was used to monitor that the output level at the position of the participants’ head while seated, remained 65 ±3 dB(Z) SPL. The frequency analyser mode was selected and a LZI weighting was used in the sound level meter. The LZI function incorporates the frequency weighting ‘Z’ or ‘zero’ from the International Standard IEC 61672 2003 which has a flat response of 10 Hz to 20 kHz ±1.5 dB. The Z weighting was chosen over the commonly used A-weighting as it responds more linearly across the frequency range. A-weighted responses begin to roll off at 1 kHz; which may misrepresent levels of the /m/ phoneme which peaks around 250 Hz (IEC, 2003). The ‘I’ or ‘impulse’ function is suggested for stimuli duration which were shorter than 35 ms. Levels were rechecked weekly with a sound level meter over the duration of the study.

For the HEARLab system, the software limits the selection of presentation levels to 55, 65, and 75 dB SPL. The same calibration method was used as above, as well as using the volume control on the active Edifier speaker to attain more accurate output of 65 ±3 dB(Z) SPL. For a comparison of calibrated input levels, see Appendix 3.

3.5.2 Evoked potential systems set up

Two evoked potential systems were used in part I of the investigation: the Bio-logic® Navigator® PRO and the HEARLab from National Acoustics Laboratory and Frye Electronics. Both systems were made compatible with an Edifier MP210 active speaker via an audio connecting cable and a transformer. A connecting cable with mono to mono plugs was used to switch from one system to another to ensure the stimulus was delivered through the same speaker.

3.5.2.1 Bio-logic® stimulus and recording parameters

The Bio-logic® Auditory Evoked Potentials version 7.0.0 software was used to deliver the stimuli. Stimulus, amplifier and acquisition parameters shown below were customised for each phoneme as individual collection protocols in the software.
<table>
<thead>
<tr>
<th>Stimulus</th>
<th>/m/</th>
<th>/g/</th>
<th>/t/</th>
</tr>
</thead>
<tbody>
<tr>
<td>Output level (dBnHL)*</td>
<td>65</td>
<td>65</td>
<td>65</td>
</tr>
<tr>
<td>Duration (ms)</td>
<td>27</td>
<td>19</td>
<td>27</td>
</tr>
</tbody>
</table>

Transducer type: Insert Earphones**
Ear: Binaural
Polarity: Alternating
Presentation Rate (per second): 0.9, 1.8
Inter-stimulus interval (ms): 1111.11, 555.56
Step (dB): 5
Trigger in/out: Off
Continuous stimulus: Off

**Amplifier**
Gain: 50000
Artefact rejection (µV): 70
Low filter (Hz): 1 (12dB/octave)
High filter (Hz): 30
Electrode switching: Off
Input 2: A1

**Acquisition**
Time window Epoch (ms): 533
Pre-stimulus (ms): 33
Number of points sampled: 256
Number of averages: 75
Number of repeats: 3 runs in adults, at least 2 runs in children
Blocking: None
Noise stop: Off
FSP: Off

Table 2: Settings selected in the Bio-logic AEP software for all CAEP recordings
* Note that the input levels after calibration were different for Part I and II of the investigation at different locations. These calibrated intensities are displayed in Appendix 3.
** Insert earphones had to be selected in the parameters although the stimuli were delivered in sound field, to achieve the appropriate calibrated output levels.

3.5.2.2 HEARLab system stimulus and recording parameters

The HEARLab software has pre-set parameters such as stimulus, amplifier and recording parameters set as default selections which cannot be changed by the user. Table 4 shows the selection of settings made and some of the known parameters of the system. All CAEP were conducted in the ACA (Aided Cortical Assessment) mode.
Figure 16: Components of the HEARLab CAEP system and the flow of signal. The blue circles are the active electrodes to be clipped onto gel electrodes on the head. The burgundy lines represent the interchangeable CI connectors were used in place of original connectors during this study (Frye Electronics Inc, 2010).

<table>
<thead>
<tr>
<th>Stimulus</th>
<th>/m/</th>
<th>/g/</th>
<th>/t/</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation level (dBnHL)*</td>
<td>65</td>
<td>65</td>
<td>65</td>
</tr>
<tr>
<td>Duration (ms)</td>
<td>30</td>
<td>20</td>
<td>30</td>
</tr>
</tbody>
</table>

Table 3: Settings in the HEARlab software for ACA mode

*For calibrated input levels, see Appendix 3

**The ISI of 1125 is based on studies of CAEP recordings in adults (Hyde, 1997; Stapells, 2002), and a NAL study in /m/ and /t/ stimuli in infants (Golding, et al., 2006). Golding and colleagues found a significant increase in CAEP responses when the ISI increased from 79 to 1125ms, but not in responses with larger ISI.
3.5.3 Electrode montage

A single channel electrode montage was used for all participants:

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Recording position</th>
<th>Charge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cz</td>
<td>Vertex</td>
<td>+</td>
</tr>
<tr>
<td>Fz (Ground)</td>
<td>Nasium</td>
<td>Ground</td>
</tr>
<tr>
<td>A1/Reference</td>
<td>Earlobe contralateral to CI</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 4: Electrode montage showing the recordings sites of the electrodes and their charge

The reference electrode was placed behind the ear lobe of the ear contralateral to the implanted ear (test ear); to reduce artefact in the recordings.

Figure 17: General electrode configuration from cranial view. The blue circles identify approximate positions of the recording electrodes. Note that the test and contralateral ears may be vice versa on different participants.

In bilateral cochlear implant users, one ear was chosen as the test ear while the contralateral cochlear implant was removed or switched off. The test ear was the best performing ear, or in cases where this was difficult to ascertain, the ear which was implanted first.

3.5.4 Recording procedure

Each participant was seated in a comfortable chair, with the speaker adjusted to their head height at 1 m away. They were instructed to make themselves as relaxed, comfortable and silent as possible. It was explained that excessive eye blinks, head movement, neck and
muscle tension would contribute to noise in the recordings. A neck cushion was available where needed.

The participants watched a silent and subtitled DVD for the duration of the recordings. This was used to maintain their alertness and position of their head. Young children were also kept occupied with silent toys such as soft toys, puzzles, stickers and bubbles with the assistance of a parent or audiologist.

The skin area was cleaned and prepared with an abrasive skin gel (NuPrep\textsuperscript{TM}) and preparation tape (Red Dot Trace prep tape). Ambu Blue Sensor EEG recording electrodes were then applied according to the electrode montage. Size SP-00-S were used for adults, and size N-00-S were used on children, as well as adults with small earlobes. All attempts were made to keep the impedance at \(<5\) kOhms.

For both part I and II, the Bio-logic\textsuperscript{®} system was used to acquire CAEPs in 6 conditions. Two rates of presentation, identified as slow and fast rate (0.9/s and 1.8/s respectively), were used. CAEPs were acquired for each speech stimuli with both rates. A random number generator was used to decide the order of acquisition across the 6 conditions. Three runs were conducted for each condition and averaged with the software.

For part I of the study, the order of recording from either system was decided via a random number generator. Once all recordings were made, the connecting audio cable was switched to the second system, and electrode connectors of the second system was attached to the blue sensor recording electrodes which remain on the participants’ scalp. Impedances were always monitored to remain stable and appropriate after changing to the second system.

For CAEPs acquired through the HEARLab system, two runs of at least 75 accepted acquisitions were recorded from each phoneme. The order of acquisitions were decided by a random number generator as well. The statistical analysis window was ignored in the clinical judgement of the response.
3.5.5 Exporting Data for Analysis

3.5.5.1 Bio-logic data

An average of traces was obtained for each condition in all participants for the Bio-logic AEP software. Each calculated average was exported using AEP to ASCII© version 1.6.0, with ‘tab delimited’ selected as the output format and saved as an ASCII (.txt) file. These files were copied and pasted into a Microsoft Excel spreadsheet, plotted with the appropriate time interval. The waveforms were then plotted together to compare slow and fast rates (long and short ISI), inter-stimulus and inter-participant variability.

3.5.5.2 HEARLab data

To export each run of CAEP, ‘export run data’ was selected after right-clicking on the record in the run selections window. The raw data was saved as a .txt file and exported similarly into Microsoft Excel.
3.6 Waveform Analysis

All analyses henceforth refer to waveforms as P1, N1, and P2; the measures as peak latency, amplitude, area 1, area 2 and peak-to-peak amplitudes; and stimuli as the phonemes /m/, /g/, and /t/.

3.6.1 Characterisation of the stimulus artefact

The stimulus-associated artefact in CAEP recordings were identified using the following criteria into four categories below: null, blip, pedestal and undefined. The typology, peaks of the blip and pedestal artefacts, and the end latency of the blip artefacts were characterised and agreed by two individual observers. Judgement of type of artefact was aided by viewing an overlay of traces from both rate conditions on one graph, and also with only the slow rate condition CAEP for all three phonemes.

3.6.1.1 Null artefact

- No distinctive deflections of positivity or negativity is present with significant magnitude compared to the P1, N1 or P2 response. The CAEP trace prior to P1 or N1 in adults (in the absence of P1) should be around 0 µV after baseline correction.

![Figure 18: Example of a CAEP without contamination from any artefact; categorized as a ‘null artefact’ response from adult participant 4 for stimulus /m/.](image)

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3.6.1.2 Blip artefact

- Short and rapid positivity post-stimulus onset, preceding the P1 or the N1 response in adults
- Smaller in magnitude than P1, N1, and P2 waveforms
- Can be distinctly separate or partially invasive of P1 waveform (Figure 19)
- Present usually in both rate conditions, in which amplitude remains similar
- It is commonly present in all 3 phonemes, if present at all. Thus it is often easily identified via overlaying the traces of all 3 phonemes from the slow rate condition
- Can present as a negativity wave on some occasions; mirroring the short and rapid duration seen in the positive blip artefact
- Peaks of blip artefact were picked as the point of maximum amplitude during the sharp deflection.

![Blip Artefact Graph](image)

**Figure 19: Example of contamination from a ‘blip’ artefact from the /g/ stimulus from adult participant 5. Here the artefact was repeatable in both rate conditions and is likely to be contaminating the P1 response.**

**Blip artefact offset latency**

The offset latency was defined as the point where the artefact is expected to have finished. All offset latencies of the blip artefact were recorded; regardless of degree of contamination of P1. The average, standard deviation and 95% confidence intervals of the offset latency were calculated.
3.6.1.3 Pedestal artefact

- Consists of a sharp deflection of an abnormally large amplitude of at least 5 μV, resembling a pedestal-like morphology (Gilley, et al., 2006; Xiaoxia, et al., 2010)
- Can be observed to contaminate the entire P1-N1-P2 response or altering its morphology. Often parts of the response may be apparent if the pedestal artefact is smaller in amplitude (Gilley, et al., 2006)
- Usually recovers gradually back to baseline within the recording epoch
- Present at both rate conditions
- Preceded by a blip artefact in most cases
- Peak of pedestal was picked as point of maximal amplitude at the peak of negative deflection

![Graph showing pedestal artefact](image)

Figure 20: Example of pedestal artefact in the response to /t/ phoneme in participant 21

3.6.1.4 Undefined morphology

- In some traces, the portion of the CAEP prior to P1 or N1 waveforms could not be classified as a blip nor null artefact, and it was clearly not a clean CAEP.
- The response is likely to be contaminated from noise and invasive of either P1 or N1 waveforms
3.6.2 Baseline Correction

The Bio-logic® system software did not account for the variations in the acquisition due to noise, thus baseline correction had to be manually applied. The amplitudes of pre-stimulus period (as shown in green in Figure 21) was averaged and multiplied for the amplitude across the rest of the waveform. The HEARLab accounts for baseline correction within its software, and no manual corrections were made.

3.6.3 Marking waveforms by visual identification

The peak amplitude and latency for waveforms P1-N1-P2 in adults and P1 in children were marked in the CAEPs from slow rate conditions. Two independent observers identified the markers, one of which was blinded from participants’ identities. A unified agreement was reached following discussion when the parameters picked were inconsistent. For the adults, the onset of N1 and offset of P2 was also marked to assist area calculations. The P1 onset and N250 was also marked for CAEPs in the children.

![Figure 21: Landmarks on an adult CAEP waveform. The P1, N1, P2 peaks are as shown. Light green shading indicates the period used for baseline correction. Red region indicates period of time for which blip artefact is likely to occur, with the 95% confidence interval of offset latency marked by dotted black line. The blue shading indicates the area 2 for N1 and P2, and the addition of both blue and yellow regions equate to area 1.](image-url)
For waveforms where large peak of similar amplitudes are observed, the peak closest to the expected latency for normal hearing individuals was chosen, provided that it was uncontaminated by noise. In cases where a broad region of similar amplitude was observed, the centre of the broad region was marked as the peak.

In the adults, P1-N1-P2 complex was marked as shown in Figure 21, with expected latencies around 50 ms, 80-100ms, and 200 ms respectively. P1 was observed to be much smaller or absent in adults, often difficult to differentiate from stimulus artefact, noise or shoulder of the falling negativity of the N1 response. On such occasions in adults, P1 was noted to be absent. In such cases, N1 onset was marked and defined as the point which N1 wave confidently begins sloping negatively without contamination from the artefact. The P2 offset was also identified as the point where the falling P2 slope returns to baseline. Lastly, the N1 to P2 peak-to-peak amplitude was computed from the N1 and P2 absolute amplitude.

For children under the age of 12 years, the P1 response dominates the CAEP and is a very prominent positivity of amplitude greater than average adult CAEPs (P. Davis, 1939; Hall, 1992). The N250 or N2 responses was also marked in the children, and defined as the negativity following the large P1 response with maximal negativity around 200-250 ms.

3.6.4 Estimating area under the CAEP waveform

Two methods were used to estimate the areas of N1 and P2 in the adults and P1 in the children, termed as area 1 and area 2. These are depicted in Figure 21.

3.6.4.1 Area 1

<table>
<thead>
<tr>
<th>Waveform</th>
<th>Duration of response for which area is calculated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults</td>
<td>N1 onset (or P1 peak) to P2 peak</td>
</tr>
<tr>
<td></td>
<td>N1 peak to P2 offset</td>
</tr>
<tr>
<td>Children</td>
<td>P1 onset to N250 (P1 offset)</td>
</tr>
</tbody>
</table>

Table 5: Criteria used to identify area 1

1. A straight line was constructed onto the CAEP response in Microsoft Excel joining the two points that frame the duration of the response as described by table 5.
2. The linear equation of the line was obtained in the form of $y = mx + c$ (where $x =$ Time in milliseconds, $y =$ amplitude in microvolts, $x =$ gradient of the straight line, and $c =$ $y$-axis intercept)

3. The equation was used to calculate the amplitude values for the points along the constructed straight line with the same time intervals as the CAEP waveform.

4. The difference in amplitude between the straight line and CAEP response was obtained and then multiplied by the time interval for each data point ($2.08$ ms).

5. The sum of difference in amplitudes across the duration of the response was calculated. Any negative values were excluded from the sum and attributed to noise in the waveform.

### 3.6.4.2 Area 2

<table>
<thead>
<tr>
<th>Waveform</th>
<th>Duration of response for which area is calculated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults</td>
<td>$NI$ N1 onset (or P1 peak) to horizontal bisection of rising slope of N1 wave</td>
</tr>
<tr>
<td></td>
<td>$P2$ P2 offset to its horizontal bisection of rising slope of N1 wave</td>
</tr>
<tr>
<td>Children</td>
<td>$PI$ P1 onset or artefact offset to its horizontal bisection of falling slope of P1 wave</td>
</tr>
</tbody>
</table>

Table 6: Criteria for marking the area 2

1. From the onset or offset of the waveform as described in table 6 above, a horizontal straight line bisecting the opposite half of the waveform response was drawn. These are the two points used to frame the area of the response.

2. The difference in amplitude along the time intervals were calculated by subtracting the CAEP amplitudes from the amplitude of the onset or offset in step 1 (which remains the same). The reverse subtraction was made for P2 and P1 area calculations.

3. The area of the response was then calculated following steps 4 and 5 from method 1 for area 1.
3.7 Data treatment and analyses

3.7.1 Part I

3.7.1.1 Stimulus effects on CAEPs

Repeated measures ANOVA was performed to explore the effects of the stimulus on 5 measures of N1 and P2 waveforms separately. The measures included peak latency, peak amplitude, area 1, area 2 and peak-to-peak amplitude of N1-P2. Mauchly’s test of Sphericity was utilised to confirm that the ANOVA assumptions were met. Where these were not met, the Huynh-Feldt correction was applied to the model and its significance reported. In such cases the true degrees of freedom were still reported. Tests of within-participant effects were investigated for significance of $p<0.05$.

Where significant omnibus results were obtained, paired samples t-tests were conducted to see if there was further difference between pairs of phonemes. It is not desirable to conduct multiple statistical comparisons due to increased type I error rates. Therefore, to minimise the number of comparisons made, in cases where the gradient between /m/, /g/, and /t/ phonemes was monotonic, pairwise comparisons were made between /m/ vs. /g/, /g/ vs. /t/ phonemes; where it was non-monotonic, an additional pairwise comparison was made between /m/ and /t/. Given these repeated pairwise tests, Bonferroni correction of p-values to control the type I error rate was considered. However, it was decided not to apply a correction, given this would increase the type II error rate unacceptably due to the sample size.

3.7.1.2 Speech perception and CAEPs

The four main measures of speech perception explored were HINT sentences in quiet, HINT with 10 dB SNR, CNC words and CNC phonemes. Z-scores for these tests were generated to account for the different variability in the range of scores, and a composite score for speech perception was then generated from these measures. This composite was tested and shown to be reliable (Cronbach’s alpha=0.934), implying that all four measures were consistent and comparable. Results from the CNC phonemes were analysed for the percentage of correctly identified phonemes scored for each of the stimuli tested (/m/, /g/, and /t/). This speech measure was termed the identification rate.
Based on evidence in the literature (Kelly, et al., 2005; Makhdoum, et al., 1998), least squares linear regression analyses were performed to examine if of speech perception ability predicted P2 latency for the low frequency phoneme /m/. Exploratory bivariate correlations were also performed to compare the composite speech measure against all measures of N1 and P2 (latency, amplitude, area 1, area 2 and peak-to-peak amplitude) for all phonemes (number of comparisons=27).

The identification rate for the three phonemes was compared using repeated measures ANOVA. Post hoc paired t-tests were conducted. A second set of bivariate correlations was conducted for identification rate of each phoneme from the CNC lists. For the relevant phoneme, these were compared with latency, amplitude, area 1, area 2 and peak-to-peak amplitude (9 measures for each phoneme).

3.7.2 Part II

An effect of stimuli on the measures of the P1 waveform was investigated in the child participants. The same approach was taken as part I, where paired samples t-tests were conducted if significant differences were observed across stimuli with repeated measures ANOVA.

Least squares linear regression analyses were conducted to assess whether chronological age of the child and time with a CI (CI age) predicted changes in CAEP measures. Change in measures between the two test sessions was modelled by controlling for the measure at time one in models of the same measure at time 2. This approach was used to model the effect of both age and time with a CI on change in each of the four measures.
CHAPTER 4 RESULTS

4.1 Part I: Adult CI users

4.1.1 Speech Perception Scores

Adult speech perception scores were obtained in all but one participant (participant 17) who is not included in this data set. Participant 17 struggled with the material and testing was ultimately abandoned to make changes to his map. It was also unfeasible to rearrange another appointment time. On average, the adults scored much better for the HINT sentences in quiet and poorest for CNC words. The scores for HINT with a +10dB SNR talker babble had the widest distribution of scores. Overall, the largest distribution of scores was observed for HINT +10dB SNR, and the three quarters of the scores were above 80% for HINT scores in quiet. The distribution of scores was fairly even between the CNC word and phoneme tests.

![Box plot of speech perception test scores](image)

Figure 22: Distribution of speech perception test scores for all adults except for participant 17 (n=23). The 10th percentile, lower quartile, median, upper quartile and 90th percentile in the box plot, with outliers in black dots.
Speech perception tests | HINT | CNC
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HINT</td>
<td>HINT</td>
<td>CNC words</td>
<td>CNC phonemes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>In quiet (%)</td>
<td>+10dB SNR (%)</td>
<td>(%)</td>
<td>(%)</td>
<td></td>
</tr>
<tr>
<td>Mean score</td>
<td>85.1 (21.9)</td>
<td>61.2 (32.4)</td>
<td>52.9 (20.8)</td>
<td>71.2 (18.9)</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>22-100</td>
<td>0-98</td>
<td>4-88</td>
<td>19-95</td>
<td></td>
</tr>
<tr>
<td>95% confidence intervals</td>
<td>76.2-94.1</td>
<td>48.0-74.5</td>
<td>44.4-61.4</td>
<td>63.5-78.9</td>
<td></td>
</tr>
</tbody>
</table>

Table 7: Summary of adults’ speech perception scores (n=23). Standard deviations of the mean are shown in parentheses.

The identification rate was obtained for the percentage of correctly identified /m/, /g/, and /t/ from the CNC phonemes list (see Table 7). On average, participants identified /t/ from CNC tests with the greatest accuracy, followed by /g/ and then /m/. The widest variability in the identification rates were found for the /g/ phoneme.

| Adults | CNC phoneme identification rate (%) |  
|--------|-----------------------------------|---
| /m/    | Mean (SD)                         |  
| 58.6 (26.1) |                      |  
| /g/    | Mean (SD)                         |  
| 65.9 (37.1) |                      |  
| /t/    | Mean (SD)                         |  
| 89.0 (22.5) |                      |  
| Range                                         |  
| 0-100 | 0-100 | 0-100 |
| 95% confidence intervals | 47.9-69.2 | 50.8-81.1 | 79.8-98.2 |

Table 8: Summary of identification rate for individual /m/, /g/, and /t/ phonemes in the CNC test (n=23).

### 4.1.2 Cortical Auditory Evoked Potentials

#### 4.1.2.1 Adult CAEP morphology

Cortical auditory evoked potentials were reliably recorded in the majority of adult participants. Figure 23 shows the grand average P1-N1-P2 complex recorded in participants 1-22. The grand average shows P1 latency at approximately between 50-80 ms, N1 at 100 ms and P2 at 200 ms. In some individuals, not all peaks may be present. This was most commonly found with the P1 waveform, thus P1 responses are not reported in the adults as few were reliably free of artefact. Abnormal CAEP morphology was observed for all phonemes in participants 23 and 24 (Figure 24), similar to responses from the children and were thus separated during the analysis.
4.1.2.2 Participants P1-22

CAEP recordings for all phonemes, presented with normal age-appropriate morphology in the adult CI users P1-P22 (n=22), are summarised in Figure 25 and Table 9 below. In this sample, no observable CAEP peaks were found for seven phonemes from four participants, either due to true absence of the waveform or difficulty defining the waveform due to noise and artefact. N1 of the CAEP was also absent for four phonemes across two participants. Absent peaks were distributed across the three speech phonemes. The rest of the CAEPs all included measureable N1-P2 waveforms. Latency and amplitude was identified for N1 (n=50)
and P2 (n=54) responses and the subsequent area-under-the-curve (AUC) was estimated using two methods. All participants reported that the phonemes were perceived to be different from one another but did not sound like speech. Many claimed that they heard artificial clicks or bangs due to the short duration of the sounds.

![Waveform diagram](image)

**Figure 25: Grand average waveforms for the three stimuli, in adult participants 1-22 (n=22)**

<table>
<thead>
<tr>
<th>Adult CAEP (P1-P22)</th>
<th>N1</th>
<th>P2</th>
<th>N1 Area (ms.µV)</th>
<th>P2 Area (ms.µV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak Latency (ms)</strong></td>
<td>113.3 (14.3)</td>
<td>204.2 (25.8)</td>
<td>244.1 (137.1)</td>
<td>459.5 (200.0)</td>
</tr>
<tr>
<td><strong>Peak amplitude (µV)</strong></td>
<td>-2.9 (1.9)</td>
<td>2.5 (1.1)</td>
<td>171.9 (121.8)</td>
<td>281.6 (128.9)</td>
</tr>
<tr>
<td>Range</td>
<td>79.4-152.3</td>
<td>152.3-281.4</td>
<td>19.4-535.3</td>
<td>115.6-973.1</td>
</tr>
<tr>
<td><strong>95% Confidence Intervals</strong></td>
<td>109.3-117.3</td>
<td>197.3-211.1</td>
<td>206.1-282.1</td>
<td>406.2-512.9</td>
</tr>
<tr>
<td></td>
<td>-2.3- -3.4</td>
<td>2.2-2.7</td>
<td>138.1-205.7</td>
<td>247.2-316.0</td>
</tr>
</tbody>
</table>

Table 9: Summary of adult CAEP measures averaged across stimuli for participants 1-22 (n=50 for N1 measures and n=54 for P2 measures). Standard deviations of the mean are shown in parentheses.

The average P2 latency was later than P1 latency as expected, while the waveforms’ absolute amplitude appeared similar. For both N1 and P2 waveforms, area 1 gave the largest average estimate, but possessed the most variability for all area estimates.
4.1.2.3 Participants 23 and 24

Table 10 and Figure 26 below summarises Participants 23 and 24 in the adult CI users group. These participants displayed abnormal CAEP morphology for their age; dominated by a large P1 response. This morphology was recorded from all phonemes in both participants.

Participant 23 was diagnosed with bilateral sensorineural hearing loss and fitted with hearing aids at the age of 2 years. It is likely that this cause is congenital as there is a possible X-linked genetic aetiology due to the presence of sensorineural hearing loss in the females of her family. The hearing loss was progressive and a large decline in her hearing was reported after an episode of vertigo attack in 2003 when she became profoundly deaf, and was referred to the CI programme. She received her implant 4 years later in 2007, and has been a consistent user since (CI age= 41 months). A congenital profound hearing loss was diagnosed in participant 24 at 1 year of age. She was fitted with hearing aids at 14 months and implanted at 7 years and 2 months. She has been a consistent user since, with a CI age of 158 months.

Participant 24 had abnormally large amplitude across CAEPs from /m/ and /g/ phonemes, while amplitude across phonemes in participant 23 was more consistent with expected adult amplitudes. Both displayed late P1 latencies expected for normal hearing infants under the age of 2 years. Area 1 measures were consistently larger than area 2 measures as seen in other adult participants as well. Due to absence of N1 and P2 waveforms, further investigation of effects on stimuli and behavioural speech perception data were not inclusive of participants 23 and 24.

Figure 26: CAEPs for all phonemes from participants 23 and 24 displaying presence of the P1 waveform across all ages
### Table 10: Average P1 measures of peak latency, amplitude and area estimates across phonemes for participants 23 and 24 (n=6).

<table>
<thead>
<tr>
<th>Adults CAEP (P23 and P24)</th>
<th>P1 peak measures</th>
<th>P1 area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Latency (ms)</td>
<td>Amplitude (µV)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>166.2 (29.3)</td>
<td>3.4 (1.1)</td>
</tr>
<tr>
<td>Range</td>
<td>131.5-198.1</td>
<td>2.5-5.1</td>
</tr>
<tr>
<td>95% Confidence Intervals</td>
<td>142.7-189.7</td>
<td>2.6-4.3</td>
</tr>
</tbody>
</table>

#### 4.1.3 Stimulus effects on CAEPs

For each waveform, the distribution of all CAEP measures were plotted and compared across stimuli. For further statistical analyses the data became limited to 15 of the 22 adult participants who had measureable CAEPs for all three phonemes.

##### 4.1.3.1 Latency

N1 latency was the longest for /m/ (median 122.1), compared to latencies for /g/ (median 106.5) and /t/ (median 106.5). P2 latency for stimulus /m/ (median 209.6), was also longer than /g/ (median 200.2) and /t/ (median 191.9). Overall, the spread of latencies were more variable for P2 than N1 waveforms.

![Figure 27: Box plot showing 10th percentile, lower quartile, median, upper quartile and 90th percentile for N1 and P2 peak latencies. Dots indicate outliers for each data set and the sample size (n) for each stimulus for each phoneme is reported above each plot.](image-url)
For the 15 participants from whom measurable CAEPs were obtained from all stimuli, repeated measures ANOVA for N1 latency revealed a significant difference across phonemes, $F(2,28)=9.53, p=0.001$. Pairwise comparisons revealed that there was only a difference between /m/ (mean 121.9, SD 12.3) and /g/ (mean 107.6, SD 9.4) latencies, not between /g/ and /t/ (mean 105.2, SD 12.4). N1 latency is therefore significantly later for /m/ than for the other phonemes. There was also an overall difference across phonemes for P2 latency, $F(2,28)=4.56, p=0.019$. Pairwise comparisons (Table 1) showed that /m/ (mean 208.4, SD 16.5) was different from the other two phonemes, while /g/ (mean 193.9, SD 19.8) and /t/ (mean 196.7, SD 24.5) latencies were not different.

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1 latency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>5.09 (14)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>0.54 (14)</td>
<td>0.597</td>
</tr>
<tr>
<td>P2 latency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>3.83 (14)</td>
<td>0.002</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>-0.48 (14)</td>
<td>0.637</td>
</tr>
</tbody>
</table>

Table 11: Paired t-test statistics for N1 and P2 peak latencies across all stimuli (n=15)

Figure 28: Effects of stimuli on N1 (left) and P2 (right) mean latencies after analyses for 15 participants who had measurable CAEPs across all stimuli.
4.1.3.2 Amplitude

Figure 29 below shows the spread of amplitudes across stimuli for both the N1 and P2 waveforms in adults. On average, the N1 waveform had larger absolute amplitudes for /m/ (median -3.8) than /g/ (median -2.8) and /t/ (median -2.3). The /t/-evoked CAEPs display a smaller spread of N1 peak amplitudes compared to the other phonemes. This pattern is not observed for P2 amplitudes across stimuli. The size and spread of P2 amplitudes appear to be very similar, across /m/ (median 2.6), /g/ (median 2.2), and /t/ (median 2.5).

A significant difference was observed for all 15 participants who had measureable CAEPs for N1 amplitude across the phonemes, $F(2,28)=3.68$, $p=0.038$. Paired t-tests compared between the pairs /m/ vs. /g/, /g/ vs. /t/, and demonstrated that only N1 amplitude from /m/ (mean -3.4, SD 1.8) was significantly different from /g/ (mean -2.6, SD 1.8), and thus /t/ (mean -2.2, SD 1.2). Repeated measures ANOVA revealed no significant difference across /m/ (mean 2.7, SD 1.0), /g/ (mean 2.7, SD 1.2), and /t/ (mean 2.3, SD 1.2) for the measure of P2 amplitude, $F(2,28)=1.81$, $p=0.183$ (see Figure 30).
<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1 amplitude</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>-2.74 (14)</td>
<td>0.016</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>-0.76 (14)</td>
<td>0.458</td>
</tr>
</tbody>
</table>

Table 12: Paired t-test summaries of N1 peak amplitudes across stimuli (n=15)

Figure 30: Effects of stimuli on N1 (left) and P2 (right) mean amplitudes after analyses for 15 participants who had measurable CAEPs across all stimuli.

4.1.3.3 Area Measures

Two area measures (see methods) were calculated to estimate area under the waveform of N1 and P2. Figures 31 and 32 below reveal that area 2 was consistently smaller than area 1 measures, for both waveforms. The proportional difference between N1 area measures 1 and 2 appears to be larger for /m/ and /t/, as shown from their error bars. However in the P2 waveform, the largest proportional difference between area measures was observed in responses for the /m/ and /g/ phonemes.
Figure 31: Comparison of N1 area measures for adult participants who had measureable CAEPs across stimuli (n=15). The data are grouped by stimuli and error bars show 1 standard error away from mean.

Figure 32: P2 area measures for adult participants who had measureable CAEPs across stimuli (n=15). Columns are grouped by stimuli and error bars show 1 standard error away from mean.
4.1.3.4 Area 1

Estimates from area measure 1 of N1 and P2 waveforms are shown across stimuli from all measurable CAEPs in the box plot below (Figure 33). For both N1 and P2 waveforms, the average area 1 measure appears to decrease from /m/ (N1 median 297.8, P2 median 503.0) to /g/ (N1 median 206.0, P2 median 454.4), even smaller for /t/ (N1 median 168.6, P2 median 389.1). The area 1 measure of N1 and P2 for /t/ is notably less variable than that of the other two stimuli, with a more observable difference in N1 than P2.

Figure 33: Box plot showing 10th percentile, lower quartile, median, upper quartile and 90th percentile for N1 and P2 area 1 measures. Dots indicate outliers for each data set and the sample size (n) for each stimulus for each phoneme are reported above each plot.

Repeated measures ANOVA conducted for the area 1 measure of N1 revealed a significant p-value in Mauchly’s test of sphericity (p=0.020). The Huynh-Feldt correction for sphericity was applied and an effect between N1 area 1 across stimuli was observed, $F(2,28)=6.04$, $p=0.014$. Pairwise comparisons between the same pairs as previous analyses for N1 waveform, revealed no differences (see Table 13). Thus, the significant ANOVA result is likely due to the difference between areas for /m/ (mean 288.7, SD 135.0) and /t/ (mean 174.8, SD 69.2), and less likely from /g/ (mean 253.6, SD 157.0). There was no effect observed for the area 1 measure of P2 waveforms, $F(2,28)=1.07$, p=0.358, from repeated measures ANOVA performed between /m/ (mean 494.8, SD 217.2), /g/ (mean 453.1, SD 217.2).
204.5), and /t/ (mean 416.2, SD 158.2). Grand average CAEPs for adult participants 1-22 (Figure 25) revealed that variability in the size of the P2 wave was smaller compared to N1. The waveform appeared to be very similar in amplitude, and only small differences in latency width across the P2 response were visually observed.

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1 area 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>1.65 (14)</td>
<td>0.121</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>1.88 (14)</td>
<td>0.081</td>
</tr>
</tbody>
</table>

Table 13: Paired t-test statistics for N1 area measure 1 for sample n=15 who had present responses across all stimuli

### 4.1.3.5 Area 2

Area 2 for N1 appears largest in /m/ (median 182.6), but similar for /g/ (median 130.6) and /t/ (median 126.4). Conversely, the area 2 measured in P2 is smallest for /m/ (median 253.9), while /t/ (median 291.2) was larger than /g/ (median 265.4). Similar to the findings of area 1, the spread of data was most discrete for /t/ in measures for the N1 waveform, but this effect was not observed in P2.

![N1 Area measure 2](image1.png)

![P2 Area measure 2](image2.png)

Figure 34: Box plot showing 10th percentile, lower quartile, median, upper quartile and 90th percentile for N1 and P2 area 2 measures. Dots indicate outliers for each data set and the sample size (n) for each stimulus for each phoneme are reported above each plot.
The results of the repeated measures ANOVA showed a significant difference across stimuli observed for the area 2 estimates of N1, $F(2, 28) = 4.31, p = 0.023$. An effect was only revealed between /g/ (mean 198.7, SD 148.9) and /t/ (mean 115.7, SD 59.2), but not /m/ (mean 191.5, SD 113.2) and /g/ after pairwise comparisons (Table 14). Due to the non-monotonic pattern, a third pairwise comparison was made and the difference between /t/ and /m/ was confirmed.

As shown in Figure 25, the average CAEP evoked by /g/ and /m/ are both wider and larger in amplitude than the /t/ response. No difference in P2 area 2 measures across stimuli was found from repeated measures ANOVA results, $F(2, 28) = 0.36, p = 0.699$; consistent with the small variability in size of P2 waveform observed from the grand average CAEPs.

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1 area 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>-0.27 (14)</td>
<td>0.790</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>2.18 (14)</td>
<td>0.047</td>
</tr>
<tr>
<td>/t/ vs. /m/</td>
<td>-2.70 (14)</td>
<td>0.017</td>
</tr>
</tbody>
</table>

Table 14: Paired t-test statistics for N1 area measure 2 for sample n=15 across stimuli

### 4.1.3.6 N1-P2 peak-to-peak amplitude

Repeated measures ANOVA showed a difference across stimuli on the N1-P2 peak-to-peak responses, $F(2, 28) = 4.76, p = 0.017$. However, the paired samples t-test showed no significant differences between the pairs of /m/ (mean 6.0, SD 2.3) vs. /g/ (mean 5.3, SD 2.5) and /g/ vs. /t/ (mean 4.4, SD 1.5) comparisons; thus the stimuli effect from the ANOVA findings is likely to a cause of the difference between /m/ and /t/ peak-to-peak amplitudes. This is consistent with the CAEP grand average responses in Figure 25, showing greatest difference between /m/ and /t/ in N1 amplitude, while the difference appeared minimal for P2 amplitudes.

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak-to-peak</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>2.02 (14)</td>
<td>0.063</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>1.41 (14)</td>
<td>0.179</td>
</tr>
</tbody>
</table>

Table 15: Paired t-test statistics for N1-P2 peak-to-peak amplitudes for sample n=15 across stimuli
4.1.4 Behavioural Speech Perception and CAEPs

Previous research (Kelly et al., 2005) showed that shorter P2 latencies in response to a 250 Hz tone, were associated with higher speech scores. Therefore the association between the P2 latency in response to the /m/ phoneme (spectral peak = 257.9 Hz) and speech score was tested using linear regression and checking for the influence of outliers on the model. One case which appeared to be an outlier (residual=3.00 standard deviations) was removed, and the subsequent association suggested that the longer the latency, higher the speech score, \( \beta=0.562, p=0.012. \)

![Figure 35](image_url)

**Figure 35:** Correlation between P1 latency for the phoneme /m/ and the composite speech scores generated from HINT in quiet, HINT +10dB SNR, CNC words and phonemes scores, after removing participant 21 who was an outlier (n=23).

Bivariate correlations for all other speech perception scores and the composite speech measure were compared against all measures of N1 and P2 waveforms (latency, amplitude, area 1, area 2 and peak-to-peak amplitude). No significant correlations were found between the waveform measures and composite speech measures (\( p>0.050 \)).

Effects of identification rate across stimuli were first investigated by conducting repeated measures ANOVA. Mauchly’s test of sphericity revealed a significant \( p \)-value (\( p=0.043 \)); thus sphericity did not meet the assumptions of the ANOVA. However, a difference was observed across stimuli, after applying the Huynh-Feldt correction, \( F(2,40)=8.76, p=0.002 \). Pairwise comparisons (Table 16) between /m/ vs. /g/ and /g/ vs. /t/ revealed that percent correct for /m/ (mean 58.6, SD 26.1) and /g/ (mean 65.9, SD 37.1) phonemes were different from the percent correct scored for the /t/ phoneme (mean 89.0, SD 22.5).
### Table 16: Pairwise comparisons between percentages of phonemes scored correctly between stimuli (n=23)

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identification rate</td>
<td>/m/ vs. /g/</td>
<td>-0.75 (20)</td>
</tr>
<tr>
<td></td>
<td>/g/ vs. /t/</td>
<td>-2.96 (20)</td>
</tr>
</tbody>
</table>

Bivariate correlations were conducted between the identification rate and all CAEP measures of N1 and P2. No significant correlations ($p>0.05$) were found in any of the measures of latency, amplitude, area measures and peak-to-peak amplitude for the same phoneme.

### 4.1.5 Summary of significant effects in Part I

- The N1 and P2 peak latencies for the /m/ stimulus was significantly different from /g/ and /t/, but /g/ and /t/ were not different from one another.
- N1 amplitude evoked from /m/ was significantly different from /g/ and /t/. No effect of stimuli on P2 amplitude was observed.
- Area measures were always smaller in area 2 than area 1.
- Area measure 1 of the N1 waveform was different for /m/ vs. /t/ after applying the Huynh-Feldt correction.
- Area 2 measures of N1 waveform for /t/ were significantly different from /g/ and /m/.
- No effect of stimuli was observed in P2 areas for both area measures.
- N1-P2 peak-to-peak amplitudes only showed a stimulus effect between /m/ and /t/ phonemes.
- Longer P2 latency was found to correlate with a higher composite speech perception score for the low frequency stimulus /m/.
4.2 Part II: Children CI users

4.2.1 Cortical Auditory Evoked Potentials

The P1 waveform was present in 42 CAEPs recorded from three phonemes across the 16 children. The six absent responses were either a result of true absence, noise, state of the child or stimulus artefact. Maturational effects were observed across the group; larger amplitudes and broader widths were commonly observed in children aged 0-6 years compared to their older peers. Thus the children were separated into two age groups, 0-6 years (mean 3.3, SD 1.98) and 7-12 years (mean 9.1, SD 1.73). Examples of CAEP response from different participants in the different age groups are shown in Figure 36 below.

![Figure 36: Comparison of CAEP response from a /m/ phoneme in participant 2 aged 6 years, and participant 9 aged 12 years.](image)

**4.2.1.1 Children 0-6 years**

CAEP measures averaged across stimuli for children aged between 0-6 years in the sample are summarised in Table 17 below, showing data for both test sessions. There were 8 child CI users in this category, ranging from 20 months to 6 years of age. Average peak latencies, amplitudes and area 2 measures appear consistent between both test sessions. Area 1 appears to have increased over time between test sessions. Standard deviations and range reveal wide variability in the amplitudes and area measures, which were all reduced in the second test session. Variability of P1 latency in children 0-6 years has remained consistent.
During the first test occasion, P1 was absent in three recordings across two participants (n=21). During the second round of testing, P1 was immeasurable across 8 out of the 24 recordings (n=16). This was due to difficulty recruiting for one participant for retest, and five waveforms unable to be characterised across 3 other participants. Absence of waveforms could be attributed to either noise, artefact or participant non-compliance. All other recordings reliably measured the P1 waveform.

<table>
<thead>
<tr>
<th>Children 0-6 years</th>
<th>P1 peak measures</th>
<th>P1 area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test Session</td>
<td>Latency (ms)</td>
</tr>
<tr>
<td>Mean</td>
<td>1</td>
<td>117.2 (18.7)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>117.6 (22.3)</td>
</tr>
<tr>
<td>Range</td>
<td>1</td>
<td>87.8-160.6</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>85.7-162.7</td>
</tr>
<tr>
<td>95% Confidence Intervals</td>
<td>1</td>
<td>109.2-125.2</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>106.6-128.5</td>
</tr>
</tbody>
</table>

Table 17: P1 latency, amplitude and area measures for children aged 0-6 years (n=8).

Figure 37 below displays the grand average responses for this group of children elicited from the three stimuli. Size of the CAEP appears relatively similar over the two test sessions for individual stimuli. A clear P1 morphology is observed of large amplitude occurring at approximately 100ms, followed by a falling negativity (N2/N250 wave). The stimulus-associated artefacts were visible prior to all CAEP averaged waveforms.

Figure 37: Grand average CAEPs for children 0-6 years (n=8) across two test sessions 3 months apart.
4.2.1.2 Children 7-12 years

CAEPs were also reliably measured in the 8 older children aged 7-12 years from the data set. Table 18 below indicates that P1 peak latencies and amplitudes were consistent across the test sessions over 3 months. By similar proportions, area 1 appeared to have increased while area 2 measures decreased over the time period with CI usage. Variability for this group reduced for all CAEP measures except for area 1. P1 amplitudes and area 1 measures appear to be smaller for children aged 7-12 years than their young peers (see Table 18).

For both test sessions all CAEPs across stimuli were reliably recorded and marked, except for 3 waveforms across two participants. One of the children was missing responses to /g/ and /t/ due to the pedestal artefact (see artefact section later), whilst the other child’s response to /t/ was absent when tested at both sessions.

<table>
<thead>
<tr>
<th>Children 7-12 years</th>
<th>P1 Peak measures</th>
<th>P1 area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test</td>
<td>Latency (ms)</td>
</tr>
<tr>
<td>Mean</td>
<td>1</td>
<td>116.2 (26.9)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>115.5 (19.1)</td>
</tr>
<tr>
<td>Range</td>
<td>1</td>
<td>58.6-198.1</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>89.8-162.7</td>
</tr>
<tr>
<td>95% Confidence Intervals</td>
<td>1</td>
<td>104.7-127.7</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>107.5-123.5</td>
</tr>
</tbody>
</table>

Table 18: P1 latency, amplitude and area measures for children aged 7-12 years (n=8).

Grand averages for children 7-12 years old (n=8) are shown for the two test sessions in Figure 38 below. The stimulus artefact, P1 and N250 waveforms are clearly visible on both test sessions, without much distinction within responses from the same phoneme. P1 appears to reliably occur around 100ms for all stimuli and test sessions. Similar to grand average CAEPs from the younger children, the smallest CAEP was recorded for the stimulus /t/.
4.2.2 Effects of stimulus and age on CAEPs

To compare phonemes and age groups, only data from test session 1 were used because this provided maximum power to detect effects due to lower rates of detectable CAEPs measured at session 2. There was also not a large difference observed between test sessions from the grand average CAEP waveforms. The sample size was maintained at 12 children for all further statistic analyses by only using data from test 1. As an age effect was expected due to maturation, analyses of the stimulus effects on the CAEPs were conducted with consideration of age grouping and chronological age as factors.

4.2.2.1 Stimulus effects

An effect for latency across the stimuli was observed with repeated measures ANOVA, $F(2,20)=5.00, p=0.017$. Paired samples t-test (Table 19) reveal that P1 latency for /t/ (mean 103.4, SD 20.7) was shorter than /g/ (mean 116.7, SD 14.0) and /m/ (mean 119.3, SD 12.5). No effect across stimuli was discovered for amplitude, $F(2,20)=1.44, p=0.260$.

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1 latency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>0.617 (11)</td>
<td>0.550</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>2.45 (11)</td>
<td>0.032</td>
</tr>
</tbody>
</table>

Table 19: Pairwise comparisons for P1 latency between stimuli at session 1

Figure 38: Grand average CAEPs for children aged 7-12 years (n=8) across test sessions 3 months apart.
The same pattern was observed for area 1 measure of P1; an effect was found with repeated measures ANOVA, \( F(2,20)=4.73, p=0.021 \). Pairwise comparisons also revealed that area 1 for /t/ (mean 346.5, SD 178.8) was significantly smaller from /g/ (mean 567.5, SD 238.3) and /m/ (mean 684.2, SD 428.6).

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1 area 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>1.23 (11)</td>
<td>0.244</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>2.55 (11)</td>
<td>0.027</td>
</tr>
</tbody>
</table>

Table 20: Pairwise comparisons for P1 area 1 between stimuli at session 1

Repeated measures ANOVA also found a difference across stimuli for area 2 measures, \( F(2,20)=4.43, p=0.026 \). However, pairwise comparisons between /m/ (mean 417.3, SD 260.6) vs. /g/ (mean 322.9, SD 221.7), and /g/ vs. /t/ (mean 197.4, SD 104.1) produced no differences for P1 area 2 measures. Therefore the significance seen in the ANOVA results is likely due to a difference between /m/ and /t/.

<table>
<thead>
<tr>
<th>Pairwise comparisons</th>
<th>t-statistic (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1 area 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>/m/ vs. /g/</td>
<td>1.51 (11)</td>
<td>0.160</td>
</tr>
<tr>
<td>/g/ vs. /t/</td>
<td>1.74 (11)</td>
<td>0.111</td>
</tr>
</tbody>
</table>

Table 21: Pairwise comparisons for P1 area 2 between stimuli at session 1

Age

Repeated measures ANOVA displayed no effects with the factor of age, grouped by 0-6 years and 7-12 years across the children sample, and nor were there any interaction effects between the two age groups and stimuli on the CAEPs. A second set of repeated measures ANOVA were conducted for the effect of stimuli on CAEPs, with chronological age as the factor. No effects were observed again and thus the age division was abandoned during further analyses.

4.2.3 Effects of CI adaptation on CAEPs over 3 months

The involvement of chronological age and CI age as continuous measures were investigated in CAEP changes over a 3 months while using their CI. From least squares linear regression, chronological age predicted change in P1 amplitude for phonemes /g/ and /t/, but not /m/ (Table 22). Age also predicted change in area 2 for phoneme /t/ but not /g/ nor /m/. Age did not affect change in P1 latency measures in response to any phoneme (all \( p>0.303 \)). There was a marginal effect of age on change in area 1 for the /t/ phoneme, but not for either of the
other phonemes. The pattern of effects observed for area 2 was paralleled in the area 1 data. Time with CI was expressed as the number of months since CI switches-on, termed ‘CI age’. No effects of time with CI were observed for any of the measures (all \( p > 0.514 \)).

<table>
<thead>
<tr>
<th>Stimulus</th>
<th>Latency</th>
<th>Amplitude</th>
<th>Area 1</th>
<th>Area 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>/m/</td>
<td>( \beta = 0.115, \ p = 0.708 )</td>
<td>( \beta = -0.095, \ p = 0.768 )</td>
<td>( \beta = -0.258, \ p = 0.376 )</td>
<td>( \beta = 0.192, \ p = 0.482 )</td>
</tr>
<tr>
<td>/g/</td>
<td>( \beta = 0.297, \ p = 0.303 )</td>
<td>( \beta = -0.763, \ p = 0.006 )</td>
<td>( \beta = -0.337, \ p = 0.219 )</td>
<td>( \beta = 0.126, \ p = 0.680 )</td>
</tr>
<tr>
<td>/t/</td>
<td>( \beta = -0.088, \ p = 0.821 )</td>
<td>( \beta = -0.657, \ p = 0.045 )</td>
<td>( \beta = -0.652, \ p = 0.072 )</td>
<td>( \beta = -0.727, \ p = 0.022 )</td>
</tr>
</tbody>
</table>

Table 22: Least squares linear regression results showing Beta coefficient and \( p \)- values for the measures of P1 amplitude and area 2; where significance was found in some of the phonemes.

Figure 39 depicts a significant negative correlation between phonemes /g/ and /t/ on the change in amplitude between the two test times over three months. This effect is also present for the /t/ stimulus in area 2 measures over the same period (Figure 40). These graphs show the residual values of amplitude and area 2; which was generated as the distance from the line of best fit from the linear regression model. For both P1 amplitude and area 2 measures, the data sample appears to be evenly spread out across ages.

Figure 39: Effect of age on change in amplitude between the two test times. Residual values show the distance of data from the line of best fit in the regression model (n=14 for /m/, n=13 for /g/, n=10 for /t/).
4.2.4 Effects of auditory deprivation on P1 latency

Sharma, Dorman and Spahr (2002) has previously established changes in P1 latencies from birth to adulthood in normal hearing individuals for the stimulus /ba/. Thus with the consideration of stimulus effects on P1 latency reported previously, P1 latencies for all children in part II were averaged across stimuli and graphed with the 95% confidence range of normal hearing data for further comparisons.

Several factors of auditory deprivation were of interest; the age when the child received their implant, period of time since CI switch-on, and the nature of their deafness. Hearing history available for each child was not always consistent, and thus their deafness were characterised by presence of any degree of deafness at birth and if the impairment was progressively changing.

4.2.4.1 Age of implantation

P1 latency is observed to decrease exponentially as age increases, as shown by the black lines representative of 95% confidence intervals P1 latencies from normal hearing individuals. The children were placed into three categories by the chronological age at which they received their CI (Figure 41). For those early implanted children (prior to 3.5 years) from the data set, the variability in age is well spread out up to the age of 12 years (n=9). Children implanted
between 3.6-7 years were aged between 4 and 10 years (n=4), while only 3 children aged between 8-12 were implanted after they were 7. Children implanted early displayed P1 latencies within the 95% confidence intervals of the normative data. P1 latency was observed to be even earlier than the normative range in one child. Those implanted between 3.5-7 years appeared to encroach the edge of the normative data while the late implanted children were all displaying P1 latencies later than normal hearing children.

Adult participants 23 and 24 from part I of the study were additionally incorporated into Figure 41, as they displayed prominent P1 waveforms. Both participants 23 and 24 were implanted later than 7 years of age, and displayed P1 latencies which were much longer than the normative data.

Figure 41: P1 latencies as a function of their chronological age in years, for all children and adult participants 23 and 24. Black lines represent 95% confidence intervals from Sharma et al. (2002). Participants are characterised by their age of implantation: <3.5 years (green), 3.6-6.9 years (yellow), and >7 years (red).
4.2.4.2 CI age

The factor of CI age was also considered, where it represents the amount of time as a CI user since switch-on. Children from the data set had an average CI age of 35.2 months (SD 32.0). The normative lines were expressed in months and overlaid with the CI age data. The early-implanted children remained within confidence of the normative data and displayed a range of experience with their cochlear implant. The children implanted between 3.6-7 years, displayed latencies well within the normal range. Furthermore, the late implanted children had earlier latencies than the expected values for normal hearing, and were observed to cluster around the edge of the lower 95% confidence interval. It was clear that the later implanted children have had less experience with their CI than majority of the middle and all of the early implanted children. Participants 23 and 24 from the adult study were also plotted for their CI age, showing much longer CI experience that the late implanted children.

![Figure 42: P1 latencies of all children and adult participants 23 and 24, plotted as a function of their CI age in months. Black lines represent 95% confidence intervals from Sharma et al. (2002). Categorization by age of implantation remains the same as Figure 41.](image)

4.2.4.3 Deafness characteristics

Four groups were generated by the two characteristics of deafness, and used to classify all child participants in part II. Children in the congenital severe-profound group had a hearing loss of severe to profound degrees at birth (n=9). Those in the group congenital progressive, had some degree of hearing loss at birth which deteriorated progressively until their CI (n=5). There were only 2 children who had an acquired hearing loss: one at a severe-profound level.
was diagnosed with menigitis and received an implant soon after, and another child who was a consistent hearing aid for 5 years prior to receiving an implant.

Figure 43 shows the spread of participants classified by the nature of their hearing loss. A positive correlation can be observed for the children with congenital severe-profound deafness and their CI age; suggestive of increased experience with CI as they age ($R^2=0.95$). Both children with acquired hearing losses have been recently implanted and are both young in CI age. A mixed degree of experience with CI is observed in the congenital progressive group, where some appear to have persisted with hearing aids for a long period and have only been recently implanted, and others have delayed their implantation not much longer than the congenital severe-profound children. For the children in the congenital progressive group, the further distance below the blue line indicates a longer period of auditory deprivation; the extent of which is not considered here.

![Figure 43](image)

**Figure 43:** Relationship between chronological age and CI age between different types of deafness. Line of best fit is shown for the trend in children with congenital severe-profound deafness.

The same classification of children was replotted onto the normative data from Sharma et al. (2002), with P1 as a measure of both maturation and auditory stimulation. All congenital severe-profound children displayed normal P1 latencies across the age range. P1 latencies of the congenital progressive group were clustered around the upper limit of the 95% confidence intervals. The child with acquired severe-profound deafness characteristics
displayed a normal latency while the child with acquired progressive deafness had a longer latency than the normative data.

Figure 44: P1 latency comparison between normative data and CI children (n=16) grouped by deafness characteristics: congenital severe-profound (blue), congenital progressive (red), acquired severe-profound (green), acquired progressive (yellow). Black lines represent 95% confidence intervals from Sharma et al. (2002).

4.2.4.4 Morphology of maturation in CI users

Effects of maturation on CAEPs are tracked for chronological age from the children to adults for each stimulus in Figure 45. Descending down each graph shows the grand average waveforms of the children classified by age groups of 0-6 years, 7-12 years, adult participants 23 and 24, and the rest of the adults. For all stimuli, the general change in CAEP morphology across the groups is similar. Both groups of children display distinct P1 waveforms occurring around 100 ms post stimulus onset, followed by a falling negativity of N250. The same morphology was present for the average CAEP of adult participants 23 and 24, although delayed with P1 peak latency between 150-200 ms. All adult average CAEPs reveal the P1-N1-P2 complex, with P1 at around 50ms, N1 at 100ms and P2 at 200ms/. However, the presence of P1 was also influenced by the preceding stimulus artefact, in particular for /m/ and /t/. For all stimuli, P1 decreases in amplitude, size and latency from the children to adult CAEPs.
Figure 45: Maturational effects across CAEPs for age groups: children 0-6, 7-12 years, P23 and P24, and adults.
4.2.5 Summary of significant effects in Part II

- From visual observation of grand average CAEPs, size of P1 waveforms appeared different between younger and older children. However, no effect of age group (0-6 years and 7-12 years) or chronological age were found as a factor between effects of phoneme on CAEPs

- The /t/-evoked CAEP displayed shorter P1 peak latencies than for /m/ and /g/

- P1 area 1 measures were also smaller for /t/ than the other stimuli

- The P1 area 2 measures were only smaller for /t/ compared /m/

- The change in size of amplitude and area 2 with use of CI over three months, were larger for the phoneme /t/ in younger children. The same effect is also seen in the change of P1 amplitude for /g/. However, no effects were observed when compared across CI age.
4.3 Stimulus artefact in CAEPs of CI users

4.3.1 Categorisation and characteristics

The absence or presence of stimulus-associated artefact was observed across all adults and children. Artefact data was combined between both sessions of testing in the children as there was a high test-retest reliability of artefact presence; difference between tests were only seen in child 11 and 13. Figure 46 also compares the artefact morphologies across both groups. The incidence of the blip artefact is evidently the highest among all ages; with more observed in the children than adults. The children had more CAEPs recorded without noise and contamination (11.8%), while 22% of CAEPs recorded for the adults had no artefact or noise. The pedestal artefact appeared to be present in approximately 4-6% in all CI users of this study. As such a high proportion of CAEPs display the presence of artefact, the blip and pedestal artefacts are characterised further.

![Pie charts comparing proportions of artefact morphology in CAEPs between adults and children.](image-url)
<table>
<thead>
<tr>
<th>Adults</th>
<th>Type of Artefact</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blip</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
</tr>
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<td>3</td>
<td>3</td>
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<td>4</td>
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<td>23</td>
<td>3</td>
</tr>
<tr>
<td>24</td>
<td></td>
</tr>
</tbody>
</table>

| N      | 50   | 5      | 16   | 1         |
| Frequency | 69.4% | 6.9%  | 22.2% | 1.4%     |

Table 23: Absence and presence of stimulus artefact in adult participants (n=24)
<table>
<thead>
<tr>
<th>Children</th>
<th>Type of Artefact</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>session</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
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<tr>
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</tr>
<tr>
<td>n</td>
<td></td>
</tr>
<tr>
<td>Frequency</td>
<td></td>
</tr>
</tbody>
</table>

Table 24: Absence and presence of stimulus artefact in child participants (n=16)
4.3.1.1 Blip Artefact

Table 25 shows the mean measures of the blip artefact in the adults, categorized from a sample size of 50 blip artefacts observed from 72 adult CAEPs. The mean blip artefact measures are described for each test session from 74 blip artefacts in 93 CAEP recordings in the children; as one child was unavailable to be retested for the second session (Table 26). A blip artefact was sometimes observed in the absence of the CAEP. In such cases peak and offset measures were still reported where possible. The children’s measures were highly consistent between the two test sessions over 3 months, and has a much smaller standard deviation across all measures, compared to the adults. Both adults and children displayed a blip artefact which peaked much smaller and earlier than the P1 waveform amplitude in children. On average all peak and offset measures were earlier in latency in children than adults. The upper 95% confidence interval for the offset of the blip artefact was 70.9 ms post-stimulus onset for the adults and 58.5 ms for the children.

<table>
<thead>
<tr>
<th>Adults</th>
<th>Peak latency</th>
<th>Peak amplitude</th>
<th>offset latency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>24.8 (12.4)</td>
<td>1.0 (1.2)</td>
<td>63.0 (28.5)</td>
</tr>
<tr>
<td>Range</td>
<td>4.5-66.9</td>
<td>-3.0-3.8</td>
<td>19.1-141.9</td>
</tr>
<tr>
<td>Upper 95% confidence interval</td>
<td>28.2</td>
<td>1.4</td>
<td>70.9</td>
</tr>
</tbody>
</table>

Table 25: Summary of blip artefact peak latency, peak amplitude, and the offset latency for all adults (n=50)

<table>
<thead>
<tr>
<th>Children</th>
<th>Blip Peak</th>
<th>Blip offset</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test session</td>
<td>Latency (ms)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>1</td>
<td>20.1 (5.8)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20.3 (4.9)</td>
</tr>
<tr>
<td>Range</td>
<td>1</td>
<td>6.6-35.7</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>14.9-33.6</td>
</tr>
<tr>
<td>Upper 95% confidence interval</td>
<td>1</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>21.7</td>
</tr>
</tbody>
</table>

Table 26: Blip artefact measures of peak latency, peak amplitude and offset latency in the children (n=74)

Figure 47 below shows that adult blip artefact offsets had similar mean latencies across stimuli. The widest variability in the offset of the artefact was observed for the /t/ stimulus, while the spread of offset latencies were the similar for the other phonemes. Thus the mean for all blip artefacts was used to plot along the grand average adult CAEP for all stimuli (Figure 48).
4.3.1.2 Pedestal Artefact

The pedestal artefact was observed in 4 adults and 1 child participant, who were all grouped together for characterisation of this artefact type (Table 27). The artefact data for session 2 for the child was similar and thus excluded as data from only one test time were available for
the adults. All responses affected by the pedestal artefact varied in degrees of contamination; as parts of P1, N1, or P2 were sometimes observed to be partially present. Nonetheless it was too difficult to visually identify peaks for waveform analysis. All pedestal artefacts were preceded by a small blip prior to the falling negativity of the pedestal; these were not reported with the blip data. The average peak latency occurs at 56.2 ms, much later than the peak latency of the blip artefact. The amplitude is also much larger by several magnitudes than the blip artefact or any waveforms from adult and child CAEPs. Figure 49 below shows the grand average response from recordings of the pedestal artefact. It is clear that although variations in the amplitudes exist, the pedestal morphology remains the same. However, even the recordings with the smaller amplitudes would be enough to eliminate the visibility of CAEP landmarks. Seven CAEPs with pedestal morphology were analysed in Table 27; of which four were recorded for the /t/ stimuli, 2 for /g/ and 1 for /m/.

<table>
<thead>
<tr>
<th>Adults (P19-22)</th>
<th>Peak latency</th>
<th>Blip to Pedestal Peak amplitude</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>56.2 (16.9)</td>
<td>30.8 (9.5)</td>
</tr>
<tr>
<td>Maximum</td>
<td>37.8-87.8</td>
<td>20.6-49.5</td>
</tr>
<tr>
<td>95% Confidence Intervals</td>
<td>43.7-68.8</td>
<td>23.7-37.8</td>
</tr>
</tbody>
</table>

Table 27: Summary of peak latency and amplitude form pedestal artefacts recorded (n=7)

Figure 49: Grand average response from all the recordings contaminated with pedestal artefact (thick black line). The grey lines shows the variation seen across the adults and child in this sample (n=7).
4.3.2 Artefact minimization techniques

In part I of the study, two methods of artefact reduction techniques were investigated by the Bio-logic and HEARLab evoked potential systems. In adults, the blip artefact was observed to precede the P1-N1-P2 complex and its offset was often a contaminant in the establishing presence of the P1 waveform. Figure 50 below shows the presence and therefore absence of the P1-N1-P2 complex in the adults who were tested by both systems. P1 was absent on more occasions than N1 and P2 waveforms for both systems across stimuli. CAEPs from the Bio-logic system had present N1 and P2 waveforms by similar proportions, while P2 were less frequently present in the HEARLab system compared to N1. Comparing between the systems, CAEPs from the HEARLab consisted of less P1 and P2 but N1 presence was similar to the Bio-logic.

Figure 50: Comparison of CAEP presence between the two evoked potential systems across stimuli for all adults (n=24)

Figure 51 shows two examples of the subtraction method in participants 10 and 5. For both CI users, the blip artefact in the recording is visibly replicable with both slow and fast rate presentations. With the fast presentation rate, the CAEP recorded for participant 10 showed smaller and later N1 response, while P2 response appears only smaller in amplitude compared to the slow condition CAEP. The subtraction appears to have minimised the blip artefact to the noise of the baseline in the difference waveform (in black on the right) of
participant 10, but only the N1 waveform is distinguishable, and marking of P2 peak would prove to be difficult amongst the noise.

In participant 5, although they only the P2 waveform was distinguishable the CAEPs from both rate conditions appear to be so similar that they almost overlap. No CAEP waveform was consequently observed in their difference waveform post-subtraction.

Figure 5: CAEPs from fast and slow conditions (left) and the subsequent difference waveform (right) in adult participants 10 and 5.
4.3.3 Summary of findings

- The blip artefact was the most common finding in a CAEP recording for the CI users, occurring in 69% of adults and 80% of children.
- Blip peak and offset latencies were later in adults than children; the upper 95% confidence interval of the offset latency lies at 70.9 ms in adults and 58.5 ms for children.
- The pedestal artefact was recordable in adults and children, with blip peak-to-pedestal peak amplitude larger than CAEP responses by several magnitudes. The pedestal was also more common with the /t/ stimuli.
- In the adult study, the two evoked potentials system using different artefact-minimisation techniques had different effects of producing waveforms of the P1-N1-P2 complex. P1 was absent more often than N1 and P2, and there were more P1 waveforms visually identified with the Bio-logic system.
- A variety of responses were observed using the fast and slow rate conditions. These differed in latency, amplitude and the fast rate condition did not always produce a smaller CAEP response. Thus very different effects were consequently observed in the difference waveforms.
CHAPTER 5 DISCUSSION

The presence of the newborn hearing screening programme in New Zealand is expected to see rising incidences of hearing aid fittings and cochlear implants in prelingual infants. Obligatory CAEPs may provide an objective measure which can reveal that novel stimulation from an infant’s cochlear implant has been adequate for the neural detection, and possibly processing of speech sounds, important for speech and language acquisition. Furthermore, CAEPs may assist with behavioural measures such as CI mapping in difficult-to-test populations. Thus there is much scope for research into this potential clinical tool.

The discussion will follow the aims of the current study in investigating the use of objective CAEPs and its association with neural detection and processing of speech sounds. The current study aimed to explore the differences in the neural responses evoked in CI users from three speech phonemes, representative of the low, mid and high frequencies in the speech spectrum. Based on previous evidence of an association between P2 peak latency and CI performance (Kelly, et al., 2005), it was important to investigate correlations between speech perception and CAEP measures. CAEPs potentially play a role in predicting CI performance for those unable to perform behavioural speech tests. The investigation was extended to child CI users: CAEPs were used to measure the effects of their auditory adaptation to the CI over a short time period. Lastly, the study also aimed to address a current problem with artefact contamination that occurs when recording CAEPs in CI users. The artefacts were characterised and strategies were explored for minimising artefact contamination of CAEP recordings.
5.1 Part I: Adults

The adult study was undertaken to investigate stimulus effects on cortical neural responses, and CAEP correlations with behavioural speech perception. If such correlations exist they would be an invaluable asset to CI assessments clinically, as speech perception tests are currently a main measure of CI performance in the adult programme and behavioural testing may not be possible for some adult CI users. Adult CI users provide a diverse insight into both the mature auditory system, and the effects of long periods of auditory deprivation.

5.1.1 Stimulus effects and CAEPs

Stimulus effects on CAEPs evoked by speech stimuli are expected based on previous studies of normal hearing adults. Agung et al. (2006) established a distinction between CAEPs evoked by the Ling sounds which are most spectrally diverse, but not between the sounds which were very similar. The speech phonemes /m/, /g/ and /t/ have also been explored in the normal hearing population (Beukes, et al., 2009; Golding, et al., 2007; Munro & Purdy, 2011). These three phonemes are temporally and spectrally distinct and span the speech frequency range. Thus these phonemes were employed in the current investigation with CI users, and all relationships between speech perception and CAEP measures were explored.

5.1.1.1 Latency and amplitude

For both N1 and P2 peaks, latency was found to be the most sensitive measure of differences across stimuli. The low frequency /m/ stimulus elicited longer N1 and P2 peaks than both /g/ and /t/. Several theories are in support of this finding from studies of frequency effects on latency and amplitude, using tones. Jacobson et al. (1992) reported that in individuals with normal hearing, increased latencies were observed for the low frequency tone 250 Hz, compared to 1000 and 4000Hz stimuli (Jacobson, et al., 1992). The differences in latency between high and low frequencies have been reported to be approximately 10 ms (Roberts & Poeppel, 1996), consistent with the 11-14 ms longer latencies observed in the current study for /m/ in both N1 and P2 waveforms. Longer latencies are thought to be a result of delay in signalling from the broad areas of cortical activation correlated with neurons sensitive to low frequency stimuli (Jacobson, et al., 1992).
The same pattern of a larger peak amplitude for /m/, distinct from responses for /g/ and /t/ was also observed. It has been proposed that the larger amplitude CAEPs associated with low frequency stimuli is the result of deeper generators of low frequency-specific neurons in the cortices; thus the evoked potential is required to overcome a greater distance and impedances from surrounding structures to be recorded at the scalp. At the peripheral end of the auditory system, it has been suggested that suprathreshold activation of high frequency regions also occurs with low frequency stimuli due to the mechanics of the travelling wave (Antinoro, et al., 1969). The broad activation pattern at the periphery may be maintained in the ascending pathways; and it may therefore take longer to synchronously reach activation of deeper P2 generators. However, opposing findings have also been reported with longer latencies elicited by higher frequency stimuli (Zerlin & Naunton, 1974). It is difficult to gauge the differences between studies due to use of different stimuli. Numerous generators for N1 and P2 have been identified, widespread within the auditory and associative cortices. The tonotopical mapping from periphery to central auditory systems and their subsequent conduction velocities are all important when considering frequency effects on latency and amplitude. It is also of interest to note that peripheral mechanisms occur quite differently with electrical stimulation from a CI. One must consider the influence of frequency sensitive neurons and the integrity of the auditory nerve. Furthermore, individuals will vary in the depth of electrode array insertions and the proximity of electrode placements to surviving nerve fibres.

Jacobson and colleagues (1992) suspected that combinations of both peripheral and central mechanisms are responsible for the longer N1 latency and larger amplitude observed with the 250 Hz tone. This is consistent with the larger and longer N1 peak observed for the phoneme /m/. The broad activation and summation from peripheral to central auditory systems may have generated the larger N1 response for the low frequency stimulus in the current study. However, contradictory findings have also been reported in the literature, where the same effect on amplitude was observed at lower input intensities (Antinoro, et al., 1969); which discredits peripheral recruitment of high frequency neurons. It is interesting to note that frequency effects on both latency and amplitude were not significant when results for the higher frequency phonemes /g/ and /t/ are compared. These two phonemes overlap spectrally, but /t/ has a higher frequency spectral peak than /g/. Pairwise comparisons showed a marginal difference ($p=0.051$) between P2 amplitudes for the phonemes /g/ and /t/. This warrants further investigation with a larger sample size to determine if there is a true difference
between these two stimuli or it is simply a Type I error. Antinoro et al. (1969) reported that, for their 2000 Hz stimulus, the amplitude effect was lost above 2000 Hz for high intensity levels; which may explain the current findings as 2000 Hz is situated in between the overlapping frequency regions of /g/ and /t/.

The three natural phonemes investigated here convey speech characteristics which vary temporally and spectrally. Evidence of duration-sensitive neurons in the auditory system has been established based on CAEPs evoked by speech sounds of different duration (Beukes, et al., 2009; Munro & Purdy, 2011). Longer stimulus duration was associated with reduced CAEP amplitudes. Relatively brief stimulus durations were used in the current study. The /g/ phoneme used for the current investigation was 19 ms, shorter than the 27 ms of the /m/ and /t/, but CAEPs evoked by /g/ were not larger in amplitude. However, this is a minor difference in duration compared to previous studies and thus is unlikely to trigger activation of different duration-sensitive neurons that might cause differences in the CAEPs.

Spectral differences between the three phonemes may play a larger role in determining the CAEP measures than the minor differences in stimulus duration. Several studies on normal hearing adults have compared the difference between two short speech phonemes, spectrally based in low and high frequencies (Beukes, et al., 2009; Munro & Purdy, 2011). In these studies latency and amplitude variations across stimuli for N1 are similar to those seen in the current study. Both studies reported that the low frequency /m/ produced longer and larger CAEPs than the high frequency speech stimuli. Beukes et al. (2009) used the /sh/ phoneme which is spectrally comparable to the /t/ phoneme used in the current investigation, with its spectral energy centred around 3575 Hz. Munro and Purdy used /m/ and /t/ phonemes slightly longer in duration than the /m/ and /t/ used in the current study, of 78 ms and 79 ms respectively. Beukes et al. (2009) and Munro and Purdy (2011) also found that a longer latency and smaller amplitude was observed consistently for the P2 waveform for /m/. They suggested that complex neural interactions between temporal and spectral mechanisms of processing in the auditory system may also be in effect; in which spectral characteristics may be degraded in place of temporal information. These findings along with the mixed effects seen across studies with tonal stimuli indicate that P2 amplitude is subject to a wide variety of influences. This may be an explanation for the lack of stimulus effects observed on P2.
amplitudes in adult CI users in this current investigation. The longer P2 latencies for /m/ may also be a downstream effect of longer N1 latencies time-locked to the stimulus.

5.1.1.2 Area measures

Latency and amplitude measures have been widely be reported in CAEP literature. Area measures however, have mostly been utilised in the identification of MMN. Previous MLR research has supported the usefulness of area measures over traditional latency and amplitude measures for MMN (McGee, et al., 1988). The area under the waveform is considered to represent a summary of the amplitude and latency width. Thus it considers the summation of firing from neuronal populations involved for the period of activation, and overall neural synchrony.

In part II of the study, a similar pattern of stimulus effects were observed for both area measures for the N1 waveform. Mean area 1 of the N1 waveform was significantly smaller for /t/ and largest for /m/. A similar pattern was found for area 2 estimates of N1, except CAEPs for /t/ was smaller than those for both /g/ and /m/. The area measures were generally expected to correlate well with amplitude findings but not with latency, as area is expected to represent the summation of activation, over the period of the waveform. Such a relationship was observed across stimuli with amplitude and area measures. Thus if a continuum was assumed across the stimuli based on frequency, it would appear that the increasing frequency of the speech stimulus saw a decrease in the size of the waveform, based on amplitude and area measures. The differences between /g/ vs. /t/ were not observed for amplitude, and between /m/ vs. /g/ were not observed for area measures. The poor distinction between these pairs, may be spurious or due to a small sample size of diverse adult CI users.

Neither area measure was sufficiently sensitive to find a stimulus effect for P2. This is consistent with the other measures as an effect was only observed for P2 latency but not amplitude; providing support for the relationship between area measures and amplitude. As P2 generators are involved in attention, sleep state, and many other sensory implications (B. Martin, et al., 2007), it is possible that any stimulus effects were either absent or overshadowed by other sources of neuronal activation.

The /m/ phoneme is both temporally and spectrally more distinctive than the /g/ and /t/ phonemes, as seen in their waveforms in Figure 15 of the Methods. This is consistent with the
neural differentiation between /m/ and the other phonemes revealed by N1 latency and amplitude measures. However, area measures showed little distinction between /m/ and /g/, which may be due to the different sensitivity of area and amplitude measures in evoked potentials. The CI encoding of spectral and temporal characteristics may have also have an effect on the resolution of each type of measure across stimuli.

The two area methods were designed to investigate if inter-wave interactions affected the amplitude and should therefore be considered when comparing CAEPs from different stimuli. Area 1 is based on a the traditional method used commonly in evoked potentials software using the extended trapezoid rule (Neuroscan, 2003), and takes into account the amplitude of the neighbouring peak. Area 2 was designed to avoid influences from neighbouring waves, as N1 and P2 have been established with distinctively different neural generators. In the current study, area 1 was consistently larger than area 2, as clearly depicted in the grand average CAEP in Figure 21. Area 2 measures exposed more differences across stimuli compared to area 1. Nonetheless it is still difficult to ascertain if area 2 is actually a more sensitive measure of stimuli effects on the N1 waveform, or if area 1 measures are affected by inter-wave interactions with P2 amplitude.

5.1.1.3 N1-P2 peak-to-peak amplitude
The measure of peak-to-peak amplitude between N1 and P2 waveforms was considered to investigate the strength of the findings for amplitude and the two area measures. Peak-to-peak amplitudes have been frequently used to investigate inter-wave interactions. In the adult group, the largest peak-to-peak amplitude was observed for the low frequency /m/, which was significantly different from /t/. This is consistent with the results for N1 absolute amplitudes, in support of the finding that the size of the N1 waveform is on average smaller for /t/, and largest for /m/.

Many have argued that the strength of excitation in various neural populations is too difficult to separate with a peak-to-peak amplitude measure. Ponton and Eggermont (2001) suggested that in adjacent waves of alternating polarity, such as in the case of the P1-N1-P2 complex, phase cancellation can occur when the responses temporally overlap (Ponton & Eggermont, 2001). The representation of this interaction may be reflected in the far-field scalp recording as a paired increase in N1 and decrease in P2 amplitude or vice versa. Such temporal
interactions should be considered and are likely to influence the differences observed between the methods for area estimation.

The peak-to-peak amplitude findings cannot confirm if either area 1 or 2 is a better measure. But they provide confidence that all the CAEP measures investigated in the current study have produced similar findings. It may be fair to say that these measures are all clinically useful for CAEPs in adult CI users. In such cases, the traditional use of latency and amplitude may still be the easiest to implement, as area measures and peak-to-peak amplitude require post-acquisition processing. Nonetheless, changes in individual alertness, attention and participant variables as well as noise from recordings may infer changes which appear significant when assessing the latency and amplitude across stimuli. Thus it would be interesting for future investigations to question whether area measures are more robust in their test-retest reliability.

5.1.2 Behavioural speech perception and CAEPs

Overall, the adult CI users found the HINT in quiet test the easiest, followed by CNC phonemes, HINT at 10 dB SNR, and CNC words were the most difficult. The HINT in quiet tests have been previously reported to have the highest scores in speech test batteries implemented in studies (Firszt, Chambers, & Kraus, 2002; Kelly, et al., 2005), to the extent that these scores are likely to reflect a ceiling effect (Gifford, et al., 2008). As observed in the distribution of HINT in quiet scores across the adults in the current study, the lower quartile was above an 80% score. The clustering of data for this particular test was accounted for by calculating Z-scores for each speech test and generating an overall composite speech score. The high Cronbach’s alpha for the final composite speech score also gave security and confidence in the combination of the tests and supports the reliability of the correlations.

Kelly et al. (2005) identified that better CNC phoneme scores were associated with shorter P2 latencies for a 250 Hz tone stimulus. This hypothesised link between low-frequency CAEPs and speech perception was investigated specifically with the low frequency /m/ stimulus (spectral peak= 257.9 Hz), within the adult dataset from the current study. This was identified as the only significant correlation amongst all CAEP measures inclusive of latency, absolute and peak-to-peak amplitudes, and area measures for N1 and P2 waveforms. Contrary to findings from Kelly and colleagues, the correlation was positive and suggested that those
with better overall speech perception, had longer P2 latencies. These findings may simply be spurious due to the numerous variables analysed across phonemes, and CAEP measures. Another thought on the difference may be that our speech stimuli, although short in duration, encompassed speech characteristics which were specific to more cortical association areas, important for speech processing. The broader areas of activation for P2 generation, may have resulted in an overall delay in the latency. However, this does not explain why the effect was only present for the /m/ stimuli.

One could also postulate that longer P2 peak latency may be a result of a growth in P2 area and latency width. Such an event may be the result of longer CI experience, and thus improved speech performance. Thus morphology was individually investigated and an example comparison between participants 7 and 14 was found. Figure 52 illustrates that this difference in area morphology can exist between some individuals with very different speech scores. Although P2 area measures were not correlated with any composite or individual speech perception test scores, it could be possible that statistical significance was not achieved due to the variable nature of the P2 waveform; as observed with the large distribution of P2 compared to N1 area values in the current dataset. P2 generators are also known to be influenced by the factors such as attention and sleep (B. Martin, et al., 2007), which unfortunately reduces its usefulness in understanding the interactions between speech performance improvements with CI experience.

![Figure 52: Comparison of CAEPs for stimulus /m/ from participant 7 and 14. P2 area and latency is greater in participant 14 than 7, while N1 waveforms remain similar. Participant 14 has a greater speech scores overall than S7 (85% vs. 33% for CNC phonemes). Participant 14 has also had longer experience with CI (25 months) than participant 7 (13 months).](image)

The differences may also be attributed to a wider distribution of speech scores from the 12 adult CI users in Kelly et al. (2005). However, conducting the correlation using the Z scores...
and a composite speech score across the dataset in the current study should have accounted for the clustered range of HINT in quiet scores. Furthermore, Kelly et al. (2005) also reported strongly negative correlations between better speech perception with shorter duration of profound deafness. Thus the correlation between CNC phoneme perception and P2 latency cannot be made without the consideration that longer duration of deafness, and thus deprivation, may have contributed to the effect on P2 latency as well.

The current literature comparing behavioural speech perception and CAEP measures in CI users has revealed mixed findings. Firszt et al. (2002) did not observe any significant correlations between electrically-stimulated CAEPs and a large battery of open and closed-set word and sentence tests, inclusive of tests conducted in noise. Although the tests and stimuli parameters varied, several other studies also have not found an effect on behavioural speech measures with change in processor rate (Friesen, et al., 2005), and increased number of active CI channels (Friesen et al., 2009). Makhdoum et al. (1998) found a negative correlation between spondee identification and eALR P2 latency. However, the distribution of data from this study may have also encountered a ceiling effect; as the majority of participants scored higher than 60% in spondee identification (Makhdoum, et al., 1998). Maurer et al. (2002) also found a negative correlation with eALR P2 latency based on groups with better and worse speech performance. The speech tests were less reliable as they were non-standardised sentences delivered by live voice. The importance of the relationship between speech perception improvement and growth of the CAEP waveforms has been traced longitudinally in two CI users post-implantation (Pantev, et al., 2006). Rapid initial improvements in speech perception appeared to relate in time to the growth of P1 and N1 from magnetic dipole source analysis in these two individuals. Current literature would benefit from future investigations of longitudinal effects with the changes in P2 waveform and speech perception compared for CI users post-implantation. These effects are highly relevant clinically, as abnormal CAEP morphologies in children with CI have been associated with poorer speech perception (K Gordon, et al., 2005).

The identification rate for the /m/, /g/ and /t/ phonemes was deduced from the CNC phoneme scores to explore a finer resolution of individual phonemic discrimination abilities. Overall, the adult CI users were able to discriminate the /t/ phoneme from CNC words lists with the highest accuracy, while the discrimination were significantly poorer for the mid and low
frequency phonemes. This was an unexpected result, as CI users often have proportionally longer periods of high frequency deprivation prior to implantation. The identification rate also did not correlate with CAEP measures considered for the specific phoneme. This result is consistent with the null hypothesis finding for the majority of the comparisons of speech scores with CAEP measures. However, it should be noted that the amount of /m/, /g/ and /t/ data available in an individual CNC word list was very variable, and some percentage correct scores were generated out of only one or two phonemes present in that list. Although the lists were randomised, the /t/ phoneme did appear more frequently than the other stimuli. It should also be noted that scoring for /t/ included two versions: with and without aspiration, for example, the word ‘tent’. The /t/ stimulus used for the CAEPs is a voiceless plosive and it may not be fairly represented by the two versions of ‘t’. Future studies comparing CAEPs with speech scores would benefit from newly devised speech tests that more carefully assess perception of the CAEP phonemes. However, as suggested by Tremblay (2007), small scale phonemic discrimination may not translate to overall sentence and verbal language perception abilities.

5.1.3 Auditory deprivation in adult CI users

Participants 23 and 24 were separated from the adult data set in CAEP analyses as they displayed morphologies similar and more comparable to the P1 waveform in children. Similar case examples in CI users have been reported in an early study (Ponton, et al., 1999), which discussed possible reasons for the suspended maturation of P1 seen in older children through to late adolescence. Ponton et al. (1999) suggested that cortical responses are sensitive to the progression of a layer-by-layer maturation in the cortices. Hence in congenitally deafened CI users, the duration of deprivation may reflect poor formation of neural generators responsible for the portrayal of the adult P1-N1-P2 complex. The authors also suggested that some form of rapid cortical activation post CI switch-on is likely to occur. However participants 23 and 24 were not continuously followed post-switch and thus it is difficult to ascertain if their current responses reflect changes that have occurred after implantation. Furthermore, both scored highly with speech perception tests; above 80% for HINT sentences in quiet and in noise, and above 70% for all CNC scores. In this respect, their CI performance does not appear grossly ‘delayed’ as suggested by their unique CAEP morphology. Furthermore, they were not disadvantaged by speech tests in noise, scoring above the average HINT in +10dB SNR score in the group (61.2%). Therefore, their CAEPs
were further compared with the child data set, in attempt to explore effects of deprivation on late-implanted adult CI users.
5.2 Part II: Children

The children in part II offered an opportunity to investigate the effects of stimuli previously established for the adults, for the immature but highly plastic auditory system of children. Thus effects of stimuli on the child CI users were examined with the consideration of their age and CI experience. The change in CAEPs over a 3 month period was determined, and P1 latency was also utilised to explore the effects of deprivation and type of deafness.

5.2.1 Stimulus effects on CAEPs

5.2.1.1 Latency and amplitude

Consistent with the pattern from adult CI users, shorter P1 peak latencies were observed for the high frequency /t/ stimulus compared to the mid and low frequency stimuli in the children. These results further support the temporal pattern of CAEPs elicited by the three phonemes for all participants: CAEPs waveforms for /m/ possess longer average peak latency than /t/; with the peak latency for /g/ in between. This temporal pattern can thus be explained similarly to the adults, that peripheral and central auditory physiological structures should be considered when varying spectral characteristics are encoded. These results are consistent with recent findings in normal hearing infants aged 3-7 months (Purdy, et al., 2011) where a later latency was seen for the phoneme /m/, although /m/ in this study had a much longer duration of 141 ms. Golding et al. (2006) compared /m/ and /t/ of either fixed stimulus duration or fixed ISI in the same age group, and observed that the low frequency /m/ maintained its longer latency consistently.

No stimulus effects were observed for P1 amplitude. This is not consistent with the studies mentioned previously which showed larger amplitude for /m/ than /t/ in children with normal hearing (Golding, et al., 2006; Purdy, et al., 2011). Differences in age of participants in the current investigation compared to these recent studies, and the presence of hearing loss and a cochlear implant in the current study may account for this difference. Infant CAEPs are known to undergo impressive maturational changes within the first 12 months (Kushnerenko, et al., 2002; Pasman, et al., 1999). This rapid maturation period that occurs in typically developing infants with normal hearing is not included in the current study as the youngest participant was 20 months old. As the P1 waveform is prone to effects of neuromaturation
and auditory deprivation (Ponton, et al., 2000), it is likely the school-aged children in the current data set will have an impact on P1 amplitude results. As reported by Ponton et al. (2000, 2002) P1 reduces in amplitude as N1 and P2 appear in the CAEP waveform recorded at the vertex and similar electrode locations. The current data set is relatively divided across ages between 20 months to 12 years, and an attenuation of the large P1 amplitude seen in younger children is expected in the older participants due to neuromaturation. Thus age was factored into the analyses and affected P1 as expected, but no further stimulus effects on P1 amplitude were found. These results may indicate that the stimulus effects on amplitude may only be present in normal hearing infants aged 3-7 months, and are out grown for P1 by around 2 years of age, or other factors account for this finding.

### 5.2.1.2 Area measures

The same pattern of stimulus effects were also found for both area measures: the high frequency /t/ stimuli evoked smaller P1 waveforms than /m/. P1 area 1 for /g/ was also significantly larger than the P1 from /t/. Interestingly, a significant difference between /g/ and /t/ was also seen for area 2 for the adults. This suggests that the two area measures may be equally sensitive to waveform changes, or the effects seen for /g/ may be a spurious finding due to Type I error. Further studies with a larger sample size are warranted to determine the usefulness of CAEP area measures. In adults there may be inter-wave interactions between N1 and P2 due to their temporally overlapping auditory generators, compared to the immature generators of P1 and N250. This could account for differences in stimulus effects on area measures in adults versus children.

The overall stimulus effects observed for the main CAEP measures for both adults and children have been highly consistent. The P1 waveform in children under 12 years and the N1 waveform in adults can both show different responses for the /m/ and /t/ phonemes. The temporal contrasts between this pairing of phonemes should be noted. The /m/ phoneme is a nasal, voiced consonant which is temporally slow-rising across the duration of the phoneme. In contrast, the temporal concentration of energy is increased rapidly towards the end of the stimulus duration for the voiceless stop consonant /t/. The effect of the stop consonant should also be considered, as previous evidence have suggested shorter CAEP latencies for them (Gage, et al., 1998). Phillips and colleagues (2002) have previously proposed that central auditory system is well-tuned to the properties of the stimulus onset, and for the case of tones,
its onset ramp. However, Purdy et al. (2011) has suggested that rapid onset of stimulus does not affect reliability of CAEP generation, in the context of speech stimuli. The effects of VOT is probably not applicable to data from the current investigations as the phoneme durations were all below 30 ms. Overall, both spectral and temporal contrasts between the pair of phonemes, have a complex role in the pattern of stimulus effects observed across part I and II study.

5.2.1.3 Effects of CI adaptation over 3 months

The age of the child was expected to factor prominently in the investigations of stimuli effects in the children. However, age by groups of 0-6 years and 7-12 years, and as a continuous measure in chronological years, did not play a role in differences between CAEPs across stimuli. Nonetheless, it was still considered as a factor when exploring effects of changes in CAEP over 3 months. This part of the study was designed with the hypothesis that a larger change in CAEPs would be observed over 3 months in those more recently implanted, that is participants with a younger CI age. Sharma et al. (2002a) has shown that the auditory pathways undergo rapid cortical development during the first 6-8 months after age-appropriate implantation in children with congenital deafness. The current data set has a wide distribution of CI experience and chronological age and hence it would have been more difficult to demonstrate such changes. Thus these factors were both considered when comparing the effects of using a CI over 3 months on CAEPs evoked by the three stimuli.

Stimulus effects were observed for the change in CAEP between the two test sessions. Significantly larger P1 amplitudes were found in younger children after three months use with their cochlear implant. The extent of these effects was strongest for the high frequency phoneme /t/, and then /g/, with no significant change in amplitude over time for /m/. P1 area 2 measure was also larger after the 3 months, although the effect was only seen for /t/ and not /g/ or /m/. One explanation may be that there is less change observed in the older children as they have had their CI for longer and thus neural structures are well adapted the stimulation which is no longer a novel experience. This would be consistent with the growing pool of evidence for a sensitive period for optimal auditory plasticity early on in life; where larger effects are seen with CI stimulation in younger children (Sharma, Dorman, et al., 2002a).
The stimulus-specific effects on CAEP change over the three month test period may be attributed to the nature of deafness in most cochlear implantees, that is high frequencies are often the least adequately stimulated prior to implant. These stimulus effects may be explained by the frequency mapping in the cortical layers discussed previously. Neural generators for the higher frequency /t/ originate in deeper cortical layers than the superficial low frequency-specific neurons (Jacobson, et al., 1992). Neuromaturation is known to progress from deep to superficial layers in both the cat and human. Immunostaining evidence indicates that the density of intra- and inter-cortical connections becomes settled and similar to adults by 11-12 years of age (Ponton, et al., 1999). It is possible that the development of these superficial layers was not sufficient to produce a change in CAEPs between the two test times. It is also possible that rapid cortical changes occurred after or prior to the three month period sampled. It is also known that synaptogenesis is fairly active over the first four years of life (Huttenlocher & Dabholkar, 1997), and hence the older children tested may be outside this period of substantial plasticity. It is also expected that complex cortical feedback patterns from higher-order processes are less evident or poorly formed in the younger children of the data set. It is possible that the detection of speech characteristics of the stimuli post-implantation may activate rapid compensation for the previously de-coupled cortical connections between primary and associative areas. This effect was not observed for the stimulus /m/, which may be masked by the effects of the variance in CI experience across the data set.

Surprisingly, when CI age was considered as a factor instead of chronological age, the change in CAEPs over time is eliminated. CI age is often a measure of ‘hearing age’ based on the idea that it is the commencement of auditory stimulation. However, this consideration was not accurate for the participants in the study, as a measure of the onset of activation of the auditory system and stimulation. Many of the older children had long histories of hearing aid usage in the programme. It is also a facet of eligibility for a CI funded through the NCIP (Northern Cochlear Implant Programme), that candidates must have trialled hearing aids to an ineffective point, pre-implantation. The current study therefore considers the CI age to be a measure of time since the onset of the most optimal stimulation available to the child. Time since the child was implanted (CI age) did not influence the change in CAEPs across the three month period. The data appears to be evenly distributed throughout the age range, and no outliers were observed to skew the presence of significant effects. It is probable that the
lack of effects seen with CI age may be attributed to the various degrees of auditory
depprivation and maturation across individuals, not fully accounted for by the CI age measure.

5.2.2 Effects of auditory deprivation and maturation

The range of hearing histories amongst the group of children was explored by comparing P1
latencies to a range of measures. The effects of auditory deprivation and neuromaturation
were considered simultaneously by plotting P1 latency as a function of chronological age, CI age, the nature of deafness onset and progression of hearing loss. For the developing auditory
system, P1 has been established as a biomarker for neuromaturation (Sharma, Dorman, et al.,
2002b). Although no age-matched normal hearing participants were recruited in this study,
there are large samples of normative data from the literature (Ponton, et al., 1996; Purdy, et
al., 2011; Sharma, Dorman, et al., 2002b; Sharma, et al., 1997). The normative data from
Sharma et al. (2002b), represented by 95% confidence intervals, were enlisted for
comparative purposes as this data has been widely used for studies concerning cortical
auditory maturation and effects of implantation throughout the literature (Sharma, et al.,
2007; A. Sharma, et al., 2004).

It is important to note the differences between the current study and the studies from Sharma
et al. (1997, 2002b), which may account for some differences that were seen. All of their CI
children had either congenital deafness or were severe to profoundly deaf by the age of one.
All testing were conducted at least six months post-switch on, using the synthetic consonant-
vowel /ba/ with 90 ms duration. The formants of the consonant-vowel transition in the /ba/
cover a wide frequency range between 234-4500Hz, although concentrated with low starting
frequencies at 234 Hz and 616 Hz. Thus for comparison purposes P1 latencies were averaged
across the stimuli /m/, /g/, and /t/ in the current study.

5.2.2.1 Age of implantation

The effects of early, middle and late implantation for the children in the current study were
consistent with data from Sharma et al. (2002b). The children implanted prior to 3.5 years
were within the range of expected P1 latencies for their age, whilst those implanted later
displayed later P1 latencies near the upper 95% confidence intervals of the normative data.
The only noticeable difference observed is that the children implanted between 3.6-7 years,
d deemed as bordering the sensitive period of auditory plasticity, displayed P1 latencies that
were all within the norms. One explanation for this may be that not all participants in the current study are congenitally deafened and perhaps were adequately stimulated by hearing aids until their CI. Thus some form of auditory development may have progressed during this period of maximal plasticity, as reflected in their normal P1 latencies. Furthermore, Sharma et al. (2002b) also suggested that age at implantation may not be the only contributing factor as there are records of late implanted children with normal P1 latencies.

Additionally, the adult participants 23 and 24 who both had progressive sensorineural hearing losses which were congenital in nature, were observed to have prolonged P1 latencies even in adulthood (Figure 42). Participants 23 and 24 are comparable to the congenitally deafened adult implantees also plotted in the Sharma model (Sharma, Dorman, et al., 2002b); adding to the growing view proposed by Ponton et al. (1999), that long periods of deprivation prior to implanting congenitally deafened adults may result in an asymptote effect for auditory cortical development. The current belief is that there is no deadline or ‘critical cut-off’ point for implantation, but the longer the period of deprivation may reduce the rate of change, if any, seen in the P1 biomarker (Harrison, et al., 2005). Although the two adult participants vary in chronological age and age of implantation, they provide interesting comparisons to the case studies identified in the literature of late-implanted adult CI users.

5.2.2.2 CI age

Ponton et al. (1996) noted that P1 latencies of implanted children can be normalized with the knowledge that they are delayed by the duration of auditory deprivation from their normal hearing peers. Therefore, when plotting the P1 latency of CI children as a function of ‘hearing age’, the hypothesis is that the spread of data should be compensated for by this correction and thus P1 latencies should be indistinguishable from normal hearing latencies; this pattern was observed here. However, when the age of implantation was considered, the children implanted later were actually well within norms or had even shorter P1 latencies. This may be largely explained by the sample of children from which the age correction was derived (Ponton, et al., 1996); whose participants had unstimulated auditory systems prior to their cochlear implant. The clustering of P1 latencies from the late implantees in the children from part II, suggests that these children were well aided prior to their CI; and their true CI age or amount of time with appropriate auditory experience is longer than indicated on the
graph. This is consistent with the cochlear implant programme policy of requiring optimized hearing aid experience prior to implantation.

Longitudinal studies following P1 latencies post-implant have found rapid reductions in the first 6-8 months post switch-on, with larger effects for earlier implantation (Sharma, et al., 2007). Sharma and colleagues (2007) also revealed that the rapid reduction in P1 latencies comes to an abrupt halt and plateaus into the normal range of P1 latencies expected from normal hearing children. Thus another explanation for the short P1 latencies in the late implanted children in the current study, may be that they have already undergone the rapid shortening of P1 latency, and will plateau into the region of normative data in due course. On the contrary, adult participants 23 and 24 appear unlikely to ever enter the realms of normative data for P1 latency, even after correcting for CI age. Participant 23 has less than 5 years as a CI user, and may perhaps have reduced P1 latency once she has caught up with the auditory delay. However, for both participants, it appears that their P1 latency may never reduce, although they were fitted with hearing aids by the age of 1 and 2 years. This further poses questions regarding the future of the group of children who were implanted after 7 years. The quality of hearing aid amplification prior to CI should be further explored as it may be a predictor of abnormal P1 development in late adolescence and adult life.

5.2.2.3 Deafness characteristics
The nature of deafness was characterised by two factors, the onset and pattern of deterioration. This categorization was based on two aspects of participants’ hearing history which could be confidently ascertained. It should be noted that several other factors may have influenced their hearing loss history, including the consistent use of appropriate amplification, genetic components to their aetiology, exposure to auditory training, schooling and behavioural interactions, residual hearing, and threshold fluctuations (Sharma, Dorman, et al., 2002b). Quality of life studies have found that consistent hearing aid usage prior to implantation has been associated with better success and performance with the CI (Klop, Briaire, Stiggelbout, & Frijns, 2007). Hence the following characteristics were compared amongst the child dataset with the considerations that interactions of these factors may also contribute in varying degrees to the child’s success and performance with their CI.
These classifications revealed some trends associated with maturation and auditory deprivation across the children. It was clear that the group who had a hearing loss of severe to profound levels at birth were all detected early and implanted early. These children can be considered as a model population for whom early intervention took place. The line of best fit of the positive correlation between chronological age and CI age (Figure 43), can thus act as visual indicator of those implanted later than this model population, which would be found below the line. These categorisations also found a group of children whose deafness were progressive in nature, although was present in some degree at birth. These children from the data set displayed a wide variety of experiences with CI, although they were all in the older children group (7-12 years). Only two children had acquired hearing losses, which reduces the reliability of their observations.

Results from the current investigation shows that those with congenital severe-to-profound hearing losses displayed normal P1 latencies, as expected when early intervention takes place and appropriate amplification or implantation is immediately acted upon. For the group of children with congenital progressive hearing losses, it is interesting to note that despite the variation in experiences with CI, it did not appear to have an effect on the P1 latency of their cortical responses. This evidence may further suggest that age of implantation is not the most important factor involved in ensuring progression of normal auditory development, reflected by P1 latencies. With an older CI age, it may be assumed that the child was implanted earlier and thus should have more of an age-appropriate P1 latency as observed in the congenital severe-profound group. However, all congenital progressive children were clustered on the upper limit of the normative P1 latency data. It is indicative that their hearing aid usage may perhaps play a larger role in explaining the consistent P1 latencies seen across this group.

The effect on P1 latency due to the characteristic of an acquired hearing loss could not be appropriately ascertained due to the low statistical power of the small sample size. The implication of an acquired hearing loss is that the auditory system may have received sufficient stimulation prior to the loss, allowing some degree of higher-order development. The child participant 9 who had an acquired progressive hearing loss displayed a delayed P1 latency, which was previously considered within normal values when accounted for her CI age. However, the accuracy of her CI age is confounded by her long history as a hearing aid wearer. Previous studies have characterised congenital and acquired nature of deafness via
prelingual and postlingual subcategories. Although considered, this categorisation was abandoned in the current study as deafness onset may have occurred during the period of oral language development and this was difficult to determine retrospectively.

**5.2.2.4 Morphological changes in maturation**

The grand averages of the two age groups in the children, and adults, consistently show age-appropriate waveform morphologies and CAEP measures across stimuli. These results are supported by the wealth of normative data recorded at Cz for children and adults throughout the literature (Ponton, et al., 1996; Ponton, et al., 2002; Ponton, et al., 2000). Furthermore, it suggests that these responses are similar to normal hearing individuals even though all participants were CI users. This provides further insight into the groups of children and adult CI users who were sampled from the current CI programmes, with no restrictions made on the types of deafness and duration of deafness. These CAEPs indicate relatively normal central auditory detection of speech sounds in these CI users. This is consistent with the 95% confidence interval of HINT in quiet speech scores of the adult participants from 76-94%, showing relatively good behavioural performance with the CI.

It is interesting to note that for the adult participants 23 and 24 who displayed a delayed CAEP morphology, their overall responses are still later in comparison to the child data. This is consistent with the delayed P1 latencies even when their CI age is accounted for. Ponton et al. (1999) suggested that in cases of post-natal hearing loss, the state of development in the auditory system may be ‘frozen’ for the structures which have completed their maturation. Once people with post-natal hearing loss were implanted, they suggested that the auditory system may also take some time to ‘thaw’ and regain accurate firing of neurons in synchrony for appropriate signalling. For participant 23, it was clear that the deterioration in her hearing was progressive, with large shifts in later stages of adult life. For participant 24, it was more difficult to ascertain if the delay in implantation was due to a progressive loss after onset of meningitis or due to social factors. It may be postulated that although participants 23 and 24 were fitted early on with hearing aids, their hearing deteriorated to a point where the auditory system has suffered a period of deprivation during the development of auditory structures and connections. Once they were implanted and ‘thawed’, the re-stimulation may have attempted to kick start a second spurt of maturation (Ponton, et al., 1999). However, this may have been hindered by how late they were implanted and the duration of the deprivation. It is also
important to note that although the adult cortical processes are still plastic (Pantev, et al., 2006), this plasticity may not be enough to reconfigure maturational processes dependent on the delay and severity of deprivation, which is reflected in their CAEPs.
5.3 The stimulus artefact in CI users

Currently one of the largest issues with recording reliable evoked responses from CI users is the presence of the stimulus artefact. Therefore, another important aim of the study was to analyse and characterise the artefact, and to explore previous techniques used for artefact reduction.

5.3.1 Artefact categorisation and characterisation

Four types of CAEP recordings were identified in respect to the presence of the stimulus artefact. Of particular interest was the presence of the blip artefact which featured prominently in both adults and children groups, and the pedestal artefact which although less common but more detrimental to CAEP detection. These two typologies were further characterised as they potentially contaminated the CAEP response in different ways; together responsible for affecting the reliability of CAEPs in over 76% of adults and 83% of children. It should be noted that there were similar proportions of each typology in adults and children supporting the idea that the artefact is most likely due to contributions of electromagnetic disturbances from the CI device, instead of physiological disturbances such as maturation of the central auditory system. The slightly higher proportion of artefact contamination in children may be explained by the device and stimulation type in the child group. The current spread associated with the Nucleus CI 24 internal implant with two extracochlear electrodes is likely to explain the high incidence of CI artefact in CAEP recordings since the launch of the device. All children were implanted with this model. The long distance between ground and active electrodes is thought to evoke an electromagnetic response at the scalp, due to the distance of the electrode return path. There were four adults with the Nucleus 22 implant, of which three were operating in bipolar mode and one in common ground mode, which may have contributed toward their slightly lower incidence of artefact presence.

Speculation that the extracochlear electrodes and monopolar stimulation are main contributors to the implant artefact is supported by experiments of electrode stimulation in animal tissue models (Xiaoxia, et al., 2010). These experiments have found that monopolar stimulation produced a large disruptive artefact compared to the null effect observed with bipolar stimulation. Xiaoxia and colleagues (2010) found that a consonant-vowel-consonant
stimulus produced more variability in the shape of the artefact than a 1000 Hz tone of the same duration. This has implications for testing CI users with speech-evoked cortical responses. The tone produced an artefact which was stable and time-locked to the stimulus, while the speech stimulus artefact varied its amplitude with the characteristics of the stimulus. It is likely that this variation may be confused with parts of the CAEP response when the artefact amplitude is smaller or less disruptive. With larger artefact amplitude, the CAEP is obliterated. However, a smaller and varying artefact may be more problematic as it will reduce the reliability of clinical judgements.

The previous reports of artefacts have mostly described a pedestal morphology which was responsible for masking the entirety of the waveforms of interest. This artefact morphology was also identified in the current investigation and although variable in amplitude, this type of artefact consistently enveloped the neural response (Figure 49). This pedestal morphology was present in adults who were stimulated via the ACE strategy, monopolar mode through a CI 24RE internal implant, and one child who was stimulated via ACE with monopolar mode in a CI 24M internal implant. The only difference regarding their implant programming between these participants was that the child (participant 16) used a faster rate of 1200 Hz than 900 Hz. However, many other adult and child participants were also wearers of the same devices and identical processing details, which suggest that these are not the only factors involved. Surgical positioning of the electrode array and the ball and plate electrodes’ positions relative to the intracochlear array, have also been suggested to influence the response (Shallop, 1993). It is perhaps possible that individual variations in CI electrode placement may predict the presence of artefact, rather than subtle differences in the positioning of scalp recording electrodes between test times. Although the scalp recording electrode orientations may affect the artefact amplitude (Shallop, 1993), the repeatability of artefacts for the same stimuli in the child participant in session two, suggests that the generation of the artefact may be mostly a result of the internal device.

It is also interesting to note that the pedestal artefact was present in the responses to high frequency and mid frequency stimuli. The single pedestal recorded in adult participant 22 to the low frequency /m/ phoneme, may be an outlier of the dataset as this participant also had absent CAEP responses to /g/ and /t/ without the presence of a pedestal artefact. Participant 22 struggled with speech perception testing and had to readjust his CI maps prior to the
CAEP recordings as he was unhappy with his map. The presence of the pedestal morphology in the high and mid frequency stimuli, rather than the low frequency stimuli may be attributed to the spectral and temporal similarities of the phonemes /t/ and /g/. The similarities could mean that they are filtered and processed by similar filterbank channels, and thus delivered to nearby regions of the electrode array. The electrodes responsible for mid to high frequency responses will be responsible for the delivery of the processed /t/ and /g/ stimuli. As has been suggested that there are only approximately 4-8 neuronal populations which exist at the electrode-nerve interface (Wilson & Dorman, 2008), it is likely the overlap in /g/ and /t/ spectral energy may be a factor. It would be of great interest in the future to obtain CT images of the individuals with the pedestal and other types of artefact for further comparison of artefact morphology and device orientation. It must be remembered that all of these propositions are only based on the findings of the pedestal morphology in five individuals; a much larger sample is required to have confidence in this characterisation.

As contamination from the pedestal morphology affects the entire CAEP recording, the literature has largely ignored other forms of the stimulus artefact. In the current investigation, a rapid and short deflection with small amplitude, usually positive in polarity and preceding P1, was categorized as the blip artefact. This is due to its rapid nature and visual presentation in the CAEP. The artefact was observed to be repeatable upon retest in children, and its presence remained under the different slow and fast conditions; a confirmation that it is a non-neural response likely due to similar factors discussed for the pedestal artefact. In both adult and child groups, this was recorded in approximately 70% of adults and 80% of children, as the most prominent artefact typology. This is also observed across many grand average waveforms in evoked potential studies throughout the literature (K. Gordon, et al., 2008; K. Gordon, et al., 2010; Sharma, Dorman, et al., 2002b). The impact of this artefact upon the identification of the neural response is not as great as for the pedestal morphology. In the children, the blip peak and its offset is earlier than the average adult, thus more likely to leave the P1 responses untouched. For the adult participants, the average offset of the blip artefact occurs at around 63 ms, which is problematic for P1 identification as the mature adult P1 waveform is situated around 50 ms. As indicated by Figure 48, CAEP waveform interference from the ‘ringing’ near the offset of the blip artefact proves problematic for visual identification of the adult P1 waveform. This may have contributed to the low number of present P1 peaks in comparison to N1 and P2 in the adult CI users (Figure 50).
The presence of a clear blip in all the pedestal recordings, leads one to suspect that perhaps there is a continuum of stimulus artefacts and the blip is simply a small version of the pedestal morphology. However, no difference in the offset latency of the blip artefacts was noted across stimuli, dissimilar to the possible high frequency bias observed for the pedestal morphology. Another thought could be that different processing strategies and features, or combinations of them, have resulted in these artefact typologies. Future investigations with combinations of processing techniques may provide more understanding of the nature of these artefacts. This area of research becomes more relevant as the technology advances and a wider variety of devices are available to CI candidates, and there is greater need for objective verification of the CI map using CAEPs as younger children are implanted. From a clinical perspective, the best candidates for CAEP evaluation of their implants are the young children identified through early intervention and screening programmes, who will receive more recent models of the implant. Because this is the population that will potentially benefit the most from mapping verifications via CAEPs it is important to develop artefact minimising strategies to improve clinical reliability of CAEPs for children in particular.

### 5.3.2 Artefact minimisation techniques

The current study had the intention of exploring the adaptation of techniques for reducing artefact suitable for a clinically focused single channel CAEP recording, which differs from previous studies utilising multi-channel evoked potential systems that have applying post-processing techniques to reduce the artefact (Casarotto, et al., 2004; Friesen & Picton, 2010; Gilley, et al., 2006; Makeig, et al., 1997). Several aspects of the study were designed with the goal of reducing the artefact:

- Short stimuli duration
- Contralateral recordings
- Pre-processing filtering
- Post-processing subtraction

The brief duration of the speech stimuli used was based on suggestions for optimal stimulus duration and rate for studying speech-evoked CAEPs (Golding, et al., 2006). The short
stimulus duration is also advantageous as it gives an earlier artefact offset as the artefact is time-locked to the stimulus duration (Gilley, et al., 2006). The slightly shorter /g/ stimulus may have produced less contamination of artefact; this is supported by the earlier distribution of blip artefact offset latencies observed for /g/ compared to the other phonemes (Figure 47). However, the trade-off for reducing stimulus duration is that less speech characteristics is contained within the phoneme and thus less is available for analyses of its neural interpretation. This is an important consideration for determining the optimal stimuli duration for studying CAEPs in CI users.

Contralateral earlobe placement of the ground electrode was also utilised in the current investigation, in an attempt to further distance the site of artefact generation from the recording. This is a useful technique for CI users in the adult programme as current government funding has produced mostly unilateral users. However, the active electrode at Cz is still close to the implant device, and even closer in those individuals with smaller heads such as children. This may be another explanation for the higher incidence of artefact found in the children. It may be advantageous to consider the use of temporal sites of recordings contralateral to the CI, although more research into normative data and the effects of various speech stimuli on obligatory CAEPs in CI users are required for comparative and clinical purposes. Furthermore, there is evidence of strengthened signalling along ipsilateral pathways after auditory re-activation with CI, as compared to the contralateral bias in normal auditory processing (Ponton, et al., 2001). Ponton et al. (2000) found that the optimal electrode montage for recording different CAEP peaks is subject to maturational changes. Thus there is still a strong argument for pursuing optimal recordings at Cz based on the robustness of CAEP recordings at this site, and the importance of considering the responses from both cortical hemispheres for unilateral and bilateral implantees.

The HEARLab evoked potential system utilises a pre-acquisition filtering technique. The connectors used for cortical recordings on CI users consist of active recording electrodes at the scalp which contains a filter as well as a filter within the amplifier of the stimulus controller. The goal is to low pass filter the recording, prior to entering and processing at the stimulus controller and software level. This appears effective for the pedestal artefact, as the HEARLab was able to reveal CAEP waveforms for all the adult individuals with the pedestal morphology recorded only through the Bio-logic system. However, it would appear that it was not able to reduce contamination of the blip artefact as effectively; as P1 was detected.
more across stimuli using the Bio-logic system in adults. P1 is much smaller in adults than children, which makes it difficult to distinguish from noise and artefact, and is generally not analysed in studies of adult CAEPs.

The replication of the subtraction method (Friesen & Picton, 2010) with the single channel recordings has revealed mixed successes. The usefulness of obtaining a purely neural response by subtracting recordings for the slower and faster two rate conditions varied (Figure 51). In some participants the temporal overlap of the CAEPs from the two rates of presentation was poor, and the peaks were clearly unsynchronised. In such cases it was difficult to analyse the difference waveform as comparable to a normal N1 or P2 waveform. It is also clear in cases where the two rate conditions elicited CAEPs of similar amplitudes, that the subtraction proved non-advantageous in producing a neural response. In principle, this approach could be highly useful for reducing blip artefact contamination. However, with the compounding effects of noise, and uncooperative young participants, it was hard to gauge the overall effectiveness of the technique in clinical applications. It should be noted that Friesen and Picton (2010) only analysed this technique in five adult CI users, and a wider study population may have shown different success rates. For research purposes, multichannel systems and post-acquisition processing strategies remain a powerful tool in reducing CAEP artefact from CI users, but these approaches are not feasible for clinical assessment.
5.4 Strengths and limitations

5.4.1 Strengths

The relatively large sample size of adult and child CI users recruited is an important strength of the study. With the size of the sample, more confidence can be felt regarding over findings and repeated measures analyses. Standardised testing techniques, equipment set-up and parameters were administered for behavioural speech perception tests and CAEPs, which improves the general applicability of the results. Furthermore, the CAEP testing was applied in a clinical fashion, with a single channel recording montage, and short duration of test time, which means that results would infer feasibility of testing young children clinically. Thus the findings of stimuli effects and applicability of artefact-removal strategies can be shifted from research aims to address clinical needs.

More aspects of the waveforms were considered than just the traditional measures of latency and amplitude. Two methods of area estimates and peak-to-peak amplitudes were obtained, in order to provide insight into inter-wave interactions: these ultimately supported the pattern of stimulus effects observed in the traditional measures. The reliability of the measures was strengthened by having two independent observers who followed criteria for marking peaks of waveforms. Furthermore, the stimuli, CAEP recording parameters and evoked potentials systems were consistent in parts I and II. The stimuli were also calibrated at the level of the CI microphone to ensure their transduction through the speaker and acoustic environment did not significantly change the characteristics of the phonemes. CNC phonemes were further explored with the identification rate for each particular stimulus phoneme used for CAEP testing; this provides important considerations for future investigations.

Another strength of the study is the longitudinal insight into changes in CAEPs over time with increased CI experience. There is a need for longitudinal understanding of the exposure to long duration of deprivation and effects of age of implantation at varying chronological ages. Furthermore, this study was able to observe the same pattern of stimuli effects across both adult and child groups, which reveals that the neural detection of varying speech characteristics can be found with different degrees of auditory maturation in CI users.
Additionally, it joins the growing evidence for benefits of early implantation, and data for the well-studied phonemes /m/, /g/, and /t/.

5.4.2 Limitations

Recruitment restrictions based on the deafness history and aetiology were not made although differences in hearing pathologies, stimulus parameters and different CI devices are all known to have an effect on CAEPs. This should be considered with the correlation between P2 latency and speech perception, and the finding that changes in CAEPs are larger over 3 months as a function of age in the children. Furthermore, to observe effects of CI adaption over 3 months, some of the participants in part II were tested within 1 month post switch-on. This is both a strength and a limitation: as the immediate post-implant effects of auditory plasticity are of interest, but the CAEPs recorded may producing an apparent effect of plasticity in the form of unsettled CI maps. Another limitation is in the variance in CI device settings, especially across the adult group. This issue was addressed by the use of repeated measures analyses in which comparisons were made within individuals. For future consideration, integrity tests on implant electrodes for the different types of device users in the study may have been helpful to ensure variability seen in cortical response measured were not attributed to such factors.

Age-matched normal hearing controls were not recruited in this study, and may have been useful for comparison of effects across stimuli and speech perception, under the same testing conditions and CAEP acquisition parameters. This is a limitation because studies in this field have utilised an assortment of stimuli, parameters and CAEP protocols, so the findings, while relevant to the CI population, would be difficult to generalise to others. However, normal hearing trends in CAEPs have been established in children and adults across different stimuli and electrode recording sites (Ponton, et al., 1996; Ponton, et al., 1993; Ponton, et al., 2000).

Previous findings in hearing aid wearers suggested that CAEP assessments should take into account the change in the stimulus which arrives at the ear drum post-processing from the hearing aid (Mercer, 2010). Stimuli delivered to the CI microphone were recorded and shown in Figure 15, but no measure of implant processing was obtained at the electrode and nerve interface: the final point after the implant’s processing and thus analogous to the ear canal for an acoustic hearing aid. Future research should consider investigating effects of CI
processing on the chosen stimuli and their CAEPs, by comparisons with electroodogram outputs for specific devices, processing strategies, modes of stimulation, pulse width and rate of stimulation.

Variations in the acoustic environment between child and adult CAEP recordings may have occurred because they were conducted at different sites. This should be factored into the comparisons of P1 latency and morphologies between adult participants 23 and 24 and the children in part II. However, everything possible was done to minimise effects of the different recording rooms, as all CAEP parameters were maintained. Another limitation was the short duration of pre-stimulus recording that was used for baseline correction of waveforms. While this would be adequate for clinical purposes, it may have been too short to allow the baseline cortical potential to be estimated accurately enough to allow precise identification and calculation of the outcomes used in the research. An example of a CAEP which may benefit from a longer pre-stimulus baseline correction can be found in participant 1 for /m/ (Appendix 5).

CAEPs were generally recorded after the participants’ clinical appointment with their CI audiologist, to assist participant recruitment and to ensure that maps were optimal. Participant 17 had had large changes made to his CI map after poor speech perception results were found, and his CAEPs were either absent or contaminated with the pedestal artefact. This may be due to actual absence of CAEPs or it could be due to the significant map changes. No significant map changes for other participants were reported. This procedure was not followed in the children, because time constraints on availability of the room meant that most CAEP sessions were conducted at separate times from their audiology appointments. Therefore, the child CAEPs may possibly have been in response to suboptimal CI stimulation maps.

Variability in co-operation of the young children was a factor in data collection. The CAEP acquisition procedure had to be flexible around each child’s behaviour. This meant that recordings were affected by acoustic noise from movement and the environment, electrical noise from muscle activity, and less than optimal electrode impedances. Thus, accuracy and reliability of CAEP traces were lower in the younger children than others. Where absent CAEPs were reported, it was difficult to ascertain if it is a true neural absence or simply due
to the reduced reliability. However, this was compensated to a degree by taking breaks between parts of testing, and attempts to ensure optimal behaviour with the assistance of a parent, audiologist and silent distractions.
5.5 Future directions and implications

Electrodograms should be included in future studies as they portray the signal temporally and spectrally, and display the configuration of activation across the CI electrodes. Further understanding of the relationships between electrodograms and CAEPs may be important clinically; and a combination of both objective measures to assist CI mapping may be useful.

An increase in P1 amplitude was found over time in younger children in response to higher frequency phonemes. This would reflect greater plasticity in the younger auditory system, where rapid cortical developments would occur post-implantation. On the other hand, the age of implantation may not be the only factor involved in prediction of P1 maturation. Current findings suggest that there is a large scope for longitudinal exploration of auditory experience, duration of deprivation and deafness characteristics found in CI users. The factors in this field are highly interrelated and influential on the morphology and measures of the waveforms. It will be important to trace the changes over time to ascertain if maturational changes and increased CI experiences will alter the responses across stimuli in a different way. Perhaps other waveforms such as N250 and its measures may have greater sensitivity for maturational changes in young CI users. Further understanding of these mechanisms may be the key to comparing CAEPs and fine adjustments in CI mapping for future clinical use.

Abnormal morphologies from prolonged deafness duration in late adolescents provide another interesting area for further investigation. An implication is that with the growth of early intervention and newborn screening programmes, it will be difficult to recruit participants with long periods of auditory deprivation, or those implanted late with severe to profound congenital hearing impairments. Thus, research into those who have experienced this outcome should be conducted soon, while they are still available. Studies of deprivation and plasticity in animal models may become the only practical option to research this phenomenon in the future. There is much scope for understanding the exact mechanisms and progression of the morphologies and their related causes. Several recent reports of atypical morphologies in children are interesting and such cases are worth following longitudinally with CAEPs (K Gordon, et al., 2010; Sharma, et al., 2009). Gordon et al, (2010) have also suggested a genetic basis in GJB2 mutations for some of these morphologies; which may be grounds for further genetic investigations with abnormal CAEP morphologies in CI users.
Contrary to previous evidence (Kelly et al., 2005), better behavioural speech perception predicted longer P2 latencies in the adult CI users. This raises questions regarding the variability of the P2 waveform under the influence of auditory experience with cochlear implants, and its effectiveness as a predictor of speech perception performance. Future research should investigate the variability in the P2 waveform. Greater understanding of its relationship with speech perception may be advantageous for CI mapping clinically. One possibility is to use specially designed phonemic perception and discrimination tests which focus on the same phonemes used as stimuli for CAEPs; similar to the identification rate derived from the CNC phoneme test in the current investigation. It may be possible that the array of general speech perception tests, although an indicator of CI performance, are not specific enough for effects to be revealed when comparing with CAEPs evoked from speech phonemes.

Research with obligatory CAEPs may be valuable despite the growing interest in the acoustic change complex; which provides more speech processing information. Tremblay (2008) suggested that the acoustic change complex may become useful once more research has established effects of various parameters. However, obligatory P1, N1 and P2 waveforms remains currently the most clinically effective objective measure. The differences observed across /m/, /g/ and /t/ in the current investigation indicate that CAEP recordings to these phonemes may be useful clinically. Should this be the case, larger scale studies may wish to consider obtaining normative data for CI users. It is also possible that the difference between /g/ and the other phonemes can be ascertained with a larger sample size. It is important that further research investigates the feasibility in using CAEPs for mapping changes because it is currently uncertain if CAEPs can detect fine changes in hearing aid amplification (Tremblay, et al., 2006).
CHAPTER 6 SUMMARY AND CONCLUSION

The current study has revealed that three phonemes, spanning the low, mid, and high frequency regions of the speech spectrum can produce different CAEPs in adults and children with cochlear implants. The N1 waveform in adults had longer latency, and was larger in amplitude and area for the stimulus /m/ compared to /t/. On average, CAEPs in response to /g/ fluctuated between the values for the other stimuli, and thus the main difference exists between the high and low frequency phonemes. The same pattern was observed for P1 latency and area in the children. These findings suggest that the neural detection of differences in speech characteristics is present in CI users. Longer P2 latency was revealed to be associated with better speech perception, which may be an effect of using speech stimuli.

Stimulus artefacts were categorised as having a blip or pedestal morphology. The offset latency for the blip artefact may be useful in considerations for future filter settings in evoked potentials systems, and clinical judgements of P1 and N1 presence. Artefact minimisation using the subtraction method yielded small success in reducing the blip artefact in single channel recordings. The HEARLab recordings indicated that the pedestal artefact may be minimized with pre-acquisition filtering techniques. These implications may be useful for both research and clinical purposes.

Obligatory CAEPs are a feasible tool in assessing phonemic processing in CI users. The objective nature of the measure makes it appropriate for difficult-to-test populations and children implanted prelingually. CI users’ CAEPs could be monitored across assessments from CI candidacy, switch-on, to the development of spoken language. There is potential for its use in providing frequency and/or phoneme specific information for monitoring rehabilitation, and the identification of need for explantation. A useful initiative would be to establish a foundation of evidence for optimal parameters and protocols for speech-evoked CAEPs, and to determine if CAEPs are of use for fine-tuning CI maps. Further insight into the relationship between P2 and speech perception tests targeting phonemic discrimination would be useful as well.

Finally, with the expected increase in CI users due to newborn hearing screening, the use of CAEPs is likely to shift to a stronger clinical focus. Larger scale studies may be required
obtain normative data for clinical use, and longitudinal exploration of CAEP changes with CI experience for various hearing pathologies would be invaluable. Understanding these longitudinal effects on the auditory system would assist the provision of clinical advice for CI candidates who are hesitant to proceed with implantation, and thus optimise success rates for future CI users.
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## APPENDICES

### Appendix 1: Participant demographics for part I and II

**Adult demographics**

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R= right ear, L= left ear. For participant 18 who was bilaterally implanted, the right implant was switched off during testing. Types of deafness were categorized into those who had some degree of deafness at birth (congenital) and those who acquired a hearing loss later in life (acquired). Duration of profound deafness was a behavioural measure of their hearing loss became ‘profoundly disabling’; which was ascertained by the time since they last had a two-way conversation on the phone. The CI age was defined by number of months since their CI switch-on.
### Child demographics

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Age is described in years and at the time of the first test session with the child. Five of the sixteen were implanted bilaterally, but not necessary during one surgery. The implant used for both CAEP and speech perception testing is indicated in parentheses. Type of deafness were classified as congenital severe to profound (CSP), congenital progressive (CP), acquired severe-profound (ASP), and acquired progressive (AP). Duration of deafness is defined as months of time from diagnosis of profound deafness to switch-on of CI. Their CI age is the duration of months since switch-on and use of CI.
### Appendix 2: CI processor and implant details

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* With speaker volume control set at maximum
**With speaker Volume control set at 2 steps/levels below maximum level
UOAC=University of Auckland Clinics (Part I)
HH=Hearing House (Part II)
Appendix 4: Part I (adults) individual CAEP measures

For all stimuli, main CAEP measures of latency, absolute amplitude, area 1 (A1), area 2 (A2) were obtained for N1 and P2 waveforms in adult participants 1-22. The same CAEP measures were obtained for the P1 waveform found in participants 23 and 24.

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Appendix 5: Averaged CAEPs for adult participants for the slow rate condition
Note that for P1-17, P18-22 and P23-24 different sets of amplitude scales were used for comparative purposes within individuals with normal CAEPs, pedestal artefact, and P1 morphologies respectively.
Appendix 6: Part II (children) individual CAEP measures

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Appendix 7: Averaged CAEPs for child participants for the slow rate condition
T1= test session 1 and T2= test session 2. Note that participant 1 was not tested for a second time, and that participant 16 has a different scale due to the presence of pedestal artefacts.
Appendix 8: University of Auckland human participant ethics committee approval letter

Office of the Vice-Chancellor
Ethics and Biological Safety Administration

21 May 2010
MEMORANDUM TO:
Dr David Welch / Ruth Lin
School of Population Health

Re: Application for Ethics Approval  (Our Ref. 2010 / 215)

The Committee met on 19-May-2010 and considered the application for ethics approval for your project titled "The neural processing of speech stimuli in cochlear Implant users". Ethics approval was given for a period of three years.
The expiry date for this approval is 19/05/2013.
If the project changes significantly you are required to resubmit a new application to the Committee for further consideration.
In order that an up-to-date record can be maintained, it would be appreciated if you could notify the Committee once your project is completed.

Please contact the Chairperson if you have any specific queries relating to your application. The Chair and the members of the Committee would be most happy to discuss general matters relating to ethics provisions if you wish to do so.

ALL COMMUNICATIONS WITH THE UAHPEC REGARDING THIS APPLICATION SHOULD INDICATE OUR REFERENCE NUMBER.

Lana Lon
Executive Secretary
University of Auckland Human Participants Ethics Committee

C.C. Head of Department / School, School of Population Health

Ruth Lin
Dept of Audiology

1. Should you need to make any changes to the project, write to the Committee giving full details including revised documentation.

2. The approval is for three years. Should you require an extension write to the Committee before the expiry date giving full details along with revised documentation. Extension can be granted for up to three years, after which time you must make a new application.

3. At the end of three years, or if the project is completed before the expiry, you are requested to advise the Committee of its completion.

4. Do not forget to fill in the ‘approval wording’ on the Participant Information Sheets and Consent Forms giving the dates of approval and the reference number before you send them out to your participants.

5. Please send a copy of this approval letter to the Manager - Funding Processes at Research Office if you have obtained any funding other than from UniServices. For UniServices contract, please send a copy of the approval letter to the Contract Manager at UniServices.

6. Please note that the Committee may from time to time conduct audits of approved projects to ensure that the research has been carried out according to the approval that was given.